Obesity and Negative Affect:

A Multi-Disciplinary Exploration of the Interaction Between Mental and Physical Health and its Effect on Cognitive Function

by

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RESEARCH STUDENT DECLARATION FORM

Type of Award Doctor of Philosophy _____

School **Psychology and Humanities**_____

1. Concurrent registration for two or more academic awards

*I declare that while registered as a candidate for the research degree, I have not been a registered candidate or enrolled student for another award of the University or other academic or professional institution.

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***I declare that no material contained in the thesis has been used in any other submission for an academic award and is solely my own work.

3. Collaboration

The candidate's individual contribution and the extent of the collaboration:

The data used in Study 1 were obtained as part of a wider collaborative research project. The work was published in Journal of Youth and Adolescence as 'Reciprocal Prospective Relationships Between Loneliness and Weight Status in Late Childhood and Early Adolescence' by Qualter, P1., Hurley, R2., Eccles, A., Abbott, J3., Bovin, M., & Tremblay, R. [, 1=Supervisor, 2= Student, 3= Director of studies].

RH completed the data request, selected the variables of interest for Study 1 from the wider data set. Carried out background research, data translation, cleaning, devised the strategy to calculate child and gender specific BMI, prepared the data set and performed initial exploratory analyses, wrote an initial draft of the introduction, results tables, contributed to the interpretation of results and helped answer reviewer questions during the paper review process._____

4. Use of a Proof-reader

*No proof-reading service was used in the compilation of this thesis.

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Abstract

Background

In 2015 The ROAMER project used expert consultation to set priorities for public mental health. These included increased research into the interaction between mental and physical health conditions. This thesis examines the interactions between obesity and affective disorders, two major foci for public health, using a range of investigative methods. The research was used to develop a neurocognitive model to explain mental-physical health links between the conditions.

Methods

Study 1 BMI and loneliness: analysed longitudinal research data from a large high-quality multidisciplinary health study (the Quebec Longitudinal Study of Child Development; n=1042: female = 572) utilising anthropometry and loneliness measures to examine the interaction between adiposity and socially mediated negative affect over time. Confirmation of the longitudinal relationship between these variables was used to develop a novel theoretical model to explain the neurocognitive mechanisms that link and reinforce these conditions.

Study 2 Body mass, negative affect, and cognitive function: A multi-disciplinary investigation of anthropometric, affective, and neurocognitive indicators in a single sample of young adults (n=90). Measures of BMI, waist circumference (WC), depression, anxiety, rumination and worry, cognitive performance (Continuous Performance Task of attention, Simon task of spatial response inhibition), and self-reported executive function problems (BRIEF scale). Neurovascular activity (functional near infra-red spectroscopy) and functional connectivity were examined by health group during resting state and task.

Results

Study 1: A series of hierarchical regressions found a significant linear effect of BMI on loneliness (Beta = 0.11) and a significant curvilinear effect of loneliness on BMI (Beta = 0.02) one year later. These reciprocal effects are likely to be mutually reinforcing over time. The study shed light on the time-course of physical and mental health effects between negative affect and body mass.

Study 2: Negative Affect and Adiposity: brooding rumination predicted BMI and WC; WC predicted rumination (explaining around 5% variance). **Cognitive performance:** The high BMI group (>30) displayed more attention and inhibition errors than low BMI. The high depression and brooding rumination groups displayed more response inhibition errors. **Self-Reported Executive Function Problems**. The high self-reported inhibition group had more attention errors, and the inhibition error rate was approaching significance. BRIEF subscales were strongly and positively associated with negative affect, and moderately associated with BMI (r=.30) and WC (r=.32). **Neurovascular activity in the frontal and temporal lobe**. In the resting state higher WC was related to reduced left frontal neurovascular activity (6% variance explained). Higher worry was related to more activity in the right temporal region (explaining 8% of variance). Those high in brooding rumination had greater functional connectivity in the temporal lobe and frontal gyrus than low brooders (superior and medial) during the resting state, but the reverse pattern was observed during the cognitive tasks (functional connectivity was much less extensive). The high WC risk group had reduced functional connectivity in the resting state and attention task, but widespread functional connectivity during the inhibition task.

Conclusion

Study 1 makes important contributions to our understanding the temporal relationship between adiposity and negative affect. Study 2 identifies repetitive negative thinking as an important predictor of health and cognition and a potential target for intervention to address the negative reinforcement between weight gain and negative affective conditions. Evidence also supports the proposed theoretical model which to my knowledge is the first to attempt to explain the neurocognitive processes that mediate physical and mental health through repetitive negative cognition.

Table of Contents

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Abbreviations

Abbreviation	
ACC	Anterior Cingulate Cortex
BMI	Body Mass Index
BOLD	Blood Oxygen Level Dependent
BRIEF	Behaviour Rating Inventory of Executive Function
CAMMPI	Cognitive-Affective Model of Mental and Physical Health Interaction
CT	Continuous Performance Task
DL PFC	Dorsolateral prefrontal cortex (superior frontal lobe)
DMN	Default Mode Network
EF	Executive Function
FC	Functional Connectivity
FL	Frontal Lobe
TL	Temporal Lobe
fMRI	Functional Magnetic Resonance Imaging
fNIRS	Functional Near Infra-Red Spectrometry
IFG	Inferior frontal Gyrus
LF	Left Frontal Lobe
	Left Temporal Lobe
MA	Movement artefacts
Mrt/ MRT	Mean Reaction Time
MTG	Medial Temporal Gyrus (middle temporal lobe)
OHb	Oxygenated Haemoglobin
PFC	Prefrontal cortex
RCT	Randomised clinical trials
RNT	Repetitive Negative Thinking
RF	Right Frontal Lobe
RT	Right Temporal Lobe
RS	Resting State
RUM; RUMb; RUMr	Rumination; Brooding; Reflective
ST	Simon Task
VL PFC	Ventrolateral prefrontal cortex (inferior frontal lobe)
WC; WCr	Waist Circumference; WC risk
MDD	Major Depressive Disorder

Figures

Prelude

Disorders related to adiposity and mood and anxiety disorders (characterised by negative affect) are common. According to the ONS around 16% of adults in the UK (Attwell et al., 2022) experience moderate to severe depression symptoms (pre-pandemic levels were 10%). Depression and anxiety are leading causes of disability (Kessler & Bromet, 2013) yet experimental method and the medical model has thus far failed to yield an effective explanation (Moncrieff et al., 2022) or treatment for the majority of sufferers (Thornicroft et al., 2017). An extensive expert review of European public health priorities in 2015 identified that mental health research lacks integration with physical health research (Forsman et al., 2015) and it fails to take account of a spectrum of causal mechanisms revealed by recent scientific advances (Schumann et al., 2014).

The thesis examines adiposity and negative affect and seeks to establish the extent that these conditions are mutually reinforcing. It also explores the possibility of a common mechanism of effect. The program of study takes a multi-variable, cross-disciplinary approach to investigate high priority medical concerns and identify important variables that could guide future study. The initial chapter outlines the strengths and challenges of holistic consideration of mental and physical health in research and treatment. This is followed by an outline of key variables in the study (see Figure 1) including approaches to the measurement and evidence for physical and mental health interaction. Potential mechanisms of effect to explain the interaction of adiposity and mental health are outlines with reference to likely somatic pathways identified by Schumann et al., (2014).

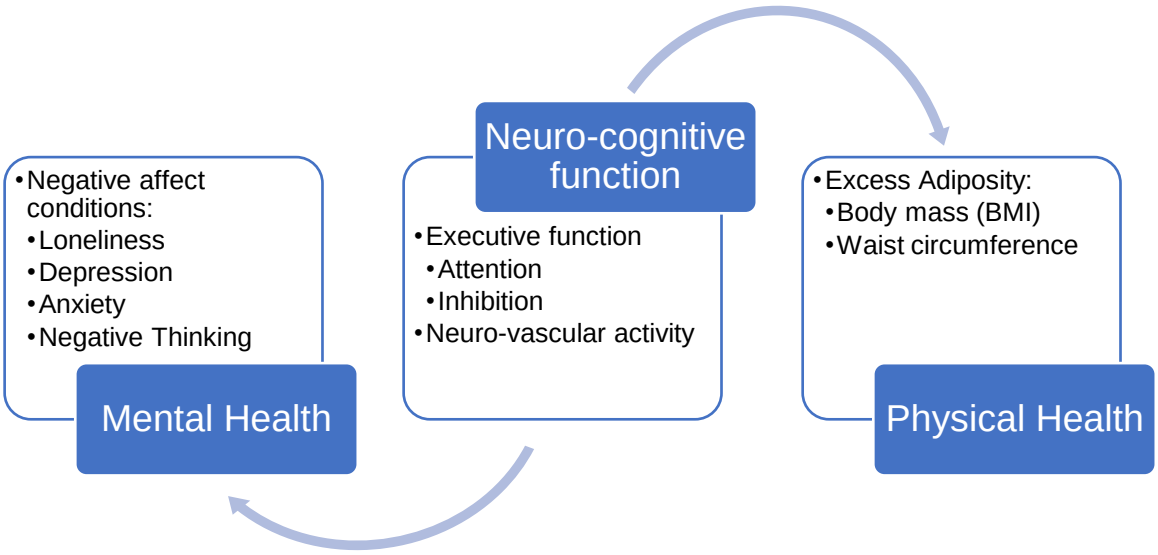


Figure 1
Key Variables Studied in the Thesis: Investigating the Link Between Mental and Physical Health Via Neurocognition.

Chapter 2 of the current thesis then proposes a cross-disciplinary health model to help explain potential interactions between physical and mental health variables (the Cognitive-Affective Model of Mental and Physical Health Interaction; CAMMPI). The theoretical model drawings on existing health models and research for the health effects of adiposity and negative affect (including repetitive negative thinking; see Chapter 3).

Study 1 (Chapter 4) examines loneliness as a negative affective condition (like depression and anxiety) which research suggests also results in poorer physical and mental health outcomes. The published manuscript is included in Appendix D. Evidence for potential shared mental and physical causes of loneliness and obesity were investigated in cross-lagged analysis. The data for Study 1 was obtained from a large, high quality, long-running cohort study, (Quebec Longitudinal Study of Child Development, for the years 2008-2013). Loneliness is a well-established source of negative affect in the lifespan; however, many studies focus on older adults where

results are confounded by age-related cognitive effects. Hierarchical regression analysis (n=1042: Female = 572; Male = 470) was used to examine the direction of the relationship between adiposity and negative affect in adolescents over a five-year period. The study found significant predictive effects of both variables after one year, indicating the variables could be mutually reinforcing over time and lending tacit support to ideas within the proposed model.

Study 2 (Chapter 5 to 8) is a large program of work that investigates key ideas from the model including neurocognition as a link between mental and physical health. The neurocognitive and socio-emotional association between several indicators of physical and mental health are examined in a novel cross-disciplinary design. A single group of participants completed tasks measuring physical and mental health from different physiological and psychological approaches:

- Anthropometric body measurements of BMI and WC.
- Clinical mental health measures of depression, anxiety, and repetitive negative thinking (rumination and worry).
- Cognitive performance measures (attention and response inhibition).
- Neurovascular activity (fNIRS scans measured brain oxygenation) during resting state and task.

The study findings (see Chapter 7) outline the link between physical and mental health measures, cognitive performance (attention and inhibition), self-reported cognitive problems and neurovascular activity. Neurovascular activity and functional connectivity were examined in relation to health during the resting state and during cognitive performance tasks. Chapter 7 also provides an overview of the findings including visual summaries of Study 2 results.

Study 1 found significant reciprocal links were found between negative affect and BMI and Study 2 found that brooding rumination (a form of repetitive negative thinking) links mental and physical health as it explains variance in both obesity and depression. Further, rumination and waist circumference had a significant effect on attention and inhibition errors and neurovascular activity. Findings are discussed in relation to the proposed theoretical model and suggestions are made for model amendments and future research (see Chapter 9).

1.0 Introduction

Adiposity and Negative Affect (Physical and Mental Health)

This chapter summarises the motivation to study the different ways that physical and mental health affect each other using insights from a key paper that influenced this thesis (Forsman et al.,2015); in short so that health research and care better reflects peoples' real lived experience in that mental and physical health needs are not separate entities. The following subsections outline key issues identified from the Forsman paper and a related paper by Schumann et al., (2014).

Physical and mental health are not considered together.

"The difficulty lies not so much in developing new ideas as in escaping from old ones." -- John Maynard Keynes

The social and economic impact of mood or affective disorders far exceeds many physical illnesses (Simon, 2003), but people face a challenge in proving the effect on their health and well-being is real. The rise in obesity levels has been dubbed an international epidemic, whereas the steady rise in mood disorders and suicides since the 1980s has attracted much less attention (Twenge et al., 2019). In 2014, a large consultation of public health experts, the Roadmap for Mental health Research in Europe (ROMER) project, called for large scale reform in the research of mental health conditions (Forsman et al., 2015). The project made recommendations (principles and goals) for far reaching changes to improve mental health research, including a lifespan approach to identify causes, risks, protective factors, and processes. A fundamental requirement to achieve this goal was better integration of physical and mental health research, including interdisciplinary

conceptual definitions, developing high quality novel frameworks, and use of robust standardised measures to move science forward.

> "*The complexity of mental health … requires complementary research approaches and interdisciplinary collaboration to better serve the needs of the European population*." (Forsman et al., 2015), p250)

Attempts to understand mental health through the lens of the diagnostic symptom clusters of the medical model (DSM/ ICD manuals) have long been critiqued (Shah & Mountain, 2007). Focusing on the pathology of mental health problems precludes research of mental wellness factors, fuels stigma and leads to an emphasis on somatic, pharmacological treatments which do not fully address the complex psychosocial causes. To illustrate this point, three out of the five causes of the socioeconomic burden associated with mental health problems are identified as **stigma**, **under-detection,** and **under-treatment** (Forsman et al., 2015), so the argument for a new approach to understanding health is extremely compelling.

Biomedical research is not being effectively translated into mental health research, and clinical practice.

> *"This dissociation between **basic science** and **clinical experience** is a major barrier for translation of findings from basic to applied research, assessment and intervention." (Schumann et al., 2014, p.26)*

Schumann et al. (2014) underlines the importance of interdisciplinary approaches to mental health research, not least because those with severe mental disorders are much more likely to suffer from somatic illnesses. Their paper gives a 'state of the art' overview of biomedical scientific findings relevant to mental health. Table 1 for a summary of key points from the paper with section 2,3 and 6 being

most relevant to this thesis along with the call for research into the shared symptoms and associations between physical and mental health conditions.

> "A greater understanding of the associations between clinical constellations of symptoms of both psychiatric and somatic disorders as they develop over time …is needed" (Schumann Pg33)

An integrated biomedical understanding of symptoms linking physical and mental health, and how they develop over time would be beneficial to advancing health outcomes and pave the way toward more effective and personalised medical care. The paper emphasises insights into the role of the brain as a cause or indicator of health problems. Integration of neurological and psycho-physiological knowledge (i.e. brain function, behaviour, and bodily measurements of their effects) was identified as a step toward addressing the disconnect between scientific understanding and clinical practice (Dalgleish et al., 2020; Schumann et al., 2014). The work is rooted in key ideas from the Research Domains Criteria (RCD) project by the National Institute of Mental Health (Insel et al., 2010). The RDC matrix (NIMH, n.d.) seeks to add validity and reliability to medical diagnoses by ensuring they are better aligned with physiological structures and existing research findings. Knowledge of recent biomedical findings can help ensure that mental and somatic research is moving forward together and not focusing on redundant concepts or low impact areas. Schumann indicates that more fruitful areas of study are those that are supported by multiple avenues of research.

One example of a theory which is supported by multiple studies of observed behaviour and neuro-physiological indicators as well as evolutionary theory (see Table 1) is the transdiagnostic approach to mental health. This approach is still developing, however interventions from this type of 'joined-up' science are showing

promise. Dalgleish et al. (2020) identify five challenges for multi-perspective approaches, like transdiagnostic science, which complement issues identified by Schumann et al., (2014):

1. Development of theoretical models that are not bound to existing diagnostic frameworks.
2. The mental content (negative thoughts, dysfunctional beliefs) needs to be linked to and understood alongside the neurophysiological processes.
3. Movement away from traditional diagnostic frameworks to allow radically different alternatives to be considered.
4. Developing fit-for-purpose research methodology including hybrid designs and multiple co-primary outcomes that spell out the underlying mechanism of change, including intra-individual patterns.
5. Prioritising cross-disciplinary joined-up thinking in health research.

Table 1

Biomedical research relevant to physical and mental health

Core state of the art concepts and biomedical research areas that should be considered in relation to physical and mental health, summarised from an expert consultation paper by Schumann (2014). Yellow highlighted areas are particularly relevant to this thesis.

Core Biomedical Concepts That Interface Between Physical & Mental Health	Health Research Challenges &Opportunities
1. Arousal and Stress Regulation Biomedical Research Areas	
• Chronic stress and vulnerability to psychiatric disorders • Stress and arousal mediators: action mechanism.	Identifying • Resilient or vulnerable groups • Stress / arousal mediators and action mechanism.
2. Cognitive Processing Systems Biomedical Research Areas	
• Perceptual dysfunction and electrophysiological biomarkers • Attention and neural networks of cognition. • Conscious/unconscious processing • Top-down vs bottom-up processing. • Working memory and cognitive training • Long-term memory and plasticity • Decision-making, learning and neurocomputation deficits. • Metacognition, Executive Function, and social cognition	• Identifying subgroups (cognitive phenotypes) within different disorders to enable a better understanding of what treatments will work and for whom. • Neural basis of attention and the default system which is deactivated during most cognitive tasks. • Use of network analysis and functional connectivity to investigate neurological function. Investigations of: - Default Mode Network in cognitive processing deficits. - Neurocognitive changes that may signal different endophenotypes. - Anormal patterns of electrical brain activity (attenuated or absent mismatch negative (MMN) potentials to auditory, and even visual, stimuli).

Core Biomedical Concepts That Interface Between Physical & Mental Health	Health Research Challenges &Opportunities
3. Positive and Negative Valence Biomedical Research Areas	
Evolutionarily essential states (reward and punishment) which underlie approach and withdrawal behaviour. • Transdiagnostic approach. • Research Domain Criteria (RDoC) Insel et al., (2010). • Positive valence: - approach motivation, - responsiveness to reward (initial/sustained) - reward learning and habit. • Negative valence: - active threat ("fear"), - potential threat ("anxiety"), - sustained threat, loss and frustrative non-reward.	• Different brain cells in behaviour regulation (e.g., astrocytes, microglia, immune cells). • Neurochemical and cognitive pathways in processing of fear, anxiety, and long-term hypervigilance to stress. • Reward and punishment as behavioural motivators in different conditions. • The effect of habit formation. • Common anxiety mechanisms of the fear/anxiety circuitry (under studied). • Addictive behaviour as a form of self-medication to protect from negative thought processes.
4. Systems For Social Processes Biomedical Research Areas	
• Neurobiological/ neurochemical understanding of bodily Systems for Social Processes (SSP) e.g., - Sensory perception and feedback: Hearing, Touch, Eye contact. - Sense of self, Imitation, - Empathy (theory of mind), - Recognition of emotional expression • Behavioural systems (motion, human action, goal-directedness) • Language	• Sensory processing deficits in relation to social interaction and how that may be linked to mental health issues. • Disconnect between classification systems (ICD/DSM; largely based on clinical opinions) and current knowledge in SSP neurobiology and neurochemistry.

Core Biomedical Concepts That Interface Between Physical and Mental Health	Health Research Challenges &Opportunities
5. Pharmacological Treatments Biomedical Research Areas	
• Clinical pharmacology and drug development • Symptom clusters • genetic/epi-genetic biomarkers • Animal models	• Need Integrative approaches to examine the neurobiology and pharmacology of the endophenotypes, e.g., via translational neuroscience and experimental medicine. Areas include: - the biology of affect (negative and positive valence), - arousal and regulatory systems, - social processes and cognition.
6. Psychiatric Somatic Comorbidity Biomedical Research Areas	
• Mental health conditions and their relation to somatic disorders, namely: - depressive disorders, - anxiety/PTSD, - health behaviours (alcohol/ smoking/ diet), - schizophrenia/bipolar, - medication-related disorders. • Potential causal links between mental disorders and somatic diseases: - shared predispositions (genetic, temperamental and personality traits), - shared risk factors (stress, trauma, food intolerance, lifestyles, social support, negative emotions) or, - shared mechanisms (coping, resilience or defence mechanisms, endocrine and immune disruption) • Mechanisms - Allostatic load theory (chronic exposure) - Inflammation - Stress and HPA axis. - Health behaviours	The mortality gap increase: Overarching need to establish new research to help reduce the mortality gap and negative health impact, plus better understand the causal relation between comorbid diseases (Schumann proposes allostatic load as an overarching theory to explain the causal relationships between physical and mental health comorbidities) P35. Investigating negative co-morbidity (i.e. studies where variables are showing the opposite relationship than expected) to understand mediating/ protective factors. Problems with existing studies: • cross-sectional epidemiological studies • inaccurate clinical diagnosis • clinical studies of patient cohorts afflicted by Berkson's bias (creating a false dependency between variables due to the test group selected(de Ron et al., 2021)) or with • insufficient clinical or biological information about participants

Note: Coloured and highlighted areas are most relevant to this thesis

Having established that mental and physical health need to be explored in a more unified way, Schumann highlights common mental health conditions that frequently co-occur with physical health conditions, and potential causal links and mechanisms indicated by biomedical research (Table 1 *Biomedical Research Relevant to Physical and Mental Health and Comorbidity*). Conditions that show evidence of mental-somatic health comorbidities include depressive disorders, anxiety/PTSD, and health behaviours (colloquially considered “bad habits” which can negatively impact health) such as consumption of alcohol, smoking, sedentary behaviour or eating a diet. Loneliness is a further condition which could be added to this list as it is associated with detrimental physical and mental health effects like heart disease, type 2 diabetes, depression, dementia, and earlier mortality (Christiansen et al., 2021). A high-profile physical condition linked to several of these areas is obesity (see Chapter 2 Physical Health) and Chapter 3 examines the link between obesity and loneliness over time.

Studying the Interaction of Comorbid Conditions

Randomised clinical trials (RCTs) and systematic reviews or metanalyses that summarise these are considered the gold standard method to research cause and effect in health research (Evans, 2003; Murad et al., 2016). In accordance with empirical methods, research trials seek to examine the outcomes of a test group compared to a control group, but the more variability in the sample the lower the confidence that the experimental effect (be that drug trials or observed outcomes) are caused by the experimental variables. RCTs understandably aim to reduce bias and participant variability, but this frequently means that i) comorbid conditions and

certain groups of participants are under researched and are less likely to get optimal health treatment; ii) studies that seek to triangulate findings across different methodologies or disciplines may have more power to 'bring the science on' (in line with Schumann's 'state of the art' concepts), but triangulation of methodologies is not given systematic consideration in the way that RCT meta-analyses are. Examples of groups excluded from RCTs include women (legally required to be included in government funded drug trials only from 1993 and still commonly excluded from trials (Ravindran et al. (2020), left-handed people (screened out of neurological studies; Willems et al., 2014), adolescents and the elderly (Crome et al., 2011; Noel et al., 2021; Pitkala & Strandberg, 2022). Studying co-morbid conditions across the lifespan (as advocated by Forsman, 2015) could give opportunity for parity of care across mental and physical health and more equitable division of health research between the areas. This may not mean including groups in every trial but ensuring that all age groups are researched.

A further methodological issue which has extra impact in studies of comorbid health conditions is that measurement and definitions of conditions can vary widely. Measurement issues (for example definitions of overweight versus body mass versus fat mass– see Chapter 2 variable measurement) affects the extent that previous research can be relied upon and compared. In a study of co-morbid conditions that aims to understand causal links, it is important to carefully establish definitions and be clear about validity – what exactly is being measured, and reliability of that measurement (Schumann et al., 2014). Taking this a step further, and tied to the issue of impactful research, is the matter of when a significant association between variables is detected, does it have clinical importance and meaningful impact on a person's lived experience or life quality? (Abbott & Hart,

2005). Too many studies only focus on physical health outcomes, ignoring essential wider health and well-being indicators such as functional cognition, and mood (Zubritsky et al., 2013).

Impactful Research: Relevant To Real-Life Outcomes and Mechanisms of Effect

Cognition could be an important mechanism of effect between mental and physical health. Co-morbidities associated with depression, anxiety and loneliness overlap with that of obesity and are also associated with cognitive effects. For example, there is a body of work that indicates that people in the morbidly obese weight category may experience more cognitive issues than normal weight controls (see Chapter 2 Study variables and Chapter 5 Study 2 Introduction). Negative affective conditions are known to be related to thinking biases that perpetuate negative affective symptoms (Grafton et al., 2012; Spithoven et al., 2017). Additionally, these conditions are related to Executive Function problems that can interfere with the ability to conduct everyday tasks (Cacioppo & Hawkley, 2009; Warren et al., 2021). Results vary and there appears to be a lack of evidence as to the extent of the cognitive difficulty these conditions impose (individually or as co-morbidities). However, the ubiquity of cognitive effects across these conditions could indicate a potential mechanism of effect in linking physical and mental health. Clarification of the extent of cognitive impact may add important context to the lived experience of people with these conditions.

Summary/conclusion

To make progress in the effective understanding and treatment of mental health conditions, research needs to develop ways to integrate our understanding of mental and physical health, by bridging the gap between biomedical science, psychology, and clinical practice. Methods to investigate comorbid mental and physical health conditions should be carefully considered, reflect cross-disciplinary best practice, and seek to ensure that the constructs of interest have tangible impact on people's lives. Obesity and mood / affective disorders (including depression, anxiety, and loneliness) are prevalent comorbid conditions of international public health concern that may benefit greater understanding of their shared features. This includes their neurocognitive components of repetitive negative thinking, and Executive Function that may impact functional daily living and health.

General aims

This thesis looks at the intersection of mental and physical health through examination of the associations between excess weight (body mass, obesity, or excess adiposity), and negative affective disorders though their relationship with functional neurocognition. The following chapter will outline the key variables in the study including measurement considerations and research findings.

2.0 Variables in the thesis: Obesity, Negative Affect and Cognition

Physical Health	Mental Health	Neurocognitive Function	Demographic Variables

This chapter provides definitions, causes and measurement issues surrounding the main variables discussed in the thesis (see Table 2 Health Variables and Measures in Chapter 2).

Table 2

Health Variables and Measures in Chapter 2

Broad health area	**Physical Health**	**Mental Health**	**Neurocognitive Function**	**Other**
Topic	*Obesity*	*Negative affect*	*Executive Function*	*Demographics*
Variables	BMI Waist Circumference Measurement of Adiposity	Depression Anxiety Loneliness Repetitive Negative Thinking	Attention Inhibitory control Neurological scanning Self-reported executive function	Age Gender (sex) Socioeconomic Status Education Ethnicity Handedness Medication General Intelligence

Physical health – 'Obesity - The global epidemic'

Causes	Measurement	Adiposity Health Outcomes

Obesity is a term to describe a state of excess body fat – defined as a Body Mass Index (BMI, Kg/m^2) of greater than 30 (WHO, 2000). There has been debate over whether to classify obesity as a disease (Rosen, 2014; Wilding et al., 2019). Regardless, it is a high priority public health concern, as it is a major risk factor for

cardiovascular disease, metabolic diseases and musculoskeletal problems which impact life experience, healthcare costs and increase the chance of premature death (Bluher, 2019; Flegal et al., 2013; Jayedi et al., 2022). Obesity is an international priority for health action because it has undergone an increase in prevalence since the 1980s (NCD-Risk Factor Collaboration, 2017).

Although obesity is regarded as a physical health condition, it is heavily bound in socio-emotional issues such as negative stereotypes. It is often regarded as preventable and a choice (Puhl & Brownell, 2001; Puhl & Heuer, 2009) despite strong genetic heritability (Thaker, 2017). Figure 2 shows Google auto search options which reflect the prevalence of some of these concepts in common search trends (Google, 2020).

Figure 2
Google Autofill Frequent Search Terms for 'Obesity Is…' July 2023 (Google, 2020)

Causes of obesity

At a population level the cause of obesity is explained as the result of lifestyle factors which predispose us to increased calorie consumption e.g., wider availability of energy-dense foods, and reduced calorie expenditure, e.g. increased sedentary

work and use of transportation (WHO, 2021). On an individual level, like mental health conditions, the causes of obesity are multifaceted, resulting from genetic predispositions, life-experience risk factors (eating habits, parenting style, stress, trauma, lifestyle, social expectations). A series of genetic twin and experimental studies which investigated appetitive traits controlled by the FTO gene/ hypothalamus (Llewellyn et al., 2010) culminated in the Behaviour Susceptibility Theory (BST; see Figure 3) which explains the genetic and environmental risk factors that lead to obesity (Llewellyn & Fildes, 2017).

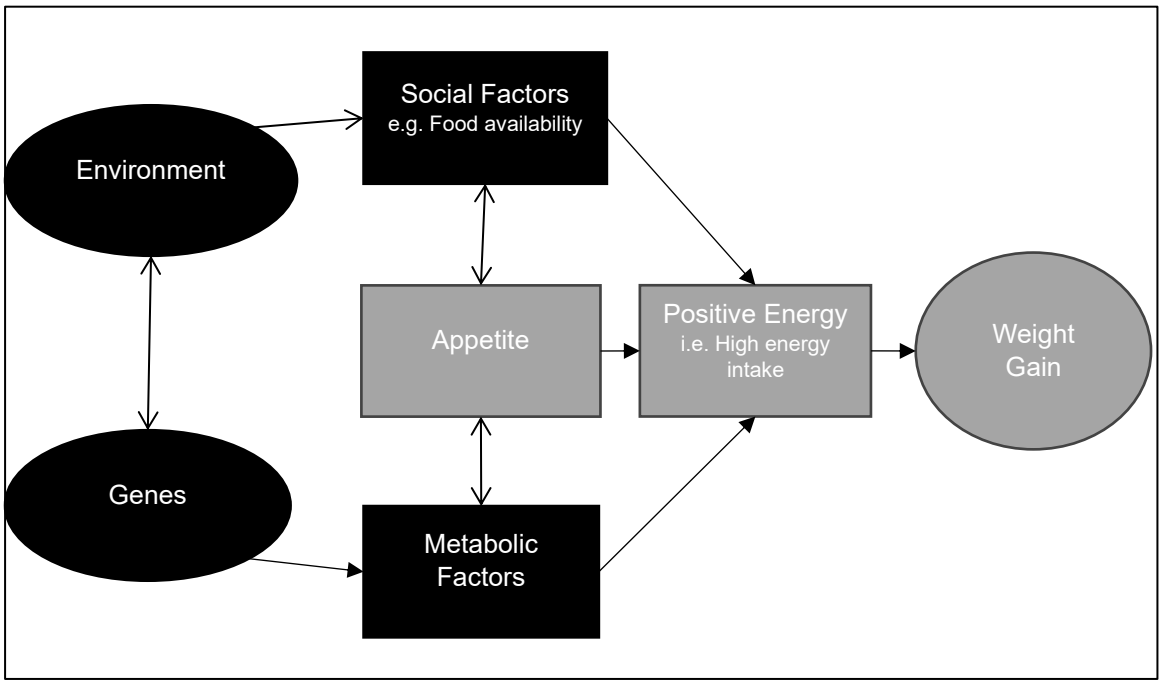


Figure 3
Behaviour Susceptibility Theory (Llewellyn & Fildes, 2017)

Further causal factors include medication (NICE CKS, 2023) and biomedical factors, such as endocrine/immune disruption (Safaei et al., 2021) which influence hunger and metabolism. Links to some maternal behavioural factors have also been found, e.g., maternal smoking in pregnancy (Weng et al., 2012). Obesity is observed

to track from infancy and childhood into adulthood (Lobstein et al., 2004) which is commensurate with a high genetic component.

Measurement of Excess Fat – a Discussion

The relationship between obesity and other health conditions has been researched widely but methods vary, and interpretation is not straightforward. Although the term obesity is frequently noted as a health risk, the actual substrate of the risk to health is purported to come from excess circulating body fat. There is a good deal of estimation involved in the process from the definition and measurement of excess body fat to the calculation of risk to health i.e. how likely is it that someone with excess fat will develop a health condition. This section will outline key issues and measurement techniques used to determine obesity or excess body fat.

With modern techniques it is possible to measure the direct quantity of fat tissue in each cavity of the body (e.g. quantity of visceral adipose tissue) however in most cases anthropometric (bodily) measurements are used to estimate the amount of fat tissue in relation to lean (non-fat tissues) and techniques can vary widely. Fat estimates can be represented in many ways depending on the tissue which is measured e.g., percentage fat mass, percentage adipose tissue or ratio of fat to lean mass. The quantity of fat tissue can be measured directly or extrapolated from or body mass (such as Kg/m2) or Waist Circumference (WC). As in any area of science the measurement of variables is important in determining validity of the constructs being tested and comparing studies within a body of work.

Direct measurements of fat mass are considered gold standard, and these techniques are used as references to evaluate other measures. In the last 20 years the gold standard measures in anthropometry have progressed from CT to MRI (or a

combination of the two) so you can find studies where CT was used to validate MRI and vice versa. This adds to the complexity of evaluating the measures. With interrogation of the literature, it becomes clear that all fat measurement techniques have limitations and even gold-standard reference measures are subject to measurement bias. As noted, despite advancements in body scanning, most fat measurement techniques used in health research and clinical practice still rely on estimation from anthropometry (measurements of the body).

The relative advantages and disadvantages of a range of methods were examined from the extant literature. With such a wide range of anthropometric methods used in research it is difficult to compare 'like with like' but two important issues are:

i) How well the technique approximates fat mass or 'excess adiposity' (direct measures, estimates based on average formulae or proxy measures based purely on body size).

ii) How effectively the cut-off points within that measure (e.g. BMI categories or percentage fat mass etc.) predict the risk of ill-health (usually cardio-metabolic).

Criteria that influence the selection of fat measures in both research and clinical settings include the expense and accessibility of the measure, safety, time/expertise required and participant comfort in delivery. See Table 3 for an overview of the relative features and draw backs of the most common fat measurements.

Table 3

Comparison of Fat (Adiposity) Measurement and Estimation Techniques

Measure	Means	Direct Estimate or Proxy	Regional or Full body	Reliability /Validity	Cost	Accessibility	Portability	Safety precautions	Time	Issues for patients
Magnetic Resonance Imaging (MRI)	Magnetic field of hydrogen/ water (fat water imaging/ fat referencing MRI by voxel)	Direct [or sampled]	Fb or R	AAT vs dissection MD=.076kg (CI -.005kg to .147kg); CV .4-13.7% Abate et al.,1994	***	*	*	***	**	Stillness
Computed Tomography Scan (CT)	3D X-ray (cross-sectional imaging of bone, muscle, fat volume)	Direct [normally sampled due to radiation	Fb or R	Ref: BMI SAT f .88; m.83 VAT f .75; m.71 Fox et al. (2007)	***	*	*	***	**	Ionising radiation
Dual energy X-ray absorptiometry (DXA)	2D X-ray (image mineral bone vs soft tissue)	Direct, but tissue type estimated from anatomical models	Fb or R	Ref = FR MRI Wb AT CV=4.5, *r*=.99 VAT CV>20% Borga et al. (2018)	**	**	*	**	*	Ionising radiation
Hydrodensitometry	Underwater weighing	Estimated composition from body density (assuming a consistent fat to lean ratio and density)	Fb	Ref= DEXA CV % BF=4.8% (3.8-6.6%) Pritchard et al. (1993)	**	*	*	**	***	Full water immersion
Air Displacement Plethysmography (ADP) 'Bod Pod'	Air displacement	Estimated composition from body density	Fb	Test-retest Reliability CV 1.7 to 4.5% Body fat. Fields et al. (2002)	**	*	*	*	*	***
Bioelectrical Impedance Analysis (BIA).	Weak electric current (conductance of fat mass/ non-fat mass)	Estimated composition from electrical resistance	Fb	Ref MRI VAT *r*=.40 to.78 DXA TAT *r*=.91-.97 Pietiläinen et al. (2013)	*	***	***	*	*	***
Skin fold measurement	Skin fold measurement	Estimated composition from subcutaneous fat	Fb	SF% fat Calc: Durnin & Womersley 1974 BMI: partial *r*=.68 to.84 Ahmad et al. (2013)	*	***	***	*	**	***
Body Mass Index (BMI)	Height and weight measures (weight kg/height m^2 or $cm/100^2$)	Proxy: Fb body mass	Fb	Ref. BIA %BF BMI 25-32: *r*=m.38; f=.40 BMI 18-25: *r*= m .21; f=.38, Meeuwsen, Horgan & Elia, (2010)	*	***	***	*	*	***
Waist Circumference (WC)	Waist measure	Proxy: Central adiposity	R	Ref. MRI TAT R2=.92 Ref CT, VAT *r*=f.78, m.73 Ross et al. (1992)	*	***	***	*	*	***
Waist to hip ratio (WHR)	Waist Circumference divided by hip circumference.	Proxy: Central adiposity	R	Ref.: MRI VAT R2=.85 Ross et al. (1992)	*	***	***	*	*	***

Notes: Ref.=Reference measure; Fb= Full Body; *R*= Regional; TAT=Total Adipose Tissue; VAT =Visceral Adipose Tissue; SAT=Subcutaneous Adipose Tissue; *r*=correlation; m=male, f=female Indicative Relative Star ratings: *= Low **=Moderate ***=High

Fat mass: Methods of measurement and estimation. Techniques that come closer to direct measurement of body composition are regarded as the best measures of body fat for health as fat quantity and its mechanical and biochemical effect on the body is the proposed cause of most weight-related health problems. Despite this, even direct scanning techniques have drawbacks in terms of what is being measured, expense, ease of use, participant comfort and safety. The strengths and limitations of the main adiposity measurement techniques are outlined below (see Table 3 for an overview).

Computed Tomography (CT) and ***Magnetic Resonance Imaging (MRI)*** are currently accepted to be the gold standard reference measures of body fat (Borga et al., 2018). CT (an X-ray scan) allows a 3D image to be built up from cross-sectional body slides, whereas MRI uses the magnetic properties of elements in body cells (e.g. hydrogen) to image the body ('fat water imaging'). Fat Referencing MRI can be used to take direct measurements of adipose tissue (which is around 80% fat) or triglycerides (fat cells) in the body. Fat composition can be reported as a percentage, or a ratio of fat mass compared to lean tissue.

A large advantage of MRI and CT is the capability to measure regional fat distribution, which can be more predictive of metabolic conditions than a total fat measure (Total Adipose Tissue or TAT). Visceral Adipose Tissue (VAT) in the abdominal cavity for example and ectopic (abnormally positioned) fat are more predictive of cardiac risk, type 2 diabetes, liver disease and cancer (Gallagher et al., 2000; Britton et al.,2013; Borga et al., 2018; Fox et al.,2007) due to their influence on hormones, insulin resistance and dyslipidaemia (van Kruijdik et al.,2009). Visceral fat is more likely to be associated with ill-health because it is more likely to influence

hormones change, dyslipidaemia (increased free fatty acids), and systemic inflammation (Chan et al., 2004; Ebbert & Jensen, 2013).

Accuracy of body fat reported from MRI and CT can vary as full body scans are rarely undertaken, and measures can be estimated from as little as one body slice (reducing their reliability). Further drawbacks of these techniques include the high cost and non-portability of scanners (often confined to teaching hospitals and well-funded research labs) plus analysis of MRI is time consuming and both methods require considerable expertise and safety precautions. Participants must remain very still during the scan (which can be challenging for some groups), and CT emits ionising radiation that makes the technique even less suitable for at-risk individuals (e.g., children and those in pregnancy) and for repeated usage within short time scales.

Dual-energy X-ray absorptiometry (DXA) provides a more accessible and less expensive direct body measurement option. DXA is a 2D X-ray scan technique which measures the volume of bone mineral and soft tissue by the degree of photon attenuation. DXA equipment is costly, but it is cheaper and more readily available than MRI and CT and requires less expertise to operate. Radiation exposure is also lower than for CT. A drawback of DXA, however, is that it relies on body compartment calculations to differentiate between soft tissue types. Borga et al. (2018) found that DXA and MRI produced comparable results in measuring body fat in a large sample of 40- to 69-year-olds, however DXA was a less reliable measure of visceral adipose tissue in obese participants than lean (Borga et al., 2018; Toombs et al., 2012) which is problematic for studies of excess weight. Measurement between different machines can also be inconsistent (Toombs et al., 2012).

Body Composition: Estimation. Before the advancement in scanning, indirect body composition estimation was used to calculate body fat from careful measurement of body density. The calculations vary in their reliability because they use notional or averages that lead to under or over estimations depending on participant characteristics (including age group, body size, fitness level, gender etc.). Calculations use the average ratio and assumed density of chemically distinct body 'compartments' (Withers et al., 1998) for example, the 2 compartment (2C) method uses the assumed density of fat mass (.90g cm^{-3}) and lean or fat free mass (1.1g cm^3) to estimate whole body fat quantity or ratio. Over the years the calculations have been expanded to include additional body compartments e.g., the 4C method uses average FM, Total Body Water, Bone Mineral Mass, plus a residual (Brodie et al., 1998; Withers et al., 1998).

Hydrodensitometry and Plethysmography . Techniques to measure body density include Hydrodensitometry (water displacement; whole body submersion in a water tank) and Plethysmography (ADP; air displacement measured inside a 'bod pod'). Hydrodensitometry is the more accurate technique, but it can be difficult for participants to endure whole body submersion, so it is considered impractical for most medical and research applications (Brodie, Moscrip & Hutcheon, 1998). The estimated standard error of Hydrodensitometry is up to 2.7%, but this is largely due to the averages used in the compartment calculations (Lohman, 1984). ADP is relatively affordable and simple for participants but when compared with DXA, ADP significantly overestimated the body fat percentage in underweight participants (6-7% higher for those with BMI<18.50) and underestimated that of normal and

overweight/obese participants (around 2% with BMI >18.50; Lowry & Tomiyama, 2015).

Bioelectrical Impedance Analysis (BIA). Estimates percentage Body Fat (%BF) using electrical resistance (impedance value) by return of a weak electrical current passed through the body. BF conducts electricity less effectively than other body tissue, so impedance value is used along with height to estimate the proportion of Lean Mass and Total Body Water and Fat Mass. A big advantage of the technique is that it is fast and non-invasive. Researcher calculations were found to be superior to automatic BIA device calculations which lack reliability and are not recommended (Franco-Villoria et al., 2016). BIA is very sensitive to changes in body state such as temperature, recent eating, and exercise so it is vulnerable to measurement error. Reliability of the technique is improved when adjustments are made for demographic variables such as age, gender, and ethnicity (Borga et al.,2018).

Skin fold measurement is a further body fat estimation technique which uses callipers to measure the thickness of skin folds on several areas of the body to estimate body density. The target areas are different for males (chest, thigh, and abdomen) and females (triceps, thigh, and just above the hip bone). The multiple sites of measurement introduce greater opportunity for measurement error. As with all estimation measures, the formula assumes average values, however the method is cheap and portable.

Proxy Measures of Adiposity

Proxy measures do not measure or estimate BF but instead rely on body measurements which are correlated with BF and cardiometabolic health outcomes.

Body Mass Index (BMI). The most common proxy measure of adiposity is Body Mass Index (BMI; weight kg/height m^2 or $cm/100^2$). It is often used as a fast screening-tool to identify individuals at risk of cardiometabolic problems more effectively than by observation or by weight alone. BMI can be used as a continuous measure, but often it is summarised as weight categories (underweight, normal weight, overweight, obese) based on 'cut-offs' that are said to reflect grades of risk of ill health (see Table 4). The World Health Organisation and International Obesity Task Force (IOTF) recommend that the optimal range of BMI is between 21-23 kg/m^2 for Caucasian/European populations (WHO, 2000).

Table 4

International Obesity Task force (IOTF) – European Obesity Cut offs and Associated Risks of Co-Morbidities Based on WHO Guidelines Https://Www.Worldobesity.Org/Data/Cut-Points-Used/)

IOTF Classification Categories		**Coding**	**Risk of co-morbidities**
Waist circumference			
Men ≤94 cm & Women ≤80 cm		0	Average risk
Men >94 cm & Women >80 cm		1	Increased risk
BMI (kg/m²)			
Severe underweight	<16 kg/m2	-1	
Moderate underweight	16·0–16·9	-1	
Mild underweight	17·0–18·49	-1	
Underweight	18.5	-1	-1 Low/Other*
Normal+	18.5 - 24.9	1	1 Average risk
Overweight	25.0 - 29.9	2	2 Increased risk
Obesity Class I	30.0 - 34.9	3	3 High risk
Obesity Class II	35.0 - 39.9	-	4 Very high risk
Obesity Class III	≥40	4	4 Very high risk

Note: *-1LowOther*= low risk in relation to adiposity related comorbidities, but increased risk of other clinical problems*

BMI is a controversial measure. The term is quite ubiquitous but the relationship between BMI and ill-health is more nuanced than most people realise. Prevalence rates of ‘overweight’ are often reported synonymously with ‘obesity’ but these categories do not carry the same association with ill-health, and effects are not always linear (Lawlor et al., 2006; Lawson McLean et al 2019). Assumptions about BMI and weight categories and the risk of ill-health are more problematic because there is a large stigma associated with weight and BMI (Puhl & Heuer, 2010) which can bias attitudes and affect research priorities and clinical outcomes. There has been a call for more discussion of factors that influence whether a person is or is not healthy at a given weight (such as the role of visceral versus subcutaneous fat in health risk; Hubbard, 2000).

It is important that clinicians and researchers are aware of the limitations of the measure and that any findings are reported clearly. It is often highlighted that BMI cannot discriminate between muscle and fat mass (Rothman, 2008). Body scans are certainly more accurate in differentiating between fat mass and lean mass, however in meta-analytic comparison (Sommer et al., 2020) BMI is quite good at identifying individuals who are *not* obese (true negative; specificity in males = 97%, females=95%;). The main drawback of BMI is failure to identify those who *do* have excess body fat (false negative; sensitivity in males =50% , in females =51%), indicating a lack of sensitivity (true positive rate) compared to gold standard body composition scanning (Okorodudu et al., 2010; Sommer et al., 2020).

Efficacy of the measure. BMI sensitivity estimates vary depending on which cut-off band is investigated. Okorodudu et al. (2010) defined excess body fat percentage as >=30% for females and >=25% for males. They carried out a systematic review and meta-analysis of studies that compared BMI with body fat

composition measures. The sensitivity of BMI to detect excess fat for the obese category (Obese BMI>30.00) was .42. Conversely, the specificity of BMI (true negative rate) was estimated to be good (.97) so 97% of cases who **do not** have excess body fat (f=<30% BF; m=<25%BF) are categorised correctly. More than half of individuals with excess body fat are not identified through BMI alone, however body fat percentage is not as predictive of ill-health as the pattern of regional distribution (particularly visceral fat in the abdominal cavity).

Central Adiposity. As research increasingly shows the importance of fat ***distribution*** in the body, greater attention has been given to measures of 'central adiposity' and abdominal fat. **Waist circumference (WC)** is measured at the halfway point between the hip bone (iliac crest) and lower rib (or the umbilical WC when the rib/hip bone cannot be isolated; WHO, 2008b, p5). Other methods include taking the measure at the top of the iliac crest (per major US health study NHANES III) or the point of minimum waist, but different protocols are not judged to substantially affect the association with health outcomes (Ross et al., 2008). Waist-hip ratio (WHR) is WC divided by hip circumference (taken at the widest part of the buttocks). WHR appears to be more affected by gender differences than WC (as males put on more weight in their mid-region than their hips) so the measure appears to be a more helpful indicator of ill-health risk in females than males (Li et al., 2006). WC requires fewer measurements which is less time consuming for the participant and reduces opportunity for error. WC also has a larger research base than WHR (Sommer et al., 2020) and the WHO Asia Pacific report states WC is the preferred measure of central obesity (WHO), however meta-analysis and review (Sommer et al., 2020)

found no evidence that WHR is inferior (WC sensitivity in males=62% females=57% ; specificity in males= 88%, in females 95%).

A recent meta-analysis (Sommer et al., 2020) compared the efficacy of BMI and measures of central adiposity found proxy measures were less sensitive than references (49-51% for BMI and 57-62% for WC with slight gender differences), however including a measure of central adiposity appears to explain separate and possibly additional variance in health outcomes when BMI is controlled for (Pischon et al., 2008; Browning et al., 2010; Sommer et al., 2020). Clinical guidelines suggest that health risk due to adiposity is better approximated using both measures of BMI and WC (NICE, 2014). The anthropometric methodology was devised with reference to WHO and NICE clinical guidelines using International Obesity Task Force (IOTF) cut-offs (see Appendix G4 Anthropometric Procedures).

Adiposity Measurement Summary. In summary, body compartment measures and direct measure sampling provide a trade-off between time/ resources and accuracy of the measurement, however, estimation techniques (such as body mass and WC) are still the most prevalent methods used to gauge excess adiposity. When interpreting research on health risk associated with body fat, different fat measures (fat mass, percentage/ ratio measure) have different associations with cardiometabolic risk, and different cut off points or values of the measure related to levels of increased risk. Additionally, some measures have more of a research base than others which is necessary to enable reliable health-risk cutoffs.

Adiposity Measurement and Health Outcomes

A review of longitudinal cohort studies of the association between health/ mortality risk and fat proxy measures (BMI and central adiposity; Carmienke et al.,

2013) found differences in their relationship to health outcomes. The precise association between adiposity measures and health outcomes are important considerations in measure and analysis choices. Cardiometabolic health risks had a positive linear relationship with WHR, a J-shaped association with WC (increasing above the established cut-offs) and a U-shaped risk with BMI (BMI between 25 and 35 reflected reduced health risks). Mediation analysis of the relation between BMI and mortality indicated that the main mediators of health effects were indicators of worse glucose, lung, and renal function (Ghulam et al., 2023).

A meta-analysis of measures of body fat (measured by CT, DXA and BIA) and health (Jayedi et al., 2022) found: a J-shaped association between percentage body fat/fat mass and all-cause mortality risk (i.e., an earlier risk of death than those of the same age and gender); Lowest health risk being at 25% of body fat (20kg fat mass) but a significant increase in risk at 35% body fat (all-cause-mortality relative risk of 1.02 at 30% to 1.35 at 40% and 1.98 at 50% BF).

Summary

- Obesity is a grade of body mass (BMI), a proxy measure for excess body fat, which is correlated with metabolic health problems.
- Obesity is a value-laden term which gives the impression that being physically bigger is unhealthy whereas the true picture is more nuanced, depending on the quantity and location of fat tissue (or circulating free fatty acids).
- BMI can fail to identify individuals that are at risk of ill-health because it is not sensitive to the distribution of fat in the body.
- Waist circumference (WC) is recommended in addition to BMI to give an indication of visceral fat. This is still a proxy measure (not a direct measure or

estimate of body fat) but the sensitivity of the measure is slightly higher than for BMI.

Mental Health

Negative Affect	Depression	Anxiety	Loneliness	Repetitive Negative Thinking

Negative Affect

Depression, anxiety and loneliness are among the most common mental health conditions and can be experienced on a continuum from transient emotional states to chronic, clinical mental health conditions. The severity of the condition is generally assessed based on self-reported symptoms through a diagnostic interview (for depression or anxiety) or questionnaire. Core components of these conditions include internalised feelings of emotional distress or negative affect (Stringer, 2013), cognitive difficulties (such as attention and memory issues) and repetitive negative thinking (Ehring & Behar, 2020). The chronic courses of these conditions are linked to worse physical health (Christiansen et al., 2021; Horenstein & Heimberg, 2020; Park et al., 2020; Richard et al., 2017).

In this thesis, depression, anxiety, and loneliness will be collectively referred to as negative affective conditions, referencing the common experience of emotional distress. Separate definitions and measurement considerations are provided, reflecting the separate research and measurement literature base, however the argument for negative affect as a transdiagnostic grouping is also outlined, including consideration of loneliness as a related negative affective construct.

Causes of Negative Affective Conditions. Developmentally, depression and anxiety in adolescence have been shown to arise from two main pathways, early temperament (emotionality) and early life experiences (such as familial adversity before age five) which account for up to 38% of symptom variance in boys and 25% in girls (Karevold et al., 2009). Early onset depression is associated with a worse course of depression which is more likely to be severe and or reoccur, and depression that occurs later in life may have different causes, such as experiences of sustained stress (National Research Council (US) and Institute of Medicine (US) Committee on Depression, 2009; Patriquin & Mathew, 2017). Similarly, loneliness can occur at any age (Hutten et al., 2022) and the experience can be short-lived or chronic (Qualter et al., 2015). Approximated 40-50% of cases of loneliness are driven by genetic characteristics (Goossens et al., 2015). Hawkley and Cacioppo (2010) attribute the tendency for some individuals toward chronic loneliness to an increased sensitivity to social threat (hypervigilance) due to feeling unsafe which is similar to explanations put forward for genetic predisposition to anxiety. Psychosocially, loneliness is linked to having less frequent social contacts but examination over the life course indicated a link with perceived failure to be meeting societal expectations at a given age (Hutten et al., 2022).

Negative affect and the Transdiagnostic View. The term negative affect was initially used in respect of personality research to describe the disposition of some individuals towards negative mood states (Watson & Pennebaker, 1989). Although it is normal to experience negative emotions, prolonged feelings of depression, anxiety or loneliness that continue even in the absence of overt stressors can be a risk factor for more enduring mental health disorders (Watson & Clark, 1984) such as major

depression (MD), or general anxiety disorder (GAD). The diagnostic manuals for mental disorders currently retain separation between depression and anxiety disorders, but negative affect theorists advocate for a joint diagnostic and therapeutic model due to a common aetiology. There is certainly an overlap in symptoms and common co-morbidity and progression over time (r=.62 in Kessler et al; Coryell et al., 2012; Kessler et al., 2010). The Hierarchical Taxonomy of Psychopathology (HiToP) is a transdiagnostic alternative to traditional mental health diagnostic categories and this taxonomy groups disorders under several different dimensions according to structural equation modelling (Kotov et al., 2017). In HiToP, depression and anxiety appear under the spectra of internalizing disorders in subfactors of distress and fear respectively.

'Negative Affect Syndrome' (NAS) explains positive effect and negative effect in relation to the underlying concept of approach (being drawn towards positive / rewarding stimuli) and avoidant behaviours (a tendency to avoid negative or threatening stimuli). The concepts of approach / avoidance are actively used as a transdiagnostic explanation for behaviour across multiple fields including evolutionary, neuropsychological, and cognitive behavioural approaches and therapies (Moses & Barlow, 2006). Under this conceptualisation, anxiety is symptomatic of an increased perception of threat, and depression is linked to a reduced tendency or capacity to find stimuli rewarding.

Transdiagnostically, Moses & Barlow (2006) couch both anxiety and depressive symptoms as difficulties in emotional regulation, because they represent emotion-driven behaviours that reinforce emotional suppression (inhibition) and avoidant coping (shielding from perceived threat or discomfort of intense emotions). Subsequent systematic reviews have posited emotional regulation as a

transdiagnostic explanation for depression, anxiety, eating disorders, and substance use disorders (Aldao, 2012; Sloan et al., 2017), citing the common use of maladaptive coping strategies of rumination, suppression, and avoidance instead of adaptive strategies (acceptance, problem solving and re-appraisal). In support of this view, neurological investigations show that depression and anxiety are associated with alterations in the connections between the orbito-frontal Pre-Frontal Cortex (important in top-down control of cognitive function, see Chapter 2) to the amygdala (which is highly relevant to emotion and threat perception; Maggioni et al.,2019) indicating reduced executive control over emotions. Depression and anxiety do also show areas of divergence neurologically (MD affecting the frontotemporal area, and GAD affecting parietal areas; Maggioni et al., 2019) which could represent the unique features of the conditions.

Although loneliness is not systematically grouped as a mental health condition with depression and anxiety, theorist offer a similar explanation for loneliness as anxiety, but it has a social orientation i.e., hypervigilance to (social) threat. Loneliness is defined as dissatisfaction with the quantity or quality of social relationships (i.e.., a cognitive subjective discrepancy (Spithoven et al., 2019) related to increased perception of threat (Cacioppo et al., 2016). One issue for the hypervigilance explanation is that loneliness correlates more strongly with depression than anxiety (Owczarek et al., 2022) indicating a more nuanced approach to investigation of negative affect is required. Loneliness fits with the transdiagnostic explanation of emotional regulation, employing maladaptive coping strategies which leads to reduced approach behaviour. Like depression and anxiety, loneliness also shares the cognitive component of ***repetitive negative thinking***

which has been found to sustain negative emotions and worsen mental health to clinical levels (Ehring & Watkins, 2008; Zawadzki et al., 2013). Different conditions refer to repetitive negative thinking using different terms (e.g. worry, rumination, perseveration), but it is argued that the difference is mainly in content, rather than process (Ehring & Behar, 2020; Ehring & Watkins, 2008). Further details follow about repetitive negative thinking in the form of rumination and worry which are associated with loneliness/depression and anxiety.

Depression

Depression is a prevalent mental health complaint. Statistics vary widely due to measurement criteria but a large international study (Bromet et al., 2011) estimated a lifetime prevalence of major depressive episodes as 14.6% in high income countries. Core symptoms of depression disorders per the ICD10 diagnostic manual are depressed mood, loss of interest and enjoyment and reduced energy, leading to fatigue and diminished activity. Other common diagnostic symptoms include cognitive effects such as reduced concentration and attention, and a 'Negative Triad' (Beck et al., 1979) with negative views of the self (including low self-esteem and self-confidence, feelings of guilt and unworthiness, and ideas of self-harm), world and future. Physiological symptoms include disturbed sleep and appetite (normally weight loss but a subgroup gain weight; Konttinen, 2020)- see Chapter 4 Other Psychiatric Comorbidities. In review, Nezu (2000) notes that depression has been linked to reduced self-control processes (self-monitoring, self-evaluation, self-reinforcement; Roth & Rehm, 1980), as well as reduced problem-focused coping (Nezu, 1987).

Measures of Depression. The gold standard measure for diagnosing clinical depression (and anxiety) is ***Structural Clinical Interview*** based on DSM criteria, either by a mental health professional, or lay person (Nezu et al., 2002). Examples include the Munich-composite international diagnostic interview DIA-X (Wittchen et al., 1998) which is based on a World Health Organisation clinical interview, used to assess a range of mental health symptoms, disorders, comorbid conditions and their psychosocial impact. The interview is carried out by trained clinicians which means it is costly and more time consuming than other measures such as ratings scales. The measure is judged to have high objectivity and *inter-rater reliability (κ: 0.82–0.98;* Wittchen et al., 1991).

Hamilton Rating Sale for Depression (HRSD; Hamilton, 1967) is a 21 Item clinician rating instrument which is in the public domain and designed to be completed following a diagnostic interview as standard questions to learn more about the nature/ severity of symptoms. Ideally the interview and rating scale would be completed by more than one interviewer. There are no norms for the scale. Interrater reliability is reported as >.84 but internal consistency .48 to .78 and judged to have high applicability to clinical and research used such as treatment effects (Nezu et al., 2000).

Self-report Scales (screening). Most self-report questionnaires screen for general low mood or potential disorders by assessing the frequency that symptoms are being experienced (or have been in recent weeks e.g. last two weeks) on a rating scale. Examples include the **Centre for Epidemiological Study of Depression Scale** (CES-D; Carol et al 1981). This scale was designed as a self-

report version of the clinician rated Hamilton Rating Sale for Depression and was updated with reference to DSM-IV criteria. The scale was judged to have high clinical relevance and research applicability (Nezu et al., 2000). The scale is quite long with 40 questions. Norms have been established and sensitivity (.87) and specificity (.70) are adequate but the cut-off maybe too low and it is not advocated as an isolated diagnostic measure of depression (Vilagut et al., 2016).

Beck Depression Inventory (BDI**;** Beck 1996). 21-Item scale which takes 5-10 minutes to complete and is judged to have high clinical utility (mapping on to DSM-IV criteria) and research applicability as a widely used measure (Nezu et al., 2000). The scale has good internal consistency (alpha .92-.93) and good convergent validity with the Hamilton Rating Scale for Depression (.71). The scale yields a two-factor analysis structure (somatic-affective and cognitive) and norms and cut-offs have been established.

The Patient Health Questionnaire (PHQ-8) is a compact well validated scale (Levis et al., 2019; Manea et al., 2015) used for screening. The scale is widely used in the NHS general practice and counselling sessions to monitor patient symptoms and response to treatment. The PHQ-8 and PHQ-9 are the same scale, but the PHQ-9 is recommended for use in clinical populations. The nineth item can overestimate the risk of suicidal ideation in non-psychiatric populations, the PHQ-8 is therefore recommended for research (Gomez-Gomez et al., 2023).

Efficacy of the measure. In meta-analysis in relation to clinical interview sensitivity was found to be minimally reduced in the PHQ-8 compared to PHQ-9, and the specificity was similar (Wu et al., 2019). PHQ-8 criterion validity sensitivity

ranges from .77 to .88, internal reliability alpha is .86-.89 and test re-test reliability is .84. In comparison with diagnostic interviews, the reliability was .84 and the scale was able to accurately discriminate between patients with MDD, partial and full remission (Lowe et al., 2004).

Depression and Obesity Research. Epidemiological studies and meta-analyses (de Wit et al., 2009; Luppino et al., 2010) indicate that depression and obesity have reciprocal effects on one another, but findings are inconsistent. A meta-analysis of a community sample (Luppino et al., 2010) looked at depression with both overweight and obesity (BMI) reciprocally in longitudinal studies. Obesity at baseline increased the risk of depression at follow-up (unadjusted OR 1.55 CI 1.22-1.98), as did overweight (unadjusted OR 1.27 CI 1.07-1.51). Depression increased the risk of obesity (OR 1.58, CI 1.33-1.87). Subgroup analyses (obesity as a predictor of depression) found a significant effect of continent (stronger associations for US samples) and depression assessment (stronger associations for clinical interview than self-reported symptoms). Interestingly age group and gender that were identified as moderators in cross-sectional meta-analysis (de Wit et al., 2010) did not show significant effects longitudinally (see Chapter 2 for a discussion of the importance of under-reported background variable effects in relation to obesity research). There were no significant subgroup effects for depression as a predictor of obesity either.

Investigation of prevalence rates of mental disorders (classified using the Munich-composite international diagnostic interview) in clinical and community samples Baumeister & Harter (2007) found significantly higher frequencies of mood and anxiety disorders and increased odds ratios (between 1.4 and 2.7) for those in the obese category (BMI>30) compared to those with BMI <25. In contrast, a

comparable study by John et al., (2012) found no relationship between BMI, depression, and anxiety in a community sample of 18- to 64-year-olds once smoking status and alcohol consumption were included in the analysis (these factors were only relevant for male BMI). One drawback of the study was that height and weight were self-reported, which can lead to weight under-estimation. The study did find significant relationships between BMI and age, sex and years of education.

A large Australian study (Sahle et al., 2019) examined the association between depression (n=1646; f=1012), anxiety (n=1,638; f=1008) and BMI in young adults at baseline (age 26-36) and follow up (age 31-41). More than half of the BMI values were self-reported, but a correction factor was applied. Lifetime history of disorders was assessed by diagnostic interview (Composite International Diagnostic Interview (CIDI) and the findings were adjusted for covariates in sex, age, education, family history metabolic disease and social support. Males with mood disorders showed a significant increase in BMI (Beta= 0.77 or .70 adjusted for diet and lifestyle factors). For females the Beta (.53) became non-significant after adjustment for diet and exercise. There was no significant effect of antidepressant use.

Anxiety

Anxiety is an emotion which is closely related to fear or worry that something bad will happen (threat). Fear is an alarm reaction entailing a motivation to escape and psychophysiological preparation for action (Antony, 2001). It can arise as a response to a previous stressful experience or anticipation of a new one. Some authors consider fear and anxiety to be part of the same construct as fear i.e.., a form of emotional Sympathetic Nervous System arousal (Lazarus, 1991), however anxiety involves additional components of negative affect – feelings of perceived

uncontrollability and unpredictability of future events (Antony, 2001). Anxiety disorders are reported to be the most common class of mental disorder (12-month prevalence = 25%; Bandelow & Michaelis, 2015) and are also core symptoms of a variety of other conditions, including trauma and addiction (Remes et al., 2016). Generalised Anxiety Disorder (GAD) is characterised by excess and uncontrollable worry and anxiety about multiple topics that occurs more days than not for at least 6 months, plus 3 or more of the following: muscle tension, restlessness / on edge, difficulty concentrating / mind blanks, being easily fatigued, irritable and / or disturbed sleep.

Measures of Anxiety. Methods to measure anxiety are very similar to depression and include the diagnostic interview (see Measures of Depression), clinical checklists and self-report measures.

State-Trait Anxiety Inventory (STAI, Spielberger, 1983). The STAI is a widely used measure of anxiety and has been used with clinical and community samples. The 40-item scale has separate items relating to state and trait anxiety, although the factor structure reflects four factors. Per Roemer (2001) the scale has good internal consistency (alpha .86 to .95) and adequate test-retest reliability (rs .71 and .75). There is relatively poor construct validity (between those with depression and anxiety and discriminant validity between those with /without disorders. The manual does report average scores for males and females as well as those diagnosed with anxiety disorders.

Beck Anxiety Inventory (BAI). This is a widely used self-report scale of 21 items. The authors designed it to provide greater discrimination between anxiety and depression symptoms, so the focus is on somatic (bodily) symptoms (similar to

panic) rather than cognitive symptoms such as worry or rumination. The scale correlates moderately with anxiety measures (r .48) and has a fairly low correlation with depression(*r*=.25) in psychiatric sample (Beck et al., 1998). The scale has very good internal consistency alpha .92 in psychiatric populations (Beck et al., 1988) and anxiety disorder .85 to .93 (Beck & Steer, 1993). Validity Normative sample: rs=.51 to .69 with anxiety, rs .48 to .56 with depression (Osman et al., 1997) but no age or gender norms are provided.

Generalized Anxiety Disorder GAD 7. The GAD-7 is a screening and severity measure for Generalized Anxiety Disorder symptoms as well as panic social anxiety and post-traumatic stress disorder (Spitzer et al., 2006) and is often paired with the PHQ-8. The 7 items are answered on a 0-3 scale. A score of 10 or greater is considered clinically significant, and a score of 15 is an indicator that active treatment is probably warranted. The GAD-7 has been standardised on a large primary care normative population (Spitzer et al.,2006) and show having good internal validity (alpha = .92), and test re-test reliability (*r*=.83). The scale is linked to DSM criteria and at the clinical cut-point (10) sensitivity and specificity were >.80. 89% of patients diagnosed with GAD at clinical interview had scores of 10 or more (mean=14.4), therefore cut offs have good association with severity of symptoms (Kroenke et al., 2010). Correlation with functional impairments on the General Health Survey SF-20 indicate good construct validity in relation to the mental health scale (.75; Lowe et al., 2008; Spitzer et al., 2006). The scale also showed good criterion in comparison with the Beck Anxiety Inventory (.72).

Anxiety and Obesity Research. There is less research linking anxiety to obesity than that of depression and once again there are mixed findings. A meta-analysis by Amiri & Behnezhad (2019) indicate that those with obesity experience more anxiety than 'normal' weight controls (OR 1.30, CI 1.20-1.41), and Brumpton et al., 2012 found higher longitudinal ***weight increase*** for those with anxious symptoms, but other longitudinal findings were not significant (Sahle et al., 2019). There is some evidence that anxiety disorders such as GAD are longitudinally associated with some cardiometabolic symptoms (fatal coronary heart disease and phobic anxiety; Kawachi et al., 1994), also anxiety *symptoms* are linked with increased risk of stroke (Limbiase et al., 2014).

A large well controlled Norwegian follow-up study (n=25,180; Brumpton et al., 2012) found that participants with any anxiety or depression (HADS score >11) had a significant increase in risk of obesity 11 years later (RR M:1.37; f:1.18). The study adjusted for a wide range of covariates (including age, smoking, alcohol, insomnia, physical activity, education, & economic difficulty). From baseline to follow-up weight gain of 0.95kg for males and 1.12Kg for females with anxiety was found compared to those without (slightly lower for depression). Prevalence of anxiety and depression in the sample was higher in those reporting smoking, insomnia, less physical activity, low education, and greater economic difficulties. There were no significant differences when use of antidepressants was included. Brumpton et al., note that while two studies support their findings (Bodenlos et al 2011; Strine et al 2008), most previous studies *do not find a significant effect* of anxiety on weight gain (Chiriboga et al., 2008; de Wit et al., 2010; Williams et al.,2009).

Loneliness

Loneliness is defined as perceived discontent with the number or quality of social relationships (Peplau & Perlman, 1982). This can result in painful feelings and a negative affective state that is associated with multiple physical health conditions (Richard et al., 2017), mental health conditions and considerable psychological distress (Beutel et al., 2017), including a greater instance of depression (OR 1.90) and anxiety (OR 1.21) plus a 31% increase in suicidal ideation when these conditions were controlled (Beutel et al. 2017). Social isolation (objectively low levels of social contact) and loneliness are also related to adverse health effects, however logistic regression studies indicate that loneliness has a greater impact on physical ill-health (Christiansen et al., 2021). This distinction in the definition of loneliness recognises different preferences in the amount of social contact enjoyed, and that aversive negative affect is core to the impact of loneliness on health.

Measurement of Loneliness. Loneliness is generally measured by self-report. Scales can be unidimensional although many measures differentiate between different types of loneliness based on the nature of the relationship per Weiss (Weiss, 1975) as social loneliness relating to the wider social network, and emotional loneliness which is more relevant to intimate relationships (sometimes subdivided into family and intimate partner; Cramer & Barry, 1999). The UK government currently recommends the use of the three item UCLA (University California of Los Angeles) Loneliness Scale (ONS, 2018). The full UCLA scale (third revision) is a 20-item scale with half being worded negatively to reduce response bias (Russell, 1996). The UCLA scale is widely used by researchers and clinicians. The measure has good internal consistency (alpha .89 to .94) and test-retest reliability over a 1-year period (r = .73). Construct validity was established with measures of

interpersonal relationship adequacy, and correlations with measures of health and well-being (Russell, 1996). The De-Jong Giervald 6-Item Loneliness Scale is also well regarded and used internationally to measure loneliness and has fairly good reliability (between .81 and .92; (De Jong Gierveld & Van Tilburg, 2010).

Loneliness and Obesity .There is a suggestion that obesity is related to loneliness and increased social withdrawal (Rotenberg et al., 2017; Rotenberg & Flood, 1999; Rotenberg & Sangha, 2015; Yanguas et al., 2018). As this area is under-researched, particularly for adolescent age groups, Study 1 examines the putative link in more detail including the direction of effect and mechanisms of effect (see Chapter 4).

Loneliness is hypothesised to exert its physical health effects through increased psychosocial stress leading to overactivation of immune response and inflammation (Yanguas et al., 2018). Over time, frequent or chronic activation of the body's stress system (allostatic load) can have far reaching effects on the brain and body (McEwen, 1998) and physical health problems linked to loneliness include metabolic diseases (heart disease, high blood pressure, diabetes, obesity, earlier mortality), impaired immunity, poorer self-reported health and sleep disturbance (Eccles et al., 2020; Hawkley & Capitanio, 2015). Hawkley and Cacioppo (2010) further hypothesise that hypervigilance to threat can impair a person's capacity for self-regulation by reducing the ability to focus on effortful attentional processes, like behaviours used to regulate health.

Repetitive Negative Thinking

Repetitive negative thinking or 'Perseverative Cognition' is an inability to inhibit a previous thought (although the term can also be applied more widely to a tendency to continue an action in absence of the appropriate stimuli). Repetitive negative thinking is observed in several internalising mental health conditions but there is some debate as to whether they have the same cause and source (only differing on content), or whether they are functionally different (Ehring & Behar, 2020; Ehring & Watkins, 2008). Due to the ubiquity of repetitive negative thinking in internalising mental health disorders it has been suggested that it should be investigated ***transdiagnostically*** (across disorders) rather than in isolation (Ehring & Watkins, 2008). Individual differences in the tendency toward repetitive negative thinking could account for the different physical health and functional cognitive outcomes that are observed in conditions such as depression and anxiety (Brosschot et al., 2006).

Rumination. Rumination is a repetitive negative thinking style associated with depression (r=.53 with the Beck Depression Inventory; Schoof et al.,2010) and loneliness (Raes et al., 2020; Vanhalst et al., 2012). Rumination can be categorised as brooding or reflecting (Nolen-Hoeksema et al., 2008), with brooding thoughts being more negative and more strongly related to depression. In a review of emotion regulation strategies, Aldo, Nolen-Hoeksema & Schweizer, 2010 found that rumination was positively associated with psychopathology including anxiety, and depression (Clinical samples r=.87; non-clinical samples r=.49). Other significant associations with emotional coping strategies included avoidance (r.38) and suppression (r=34), and negative associations with problem solving (r=-.31) and reappraisal (r=-.14) were negatively related to psychopathology. This reinforces the

negative association between brooding ruminative thought and more constructive problem focused cognitions.

Worry. Worry is the cognitive component of anxiety which involves feelings of self-doubt about one's ability to cope or deal with a future event. Persistent uncontrollable worry is a core symptom of anxiety disorders such as General Anxiety Disorder (GAD; APA, 2013). Worry is theorised to be a form of emotional avoidance as it temporarily relieves physiological arousal (avoidance theory of worry; Borkovec et al. (2004), other theories include worry as a motivation to problem-solve or an illusion of taking control or working on a problem which helps (short term) to deal with feelings of uncertainty over the future (Roemer & Medaglia, 2001). In the long-term, worry maintains the anxiety/fear response by preventing thorough cognitive processing of the stressful stimuli (Borkovec et al., 2004) and therefore interfering with the normal 'habituation' or decrease in physiological anxiety when a person cognitively evaluates there is nothing to fear (Behar & Borkovec, 2020). Worry has also been shown to affect cognitive performance but there is some debate about the nature of this effect (Eysenck et al., 2007).

Repetitive Negative Thinking and Obesity. There is little research on the relationship between worry (as a stand-along construct separate from anxiety) and obesity, however rumination is a recognised factor in binge-eating disorder with obesity. Wang et al.,(2017) suggest this is potentially due to ruminators being more likely to dwell on, and internalise, weight-based discrimination experiences. In a study of the 5-HTTLFR gene (for increased stress vulnerability), those who also reported high rumination had higher BMI (Schepers & Markus, 2017b). A further study found that those with the vulnerable 5-HTTLFR (S) allele *and* high rumination

also displayed an attention bias for high calorie food images after exposure to stress (Schepers & Markus, 2017a). This indicates that in vulnerable individuals, a propensity for rumination is a mediating factor that can give rise to greater mental health problems and potential physical health problems through both the stress-related inflammatory response and higher calorie consumption.

Neurocognitive Function

Executive Function	Neurocognition	Functional Measures

Chapter 2 explains that deficits in cognitive function are core symptoms of internalising mental health conditions, but, despite this, lab-based studies of cognition do not always find consistent evidence of deficits (Eysenck et al., 2007). The inconsistent finding indicates there are either issues with the way cognition is being measured or defined, or the cognitive issues could be context-dependent (rather than a permanent issue), for example, there is evidence that attention bias is context dependent as it occurs in the presence of stress and specific stimuli (rewarding food, negative faces) rather than a general global deficit. This section examines different techniques used to measure and evaluate cognitive function, including neurocognitive techniques that help to link cognitive theory with anatomical mechanisms.

Lab-based performance measures of cognitive ability have a long history in assisting with the identification of health problems that have a neuro-cognitive basis. Measures of cognitive function have been used to help categorise the type of cognitive difficulty (e.g. memory, attention, inhibitory control, emotional control) and part of this process can include isolating which brain areas are affected (Alvarez &

Emory, 2006). With growing technological advancement, the study of cognitive ability is frequently paired with neuroimaging, adding validity to theoretical cognitive constructs by mapping behaviour to underlying brain areas that are correlated by timing or metabolic activity. As cognitive experiments are necessarily simplified and abstracted there is contention about their applicability to 'real-life' cognitive effects, such as on daily living tasks. This chapter will therefore define executive function and outline common investigative techniques including experiment, brain scanning and functional (observed or self-report) measures.

Executive Function

Executive Functions are a group of co-ordinated processes that control and regulate cognition, akin to the central executive in Baddeley's multicomponent model of working memory (Miyake, Friedman, et al., 2000). The integrative model (Miyake, Emerson, et al., 2000) identifies three main components of executive function: shifting, updating and monitoring of working memory, and inhibition of prepotent responses.

Executive Function	**Shifting** (mental sets)	**Working memory** (updating/monitoring)	**Inhibition** (prepotent responses)
Low-level Cognitive function	**Attention** (sustained and selective)		

Figure 4
Executive Function Components and Attention
Notes: Based on the Integrative model of executive function (Miyake, Friedman, et al., 2000; Miyake et al., 2001) plus sustained and selective attention which underpin executive functions (Alvarez & Emory, 2006) but are generally outside conscious awareness.

Executive functions are somewhat available for conscious thought, but also they underpin higher level functional cognitions such as planning, reasoning, and decision-making (therefore having relevance to conscious health behaviour). Per Figure 4, executive function is itself underpinned by low level cognitive functions such as executive memory and attention (Diamond, 2013). Some models of executive function include sustained and selective attention within the definition of executive control (Alvarez & Emory, 2006). This highlights the difficulty with separating cognitive constructs.

Executive Function and Attention. The Attention Control Theory (ACT; Eysenck et al.,2007) indicates that attentional control can be bottom-up (stimulus driven) or top-down (goal driven, effortful control), and this top-down control is essentially derived from the higher executive functions such as inhibition and switching, and to a lesser extent, working memory /updating (Shi et al., 2019). The various facets of cognition are related in complex ways which are difficult to disentangle without real-time neurological scanning techniques. Additionally, all cognitive tests tap more than one ability and brain area (Alvarez & Emory, 2006). Some researchers evaluate a construct with multiple cognitive tasks to increase the reliability that the targeted cognitive process is that being tested. The decision to perform a test battery needs to be made judiciously based on time taken and value to the research as it is not always practical.

Inhibition, Attention and Health Behaviour. Inhibition is relevant to health behaviour because it relates to our ability to inhibit unwanted actions (that is, actions that do not align with our goals) and to some extent, unwanted thoughts (Hofmann et al., 2012). As famously illustrated in diet failure research, greater inhibitory demands can lead to less mindful attention to how much we are eating (French et al., 2012;

Polivy & Herman, 1985). As different types of inhibition are thought to draw from the same pool of cognitive resources participants who are less able to suppress internal thoughts may display worse inhibition and worse vigilance or sustained attention because these resources are divided across a larger cognitive load (Baumeister et al., 2000; Nolen-Hoeksema et al., 2008; Rood et al., 2009). Understanding the nature of different inhibition tasks is relevant to our understanding and interpretation of any mechanism of effect that arises.

Inhibition of prepotent responses. Tasks that measure the inhibition of prepotent responses (also called response inhibition) require the participant to exert cognitive control over a dominant response tendency. In consideration of an appropriate cognitive task to measure inhibition it is important to consider what is being measured and the brain areas involved in task completion.

Response inhibition tasks can be categorised into three types depending on the neurocognitive components required to make a response (Zhang et al., 2017). These are i) withholding action, e.g. Go/No-go, ii) action cancellation, e.g. Stop Signal Task (SST) and iii) inference resolution, e.g. Stroop task, Simon Task, Wisconsin Card Sort (WCST). The most common task used to investigate inhibition is the Go/No-go (GNG) paradigm, which is a simple task requiring participants to follow a given rule to identify when they should withhold a response (e.g. withholding a button press on '**no** go' trials). In the Stop Signal Task (SST), participants are given a signal to respond followed directly by a signal to stop.

Interference resolution tasks require the participant to suppress an aspect of the stimuli with a strong prepotent response e.g. Simon Task, Wisconsin Card Sorting Task (WCST), Stroop task. The Simon Task involves inhibition of response

based on the visuo-spatial location of the stimuli presented (e.g. arrows or shapes are shown on the left or right of the screen and the participant must respond using the same or opposite hand). The interference in the task relies on the natural inclination to reach or respond to a stimulus using the hand which is nearest to that stimulus. The visuospatial nature of the task means there are relatively few levels of processing involved, and the response is physical rather than verbal (see chapter 6 for further detail about the Simon Task). The WCST involves inhibition of the number, colour, or shape of the elements displayed on the cards as the participant sorts them based on feedback (“right” or “wrong”) provided by the experimenter (Barceló, 2001). The WCST has been consistently associated with executive function issues (Burgess et al, 1998), and frontal lobe dysfunction (Alvarez & Emory, 2006) however, as the card sort categories are changed by the experimenter every 10 cards, the task maybe considered to be more relevant to measurement of attentional shifting (Miles et al., 2021) and or reward-based learning (Dehaene & Changeux, 1991).

The Stroop task is regarded by some to be a gold standard inhibition task due to the reliability of the observed effect (MacLeod, 1992). Participants are asked to read lists of words in coloured ink. The response conflict in the task stems from having to inhibit their natural inclination to reading out the word rather than name the colour of the word. The task operates at multiple cognitive processing levels, including surface characteristics, phonological characteristics, and semantic characteristics of the words plus response conflict (depending on how the participant gives their responses) see Parris et al. (2022) for a discussion of Stroop processing. The neurocognitive substrates of inhibition tasks are discussed in Chapter 2 of this thesis. Wostmann et al. (2013) examined the efficacy of several inhibition tasks

including Stroop, the Stop Signal Task and the Simon task and found good test-retest reliabilities (of .7 or higher) and good internal consistency. The Stroop task is commonly used to assess deficits in brain injury; Stroop interference is strongly linked to attention deficits(including selective attention and concentration), reduced processing speed and reduced speed in colour naming, such that deficits in Stroop performance are taken to implicate problems in those areas (Ben-David, 2011). The Stroop task has also been used to examine inhibition in patients with Schizophrenia and a pooled weighted mean meta-analytic effect size (.60M(g); Heges g) was found, although more recent studies using computer-based approaches had lower effects sizes (.19M(g)). In a systematic analysis of clinical depression and Stroop inhibition, the effect of classic Stroop yielded a Heges g of .85 (highly significant; Epp 2012). Epp, (2012) suggests that due to the ubiquity of the Stroop task and as as moderate effect sizes of depression on Stroop performance are already clear, future studies should focus on less researched areas. Suggestions include research on depression co-morbidities, and cognitive tasks that can tease apart the underlying neurocognitive mechanisms, such as effects of conflict monitoring, impaired disengagement, and attentional bias.

Inhibition and Obesity Research. The relationship between inhibition tasks and obesity have been investigated. Stinson et al. (2018) examined the association between depressive symptoms, BMI, Body fat and measures of inhibition including Stroop, WCST and the Iowa Gambling Task (IGT; a decision-making task). They found that higher BMI was associated with poorer Stroop and WCST performance. Higher body fat (body composition DXA) was associated with worse IGT and WCST. Stroop and Depression independently predicted BMI and weight gain over time. In the traditional Stroop participants are given colour words e.g. blue, written in different

coloured inks and they must resolve the interference between colour perception and word meaning. A drawback of the task is that it relies on verbal ability and word decoding. Due to its complexity, there are more processes and brain-regions involved, although response conflict is fairly consistently related to the left inferior frontal gyrus in the prefrontal cortex (BA9/44; Parris et al. 2019).

In systematic review of cognition, BMI and eating behaviour Vainik et al. (2013) found that the Stop Signal Task and Stroop had the most consistent relationship with BMI (obesity and weight gain) and eating behaviour (increased food intake and eating more than intended). Go/No-go tasks had a more mixed relationship, and the effects were linked to ‘go’ reaction time. Studies have also found significant effects related to WCST performance, but this has not been tested as widely. Vainik et al., also compared the reported reliability and internal consistency of a range of cognitive tasks. The tasks each had a variety of measurement outcomes so there was not a large comparison pool, however the WCST was judged to have comparatively poorer performance (<.70), with better performance from Stop Signal and certain Stroop task measures (reaction time to incongruent trials). The similarity of the CPT task with the Go/No-go task was also noted and reliability (hit rate reaction time and commission errors) was >.70 with good internal consistency.

In summary, inhibition performance is indicated to have an important role in health behaviour (including dieting and repetitive thinking). An extensive systematic review of cognitive performance tasks (Vainik et al., 2013), indicated that the Stop Signal Task and Stroop had the most consistent relationship with the BMI and eating behaviour. Both these tasks are common measures of response inhibition, indicating

that eating behaviour and higher weight status is related to response inhibition deficits. One explanation for dieting failures and eating in absence of hunger is cognitive load, which is often assessed using sustained attention tasks. Successful inhibition requires an element of sustained attention. It is possible that these cognitive processes may both influence cognitive deficits observed in relation of obesity.

Neurological Methods to Research Cognitive Function and Health

Thought processes are not directly observable so being able to locate theoretical cognition in physical function helps support or refute theories on mechanism of effect (Kam & Handy, 2013). Neuroimaging during cognitive tasks can help provide information about which structures are being activated (spatial information), the magnitude of the activation and the timings of those activations (temporal information). These measures can help to gather more detail about the nature of different cognitive constructs, such as attention or inhibition, and the extent of their effects (Bernal & Altman, 2009; Cacioppo et al., 2015; Knyazev, 2007). Neuroimaging can also be conducted at rest to examine individual differences in the location and magnitude of brain activity (Geng et al., 2017; Knyazev, 2017; Mesquita et al., 2010). The following subsections will give an overview of the main neuroimaging techniques used in cognitive neuroscience investigations followed by neurocognitive research findings related to inhibition, sustained attention (vigilance/ cognitive load) and health.

Brain scanning techniques. Modern brain investigations take a range of forms and are no longer limited to post-mortem and lesion studies. Some techniques

measure the physiological structure of the brain, while others can investigate neural activity. Neural activity can be examined by brain waves, metabolic activity and proxy measures such as blood flow and oxygenation (Blood Oxygen Level Dependant signals). The following is an overview of the main neuroimaging techniques based on Newman (2019).

Electroencephalography (EEG) and Magnetoencephalography (MEG). EEG and MEG directly measure the electrical activity produced by neurones as they communicate. EEG scans are therefore usually time linked to stimuli presentation and event related potentials (ERPs) are measured in milliseconds. The source of the signal in the brain however is difficult to localise. MEG is a related technique which utilises the brain's magnetic field, which is localised more readily, but it is considerably more expensive than EEG (Newman, 2019).

Positron emission tomography (PET). PET scans use radiation to scan neural activity (e.g., blood flow, oxygen uptake) and was the first 3D scanning method that was relatively non-invasive (Newman, 2019). It requires the participant to inhale or be injected with a radioactive tracer, then lie on a scanner bed and be passed through photon detector rings (CT scanner) which construct up a 3D image from 2D slices. Initially the method was used to monitor oxygen changes in the brain, but it can be used to trace a variety of substances in the body. It is possible to use PET for cognitive research tasks, but the participants are fairly constrained physically, and more suited to static scans e.g., examining treatment progress. The PET scanners are more expensive than fMRI scanners and require a high degree of safety precautions. The technique is time limited due to the half-life of the radioactive tracers meaning participants need long time gaps between sessions and should not have more than 2-3 scans in one year, although this is a rule of thumb as radiation

exposure varies and is judged in relation to the health risks verses gains (Brix et al., 2009; Hosono et al., 2021; Nievelstein et al., 2012; Newman, 2019). The radiation dose delivered by radiating scans depends on various factors including the participant age, which area of the body is being scanned and the type of machine/technology used. The exposure risk in Sieverts SV or mSV (or milli-Sieverts) can be calculated to check cumulative risks to participants or researchers administering radiation (1Sv is equal to a 5% risk of developing cancer; average background exposure to radiation in daily life is 2.5mSv per year; typical PET scans in adult research would be 7-8 mSV; exposure for people who work with radiation is 20mSV per year; all figures per Newman, 2019 p 389).

[*NOTE: **Re lifetime maximum radiation exposure:** There is not much direct research on lifetime cumulative radiation effects for PET per se. The value of 100 mSv or 150mSv is mentioned in some papers as a lifetime maximum for healthcare workers Brix et al., 2009; Hosono et al., 2021; Nievelstein et al., 2012].

Transcranial Doppler Sonography. TDS is a form of ultrasound scan that can be used to detect changes in the cerebral blood flow in the large basal arteries (Purkayastha & Sorond, 2012). It is mainly used to diagnose health problems that affect the blood vessels in the brain, (such as blockages), but it has been used as a proxy measure for brain activity due to cerebrovascular coupling (Kelley et al., 1993). It has also been used to detect worse cognitive dysfunction in elderly patients (Sabayan et al., 2012; Vinciguerra et al., 2019).

In TSD Soundwaves are emitted from a doppler probe and reflected via red blood cells in blood vessels. The resultant data is used to calculate the blood flow velocity, and it has high ‘temporal resolution’. A drawback of the technique is that it

requires a detailed knowledge of the location of cerebral arteries and the direction of blood flow to apply the technique effectively. Also, the locations where blood flow can be measured are limited to four ‘acoustic windows’ (where the skull is thin enough in most patients to detect a signal) and the location of the signal is difficult to pinpoint (Newman, 2019).

Magnetic Resonance Imaging MRI. MRI scanning gives detailed high-resolution scans of internal structures based on energy released by hydrogen atoms in different body tissues, under the influence of a magnetic field. **Structural MRI (sMRI)** used discrimination between fat and water in neurological tissue to examine the volume of white matter, grey matter, cortical thickness, and morphometry. MRI is combined with a range of techniques to measure brain activation (Newman, 2019). **Functional MRI (fMRI)** is considered a gold standard brain scanning technique and is widely used in clinical settings. fMRI uses magnetic fields and radio waves to measure the Brain Oxygen Level Dependent (BOLD) signal. This is a proxy measure for neural activity (using the reliable observation that active cells use more oxygen). fMRI can locate the source of neural activation to within less than a millimetre, but the BOLD signal (and the fMRI scan rate of 1-2 seconds) is slow compared to EEG. **Diffusion MRI (dMRI)** can be used to examine the integrity of white matter axons that communicate across the brain. **Functional Connectivity MRI (fcMRI)** used the BOLD signal to identify structural connectivity patterns (Van Dijk et al., 2010) i.e. areas of the brain where activity is synchronised and therefore likely to be working in tandem. The BOLD signal is generated due to magnetic differences in oxygenated and deoxygenated blood. As well as being very expensive (equipment and running costs), MRI requires considerable constraints on participant movement. It is also not suitable for any participants with metal in their bodies (e.g. cochlea implants, and

some tattoo inks), and needs to take place in a setting with a high degree of safety measures and trained personnel.

Functional Near Infra-red Spectrometry (fNIRS). fNIRS is a non-invasive brain imaging technique that uses light in the near infrared spectrum (which passes through bodily tissue) to estimate changes in oxygenated blood in cortical regions of the brain. Like fMRI, fNIRS relies on the Blood Oxygen Dependent signal (BOLD) and the concept of a 'neurovascular coupling' between neuronal activity and regional blood flow/oxygenation. Unlike fMRI however, fNIRS obtains the signal using infrared light (at two frequencies) sent from a transmitter optode to a paired receiver optode. The transmitter and receiver create a banana shaped ray of infrared light. Oxygenated haemoglobin (OHb) and deoxygenated haemoglobin (HHb) blood are different colours, so they absorb the light spectra differently to one another. The concentration of the light waves returned are converted using the Beer Lambert Law (Cope et al., 1988) to provide a continuous measure of oxygenation of brain tissue. fNIRS is increasingly being used within clinical and healthy populations, and across the lifespan, but it is deemed to be particularly advantageous in scanning children and babies owing to its fast set up and reduced sensitivity to movement.

In some circumstances fNIRS can give better temporal information than fMRI (Tak & Ye, 2014) and fNIRS has spatial resolution of around 1cm accuracy, providing a cost and time efficient compromise between spatial and temporal information offered by other methods. A significant drawback of fNIRS compared to fMRI is the depth of scanning. In fNIRS, the distance between the source and detector optodes sets the depth of the light rays through cortical tissue but the depth of light penetration is effectivity limited to 5 - 20mm of cerebral tissue (Fukui et al., 2003). Source-detector distances of between 20-40mm are viable (Strangman et al.,

2013), distances of 30-35mm were found to be optimal (maximising depth while balancing signal to noise ratio; SNR).

To summarise, in comparison to other methods fNIRS is a relatively simple and cheap scanning technique that has extremely low risk to both participants and operators. fNIRS does not require stringent safety protocols and can be used in and out of the lab. The technique is also minimally restrictive for the participant, requiring only a cap or band to support the light emitting optodes and hold them in place. Unlike EEG caps, there is no requirement for electro-conductive gel or hair-washing meaning a faster and simpler testing session for the participant. fNIRS can be used to scan participants brain activity during the resting state as well as during tasks as it is more robust in the face of movement artifacts than other techniques. fNIRS can also be used to generate information about an additional dimension of brain activity which is functional connectivity. This can help identify which brain networks are being activated.

Cleaning Neurological Signals. There is a challenge to separate brain signals from the other physiological signals which include cardiac rhythms (1-2 Hz), blood flow in the scalp, Meyer waves (.1Hz), respiration (.3-1.0 Hz), and movement artefacts. A range of signal cleaning methods are available depending on the nature of the signal (task-evoked or non-task-evoked, neural or systemic, regular or intermittent; Scholkmann, 2014), but none are a perfect solution.

Use of filters are common, but they necessitate loss of signal data e.g., very low frequency oscillations (0.1hz-1.01) may be filtered out, however, at times these signals are important e.g. for assessing functional connectivity (Kirilina et al., 2012). Even when fNIRS data are comprehensively filtered, they are not able to remove overlapping signal frequencies (Gruber et al., 2020), or systemic signals from veins

in the scalp (Kirilina et al., 2012). The deoxygenated haemodynamic signal is less subject to such signals (heart rate from the body or skin) than the oxygenated signal (Kirilina et al., 2012), although it is lower in magnitude.

Signal noise can be generated by the instrumentation or a poor fit between the optodes and scalp leading to movement artefacts and the problem is discussed by Gruber et al., 2020. Visual inspection of the traces for evidence of abrupt movement are common, but the process is subjective, time consuming and prone to errors (Gruber et al., 2020). An alternative is automatic algorithms which detect the edge of the movement artefacts (MA). In both cases the MA is removed from the trace, or the signal may be discarded (e.g., if it contains more than 10% MA) leading to loss of data. Some methods compensate for this by patching over the missing signal. Cleaning methods have limited effectiveness and can result in bias (Santosa et al., 2017) and loss of genuine signal data. Consensus on best practice (which techniques to use and in which order) is yet to be reached (Pinti et al., 2019).

Hemodynamic Brain Activity and Neural Networks. Before giving an overview of relevant neurocognitive research, Figure 5 (the Main Lobes of the Brain) and Figure 6 (Anatomical planes) are presented to draw the reader's attention to some key brain areas and terminology that can be referred to as necessary. This is followed by contextual information about haemodynamic measures of brain activity used in fMRI and fNIRS scans i.e. blood oxygenation (Blood Oxygen Level Dependant signals).

Notable Brain Areas. Relevant brain areas highlighted in Figure 5 include the dorsal frontoparietal areas (attention), lateral frontoparietal network (control), the medial temporal/ medial frontoparietal (default mode network) and the limbic system

(centres of emotion and fear). Gibb and Kolb (2017) and Gazzenega (2014) provide a comprehensive overview of the neurological basis of cognition.

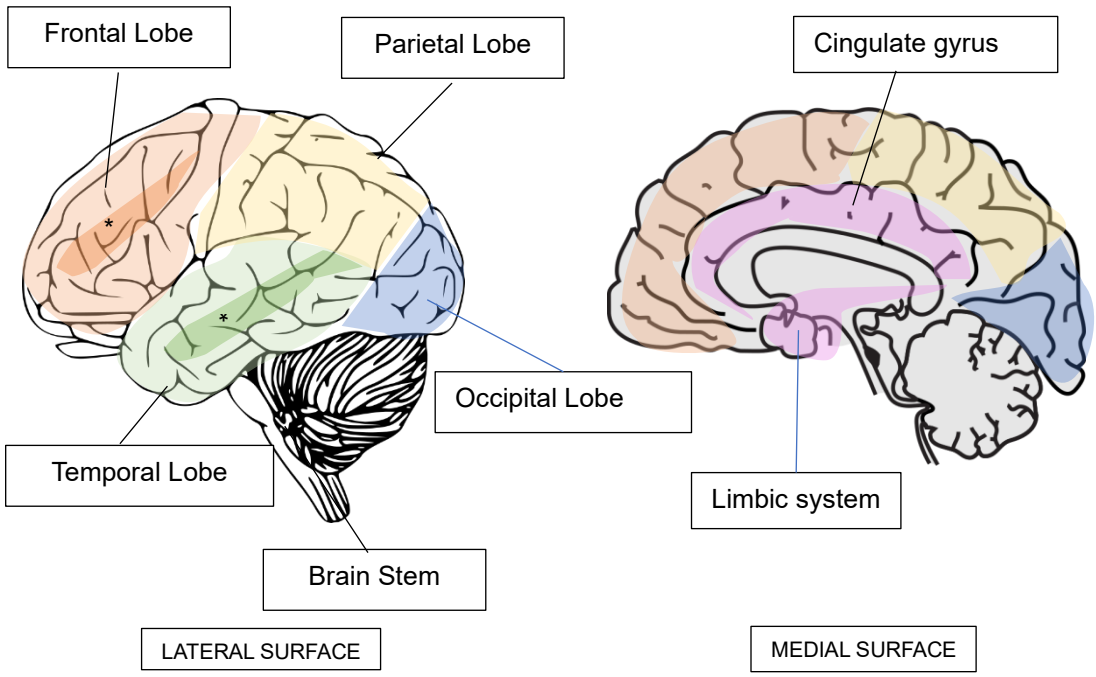


Figure 5
Main Lobes of the Brain
Note: * The Default Mode Network is a distributed network of which includes areas of the medial temporal and medial frontal lobes (Spreng et al., 2010).

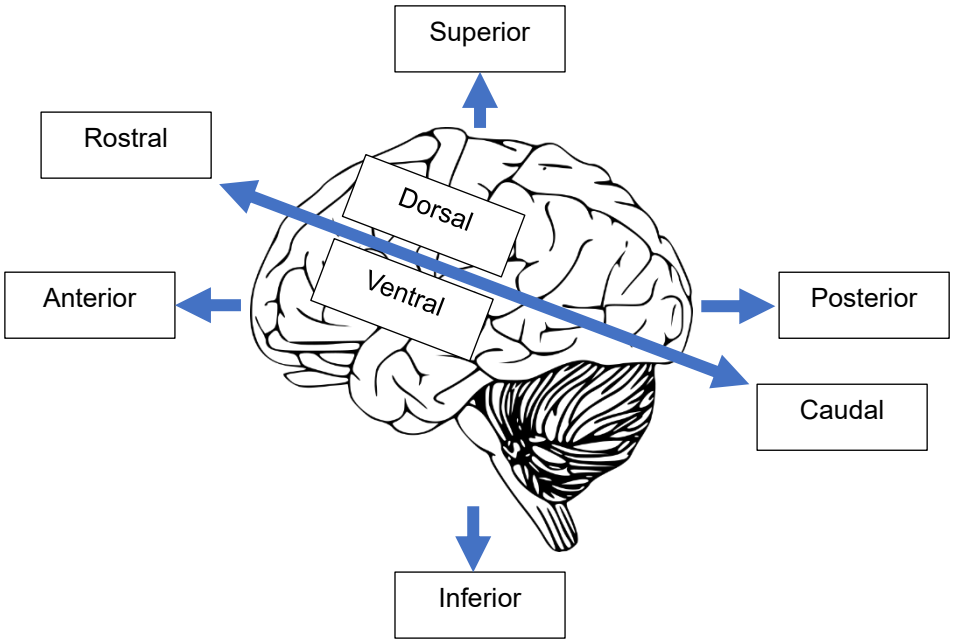


Figure 6
Anatomical Planes of the Brain

Haemodynamic Signals (BOLD response). The 'neurovascular coupling' effect is used to infer changes in brain activity from changes in blood flow. The BOLD response is used in scanning techniques such as fNIRS and fMRI. In general, an increase in the positive BOLD signal represents a net increase in neuronal activity, blood flow (due to neurotransmitters such as increased glutamate leading to vasodilation) and oxygen consumption (Hall et al., 2016). When neurones are activated, a local increase in blood flow and volume is observed leading to a rise is oxygenated haemoglobin (O_2Hb) and a commensurate (but smaller) reduction in deoxygenated haemoglobin (HHb or HbR) concentration over about 10s. This is followed by a plateau and a return to baseline. There is an average 2s time lag between the peak O_2Hb and HHb but there is large degree of inter-participant difference in time taken for the signal to peak (0-3.6s), possibly resulting from physiological differences e.g., size of blood vessels (Huppert et al., 2006).

There are also cases however where the BOLD signal does not follow its usual effect. Some neuronal activity can lead to vasoconstriction and decreased blood flow to inhibit the activity of certain brain areas. Hall et al. (2016) outlines further complications in interpreting the BOLD signal such as relative differences in what constitutes ‘normal’ signal activity for different brain regions (e.g. some brain networks are more active in the resting state than during a task), and differences due to pathology which may be difficult to separate as BOLD, as all neuroscience is still being investigated. This makes it more important to triangulate multiple measures / investigation techniques.

Functional Connectivity. Functional Connectivity (FC) is the synchronised increase / decrease of activity in different brain regions (indicating they are working together). FC is examined using the degree of correlation in haemodynamic signal between brain areas in real time. Use of FC is commensurate with our growing understanding of the brain as groups of interconnected brain networks that work together, rather than stand-alone regions of activity (Chao et al., 2021).

The Default Mode Network (DMN). Early brain scans used the resting state as a baseline of ‘doing nothing’ to help identify which areas of the brain were active during certain tasks. As scanning methods improved it became clear that parts of the brain increase in activity during a resting state scan. Certain brain regions such as the Default Mode Network (DMN) were found to be more active during the resting state than during tasks leading to the discovery of ‘anti-correlation’ in brain network activity (Biswal et al., 1995; Fox et al., 2005; Raichle et al., 2001). While ‘task positive’ networks (such as the frontoparietal network) were active, ‘task negative networks' such as the DMN were actively down regulated (Fox et al., 2005). Loss of

attention during a task is associated with greater deployment of the task negative brain network (Fox et al., 2005). Further advances have shown that these networks are not so much task 'positive' and 'negative' but actually reflect external versus internal information processing (Spreng, 2012). Specific areas within the DMN include:

- Lateral and medial temporal lobes (Spreng et al., 2010)
- Posterior Cingulate Cortex (PCC; convergence of interoception, thoughts about the self and mental representation),
- Medial Posterior Frontal Cortex (MPFC; regulates the ventral PCC and thoughts about the self),
- Left Inferior Parietal Lobule (IPL; self-related thoughts and left IPL integrating complex semantic information (Bressler & Menon, 2010; Davey et al., 2016; Poerio et al., 2017).

Barrett and Satpute (2013) discuss that the more ventral (frontal) portions of the DMN or 'mentalizing' network are active during self-related cognitions and feeling, whereas the more dorsal (upper and posterior) nodes are activated during more abstract or third person judgements, including mind-wandering.

Functional Connectivity at Rest. Neural activity can be examined at rest (often targeting the DMN) as well as during tasks. Resting state (RS) scans can be conducted with eyes closed or open and can serve as a baseline of neural activity to task-based scans, but they also have merit, particularly in investigations of functional connectivity (FC). Detailed fMRI investigations of the effect of the eye state during the scan indicate it primarily affects activity and correlations between the visual and somato-motor cortex (Laumann et al., 2015). Consistency of the method chosen appears to be more important than the eye state per se. Comparisons between

resting state scans and task scans have been used to monitor vigilance (Harrivel et al., 2013) which has direct relevance in real world settings such as vigilance dependent occupations (Navy, Armed forces, pilots) as well as healthcare. Prehn et al., (2017) for example found that RS functional connectivity in the prefrontal cortex (using fMRI) was a useful biomarker of cognitive improvements in clinical trials looking at the effects of exercise on overweight participants. Significantly increased RS functional connectivity was seen between dorsolateral prefrontal cortex and superior parietal gyrus / precuneus after moderate aerobic exercise training. RS functional connectivity has therefore proven useful in the examination of health and cognition.

Investigations into the optimal timings of fNIRS resting state scans (Geng et al., 2017; Wang et al., 2017) indicate the minimum timing as 2m, or 7m for examining functional connectivity. fNIRS functional connectivity between nodes in the same cortical network in resting state stabilised after 1m, however local and global networks were reproducible after 5 minutes (Geng et al., 2017).

Neurocognitive Research in Cognitive Load and Inhibition

Previous studies can help highlight methods and brain areas that are relevant to cognitive load and inhibitory control so these can be considered for further investigation. Haemodynamic signals in the brain have been used as a biomarker to identify issues with cognitive load and vigilance which are related to continuous attention (Aghajani et al.; Ayaz et al., 2012; Warm et al., 2008). Cerebral blood flow (measured using TDS) was related to performance in working memory tasks, and that decline in vigilance is accompanied by reduced blood flow velocity (Warm et al., 2008), particularly to the right cerebral hemisphere. fNIRS studies have also been

used to assess workload changes during working memory and attention (Aghajani et al., 2017). fNIRS field studies in piloting and air traffic control (Ayaz et al., 2012) indicated increased changes in the left dorsolateral prefrontal cortex (close to AF7, inferior frontal gyrus) were related to higher cognitive load and frustration. They also note that training resulted in reduced Hbt (total haemoglobin concentration) to this area. Hbt being strongly correlated to measures of blood flow (Huppert et al., 2006).

Kim-Spooner et al.,(2016) investigated neural and behavioural inhibition using fMRI and selected regions known to be engaged by inhibitory control related to interference (conflict) and error-processing (Feil et al., 2010; Koechlin et al., 2003; Roberts & Hall, 2008) that were significantly correlated with behavioural performance. Relevant areas included the left posterior-medial frontal cortex, right inferior frontal gyrus, left and right inferior parietal lobules, right insula, right superior frontal gyrus, and left middle frontal gyrus. Neuro-physiologically, the response itself can be divided into separate components of processing, and carrying out the action e.g. planning, motor response, and these cognitive processes can be investigated using time-bound scanning techniques such as EEG (electroencephalogram) for a fuller understanding of the neurological processes that are at play.

The Go-NoGo (GNG) and Stop Signal Task (SST) are similar tasks and use overlapping brain networks, however, they have different Event Related Potential (ERP P3) signal timing (shown in EEG inhibition studies), and deficits can be observed in one task and not in the other, indicating that the brain operation during the tasks is slightly different and different cognitive processes are taking place (Raud et al., 2020). Broadly, the neurocognitive difference lies in the fact that in the GNG the participants must quickly **decide** whether to respond (decision making: frontoparietal attention followed by motor control), whereas in the SST participants

must quickly **stop** an action that they have mentally started (because the sensory-motor response system has already been primed to respond, i.e. it is biased for reflexive inhibition). This detail is relevant to the interpretation of spatial brain scan findings as different parts of the response process come from different parts of the brain.

Neurocognitive Function and Health

In the current study we are interested in the spatial location of brain activity during tasks of inhibition and sustained attention, as there is some indication that individual differences in brain activity during task and during rest are linked to executive function, negative affect and possibly adiposity. The Default Mode Network has been identified as a source of aberrant neural activity in several mental health conditions. Various researchers (Sonuga-Barke & Castellanos, 2007; Weissman et al., 2006) postulated that attentional deficits displayed in neurocognitive testing (resulting in longer reaction times on tasks and greater intra-individual variability in response to visual and auditory stimuli) arise from a failure to suppress the DMN.

DMN and Mental Health. Anomalous DMN activity has also been observed in post-traumatic stress disorder (PTSD), and this was strongly linked to the visual cortex, hypervigilance to threat and sensory disinhibition (Clancy et al., 2020).

Delaveau et al. (2017) used fMRI to investigate cognition and neural activity (in people in remission from depression) and found that the greater the negative correlation between activity in the DMN and the TPN, the less variable their reaction times were (intrapersonal differences), and the less they ruminated. These effects have been linked to attention capacity and allocation of cognitive resources specifically in depressed patients who felt little pleasure (Dubal & Jouvent, 2004;

Warm et al., 2008). This study draws a link between depression and rumination and an increased cognitive load. The observed effect was a reduction in the normal anti-correlation (phasic negative correlation) between the Blood Oxygen Level Dependent signal at rest compared to task, and greater intrapersonal variability (less consistency) in reaction times. The study emphasises that the greater anti-correlation of the DMN and frontoparietal networks is a positive factor, related to more efficient control of attentional resources (Kelly et al., 2008) and efficient switching between internal and external foci.

DMN and Obesity. Research that implicates the DMN in some of the cognitive deficits observed in obesity. High BMI and body fat percentage have been linked to widespread decreases in white and grey brain matter volume in the brain, including areas of the DMN (Figley et al., 2016). The same study (Figley et al., 2016) found BMI and body fat were related to increased functional connectivity in the salience network during resting state fMRI, however the study did not examine functional connectivity during task completion.

Obesity has been linked to a failure to suppress the DMN during executive function tasks studied using fMRI BOLD signal. Syan et al (2019), and Sadler, Shearrer and Burger (2018) found differences in Default Mode Network functional connectivity was related to BMI discordance in a twin study. Participants with a higher BMI than their twin had stronger connectivity between insular (part for the DMN) and cerebellar networks and the authors believe this could interfere with normal satiation signalling. The authors did not find the same effects for normal weight-based samples indicating that a subgroup of high BMI individuals are affected.

Functional Measures of Cognition

Cognitive performance tasks and brain scans only provide part of the picture in understanding cognitive problems. In clinical settings, although test batteries are used, practitioners have also developed functional-skills measures that have more direct relevance to issues encountered in daily living. Functional measures are still influenced by theory and biases around Western methods of the standard classification of disorders (e.g. failing to consider life circumstances and life experience in mental health diagnoses; Allsopp et al., 2019; Rabin et al., 2006), but they should have greater ecological validity, at least in a medical care context. A limitation of many functional measures is that they are devised with a specific disorder in mind (e.g., dementia, stroke) so items measure more profound difficulties or movement problems (Green & Young, 2009) which have greater applicability to specific clinical populations.

Taking a functional problems approach to understanding executive function (Roth et al., 2005, 2013), factor analysis indicated nine functional areas of executive function difficulty (see Figure 7). T-scores of greater than 65 indicate potentially clinically significant problems. The nine scales were broadly divided into two areas or 'indexes': behaviour regulation (regulatory control of behaviour and emotion) and metacognition (problem-solving via planning and organisation).

Behavioural Regulation				**Metacognition**				
Inhibit	Shift	Emotional control	Self-Monitor	Initiate	Plan / organise	Working memory	Task monitor	Organisation of materials

Figure 7

Behaviour Rating Inventory of Executive Function (BRIEF) subscales/ components of executive function (Roth et al., 2013)

Rabin et al (2006) investigated functional cognitive deficits in daily living using a self and carer/professional report checklist (Behaviour Rating Inventory of Executive Function; BRIEF_A) for three groups of older adults i) those who had reported cognitive complaints (CC), ii) those with amnestic-Mild Cognitive Impairment (MCI) identified in brain scans and iii) matched Healthy Controls (HC). There were significant differences in BRIEF ratings between groups in the expected direction (HC having fewer problems than MCI) with effect sizes between .07 for Inhibition to .25 for Working Memory. Fifty-five percent of the MCI group reported clinical level ratings for Working Memory (i.e. BRIEF scores of 65 or greater). None of the groups showed clinically meaningful scores on the standardised neuropsychological tests performed (this included tests examining memory, attention, executive function, language, spatial ability, psycho-motor speed and standard dementia screening tests *see Note below for more detail). The authors concluded that the BRIEF may have greater sensitivity to identify functional daily living problems than traditional cognitive tests.

*[*Note: Rabin et al 2006, P723 cognitive measures: "These included the: Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975); Mattis Dementia Rating Scale, Second Edition (DRS-2; Jurica, Leitten, & Mattis, 2001); American National Adult Reading Test (ANART; Grober & Sliwinski, 1991); Wechsler Adult Intelligence Scale, Third Edition (WAIS-III, Information, Block Design, Digit Span, Digit Symbol, Vocabulary; Wechsler, 1997); Wechsler Memory Scale, Third Edition (WMS-III, LMI and LMII, VRI and VRII; Psychological Corporation, 1997); California Verbal Learning Test, Second Edition (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000); Delis–Kaplan Executive Function System (D-KEFS, Verbal Fluency, Trail Making Test; Delis & Kaplan, 2001); Wisconsin Card Sorting Test (WCST, short form; Heaton, Chelune, Talley, Kay, & Curtiss, 1993); Boston Naming Test (BNT; Goodglass, Kaplan, & Barresi, 2001). All tests were administered by postdoctoral fellows or highly trained technicians. Level of cognitive complaint was determined from responses on the Memory Self-Rating Questionnaire (Squire, Wetzel, &Slater, 1979), cognitive items from the GDS, a Neurobehavioral Function/Activities of Daily Living Scale (NBF ADL self- and informant versions; Saykin, 1992), and the Informant Questionnaire on Cognitive Decline in the Elderly (self- and informant versions; Jorm, 1997)."*]

Demographic and Background Variables

Obesity studies have been criticised due to a lack of adequate participant details and control variables (Restivo et al., 2016). Clear demographic data helps to ensure that groups of participants being compared are as homogenous as possible (Castaneda et al 2008; Snyder 2013). There are a range of background variables and participant characteristics that could potentially have a moderating (third variable) or confounding effects on the association between health and neurocognition (such as age, gender, medication). Further, background variables can be useful to help interpret the findings, or aid comparison of the study sample with other studies (e.g. gender, ethnicity, social status, fluid intelligence, handedness).

Types of third variables

(The following explanations are based on Morrow et al. (2022); see Figure 8.) **Mediator variables** are third variables which help explain the ***process*** of the relationship between two variables. Mediators explain part of the variance of an IV, which subsequently predict change in a DV e.g., the effect of calories consumed on obesity could be mediated by time of eating (more night-time eating explaining some of the variance in obesity). **Moderating variables** influence the strength or direction of a relationship between IV and DV, e.g. the effect of loneliness on obesity could be different depending on sex at birth (obese girls maybe more lonely than obese boys due to social demands). **Confounding variables** are associated with both the IV and DV but could not logically explain the association between them.

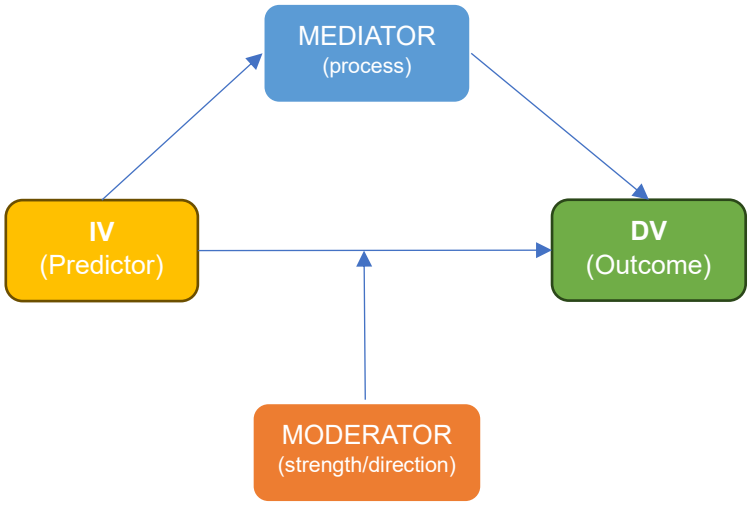


Figure 8
Visual Representation of the relationship between IV and DV and third variables (mediators and moderators).

The following section examines some common background/demographic variables (not exhaustive) that were considered in the planning of Study 2. Owing to the complexity of the interactions between the main variables in the study and their bi-directional relationships, an additional table is provided in Appendix C to help explain the nature of the relationships (and types of third variables involved) more clearly. These variables were either addressed in the study design (e.g. sample restrictions on age), or data was collected for visibility and comparison with future studies.

Age

Age is likely to be a moderating variable in the association between health and neurocognitive function. Studies have noted that the prefrontal cortex is not fully mature until around 25 years (Geier et al., 2010) meaning younger age groups may have reduced inhibitory control. In addition, cognitive capacities are known to change in older age, with greatest reductions in cognitive performance seen after age 60

(Salthouse, 2017). Although many cognitive abilities are less affected by age, executive function task performance appears to be more vulnerable to age-related decline (Alosco et al., 2014; Goh et al., 2012; Hayes et al., 2014). Older age groups are also more likely to show signs of chronic metabolic health problems linked to weight and cognitive impairment, such as obesity, and type 2 diabetes that can also affect cognitive performance. Small (2017) notes that it is useful to study cognition in younger populations to reduce the confound of age-related decline. Measures of obesity do fluctuate with age due to changes in muscle and fat (WHO, 2008), and waist circumference of young and middle-aged groups have stronger predictive associations with later mortality (Seidell et al., 1996; Seidell et al., 2010). Participants in the young adult age-group should provide an optimal age group (allowing for individual neurological developmental variance at either end of the age range) and help to reduced age-related confounds on what is a large spectrum of individual difference in cognitive ability and body tissue composition.

Sex at Birth

Sex is a potential moderator of mental health and obesity. Females often have higher proportions of body fat compared to males but their gynoid distribution of stored fat (more in the hips and thighs) is cardio metabolically protective compared to males' android distribution and deeper abdominal fat (Karastergiou et al., 2012). Additionally, there is evidence that the incidence of negative affect is higher in females (although explanations for this are primarily based on social factors). The evidence that gender moderates the relationship between obesity and mental health is mixed but indicates that the association between obesity and depression is slightly higher for females than males. For example, pooled Odds Ratio (OR) of individuals

with obesity also having depression were 1.18 compared to 1.32 for females and 1.00 for males (de Wit et al., 2009). The authors note that the male sample (in contrast to the female sample) showed considerable heterogeneity indicating that other factors could be influencing the effect of obesity on depression in males.

Although sex / gender is not a primary focus of the investigation, biological sex is recorded due to different waist circumference cut offs for males and females (based on cardiometabolic risk factors).

Ethnicity

Ethnicity has some potential to be a confounding variable in the measurement of Obesity. Body types of individuals from different geographical areas can have different body proportions (e.g. limbs in relation to body trunk) and can differ in fat distribution. Emerging data indicates that the current WHO/ IOTF BMI cut offs underestimate the morbidity risk in some body types (Misra, 2003; Misra et al., 2005). Those with Asian and South Asian body types may experience health effects at a lower BMI and waist circumference than existing cut-offs (Tomlinson et al., 2008; WHO, 2004). Data on body type ethnicity was collected to provide data for comparison with other samples and gauge whether the generic WHO/ IOTF cut-offs were applicable to the sample.

Socio-Economic Status

Socio-economic status has potential to be a moderating variable in the measurement of obesity and negative affect. Measures of socioeconomic status include annual income, median income by neighbourhood, and deprivation measures. Low income is consistently related to poorer health outcomes but the

relationship between income and affordable living can vary depending on geographical location and individual expectations. Low income has been shown to be related to obesity, although the direction of effect has been challenged. In meta-analysis (Kim & von dem Knesebeck, 2018), the only significant effects that remained significant after adjusting for publication bias indicated that obesity leads to a greater chance of low income (BMI >30, OR 1.27).

Subjective Social Status is a more wholistic measure of socioeconomic status that takes account of a person's perceived status within their social hierarchy. As such it is more sensitive to multidimensional inequality (Singh-Manoux et al., 2003) which is a better predictor of health than income (Wilkinson, 1999). The SSS has shown a stronger relationship to health outcomes than some income-based measures (Diaz et al., 2014) but shows little relationship with body mass and obesity (Demakakos et al., 2008; Adler et al.,2000). The variable was collected to aid comparison with other studies.

Psychoactive Substances/ Medication

Consumption of psychoactive substances has the potential to confound measurement of cognitive performance. The effects of psychoactive substances such as stimulants, medication and recreational drugs were investigated to gauge their moderating/confounding effects on inhibition and attention levels in cognitive testing (as acute effects). Research indicates that caffeine and nicotine affect reaction times but not response inhibition (Soar et al., 2016), although nicotine has a slightly beneficial effect on attention for smokers (Ettinger, 2017) the effects of these stimulants on the task are judged to be low. Asking smokers to avoid smoking could have considerable negative effects on cognition due to physiological cravings.

Average caffeine intake is estimated to be 2-4 cups per day (Zhou et al., 2018). Asking participants to abstain from caffeine could reduce the numbers of those willing/ able to participate or encourage participants to lie. The ubiquity of these stimulants means that asking participants to exclude them could give a less ecologically valid measure of day-to-day cognitive function. Participants were therefore asked to note the number of caffeinated drinks they have had in the last 12 hours to assess whether there were significant differences between groups.

Meta-analytic investigation of anti-depressant use has shown a modest positive effect on cognition (including executive function and divided and sustained attention; Prado et al 2018), but not for SSRIs, which are the most prescribed. In large longitudinal studies of obesity and depression, anti-depressant use did not affect either variable (Sahle et al., 2019; Brumpton et al., 2012), so they were not judged to be likely to influence the main study variables.

Recreational drugs have been shown to have considerable confounding effects on attention and cognition (Lundqvist, 2005) so participants were asked to self-deselect on this basis. Sedative or stimulant medications could affect attention. Participants were therefore advised that they should not take part in the study if they were taking medication with strong stimulant or sedative effects to the extent that they would be advised not to drive after taking it.

Handedness.

Handedness does not have conclusive third variable effects on the main outcome variables, although brain activity can present differently (in the right or left hemisphere) depending on a participant's dominant hand (Cherbuin et al., 2011). This could therefore be a confounding variable in relation to brain function where

lateralised effects are considered. Participants were asked to complete a brief Edinburgh handedness scale (Oldfield, 1971) to inform the interpretation of brain scans.

General Intelligence.

General intelligence was investigated as a potential confounding/ mediating variable as low IQ has been found to predict ill-health and earlier mortality and has been associated with cognitive performance (negatively associated with longer and more variable reaction time, R*R*=1.18;Deary & Der, 2005). General intelligence is said to measure underlying (and largely genetic) components of intellectual ability and has a relationship with executive function. Even though general intelligence and inhibition skills develop together in children, fluid intelligence does not explain inhibition in adults (Martin et al, 2021). Deary and Der, (2005) found that the effect of intelligence on mortality (adjusted for SES, education, and lifestyle factors R*R*=1.20) was non-significant when adjusted for task reaction time. The authors acknowledge that lower IQ could affect health in multiple ways including health behaviours, but findings of a systematic review by Yu et al. (2010) indicate education level maybe more important. In view of the association between childhood intelligence and adult mortality, it seems possible that lower intelligence ratings (and lower and more variable reaction times) could be a general reflection of suboptimal health (confounding variable). Overall, the potential effects of intelligence on the study variables are uncertain but there could be a confounding effect of education on health behaviour, and/ or a moderating effect of intelligence on reaction time.

In cognitive studies it is common to take a measure of general intelligence to inform sample characteristics and check for between group differences in the sample

(Lavagnino, Mwangi, et al., 2016). The gold standard measure for general intelligence is the Weschler Adult Intelligence Scale (WAIS-IV; Wechsler, 2010) but this takes considerable time to complete (75 minutes), and IQ is not a main variable of interest in this study. Measures of digit span (also included in the Weschler IQ test have frequently been used as proxy measures for general intelligence control in cognitive studies, but digit span is essentially a measure of working memory, and several studies have found issues with their usage. Reynolds (1997) concluded that scores from the forward and back digit span tests should not be combined (as per their use in the Weschler scales) to represent short term memory performance, as the two scales tap discreet areas of memory. The backwards digit span is sometimes used alone, but this taps areas of verbal ability (Li et al., 2012) which is less relevant to the current investigation.

The Raven's Advanced Progressive Matrices (APM; Raven, 1965) is a non-verbal measure fluid intelligence or reasoning ability in adults. APM performance has a moderate association with several facets of attention (.25 to.41) and inhibition (.20) and is influenced by two latent constructs: perceptual attention (path coefficient 0.48), and executive attention (path coefficient 0.25; Ren et al., 2012). There are several short forms of the APM that benefit from shorter completion time. Chiesi et al. (2012) recommends the Arthur & Day (1994) short form as was advantaged by matching the progressively increasing difficulty of items per the APM original scales (which facilitates learning during the test) and a single factor structure. They assessed the reliability and validity of this APM-SF on 2264 school and university students from 14 to 40 years old, and found it to be equivalent across age range, and a sound test of fluid ability for use in a short time period (average 15 minutes).

The 12-item Advanced Progressive Matrices-Short Form (APM-SF, Arthur & Day, 1994) was chosen as it provides a time-efficient measure of non-verbal fluid intelligence and appears to have fewer issues with gender and cultural advantages than full scale IQ (Chesi et al., 2012). Being non-verbal, the scale is less dependent on reading ability and has more relevance to the non-verbal tasks chosen for lab-based cognitive testing.

3.0 Developing a Theoretical Model of Physical and Mental Health

Rationale	Existing Models	Proposed Model	Applying the Model	Summary

Rationale: Why Develop a Theoretical Model?

Background research indicates there are associations between body size, negative affect, and neuro-cognition deficits (Seymour et al., 2015) such as attention, executive function, and emotional regulation, but is it possible that a common mechanism could influence both physical and mental health outcomes? Although the interplay between these variables is a complex area to investigate, the failure to understand the interaction between physical and mental health conditions has been identified as a barrier to improved care (Forsman et al., 2015; Schumann et al., 2014). Proponents of a more unified approach to researching mental and physical illness (Dalgleish et al., 2020; Forsman et al., 2015; Schumann et al., 2014) emphasise the importance of developing testable, evidence-based models and theories to help explain how the mental-physical interaction might work. Schumann et al., (2014) identified a range of key pathways and methods that could be combined to add a more holistic understanding of mental health and physical illness, including physiological, neural, and cognitive and psychological approaches, however few psychological models integrate physical and mental pathways to explain health outcomes. The following section outlines some health models that describe mechanisms relevant to the interaction between mental and physical health and the processes involved.

Existing Models: Physical and Mental Health

Health models explain the causal pathways that link different types of health outcomes to give researchers and clinicians a clearer understanding of the potential causal relationship between health variables. They also inform strategies to improve outcomes such as biological, functional, perceptual, and overall well-being or life quality (Wilson & Cleary, 1995). Although there is no model to explain the interaction between mental and physical health, there are several multi-level models. In their conceptual model of the **Components of Health Related Quality of Life (CHRQoL)**, Wilson and Cleary (1995) emphasise the bidirectional (and potentially negatively reinforcing) nature of psychological and emotional outcomes on different aspects of health.

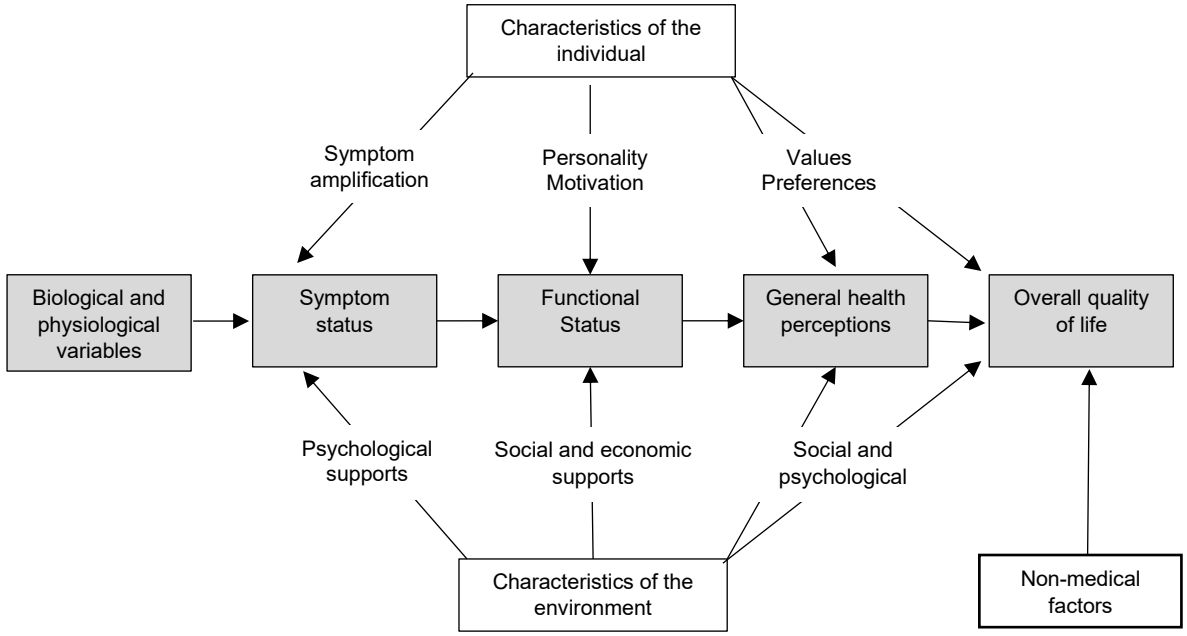


Figure 9
Conceptual Model of The Components of Health-Related Quality of Life, Wilson and Cleary (1995)

Psychological-emotional factors are not depicted as main components, but they are noted to influence all levels of the model (including physiological symptoms,

functional status, and general health). The influence of cognition and related thought processes are implied but not specifically discussed. Another example of an integrated model (Allan et al., 2016; see Figure 10) depicts a feedback loop between executive function and physiological health via health behaviour.

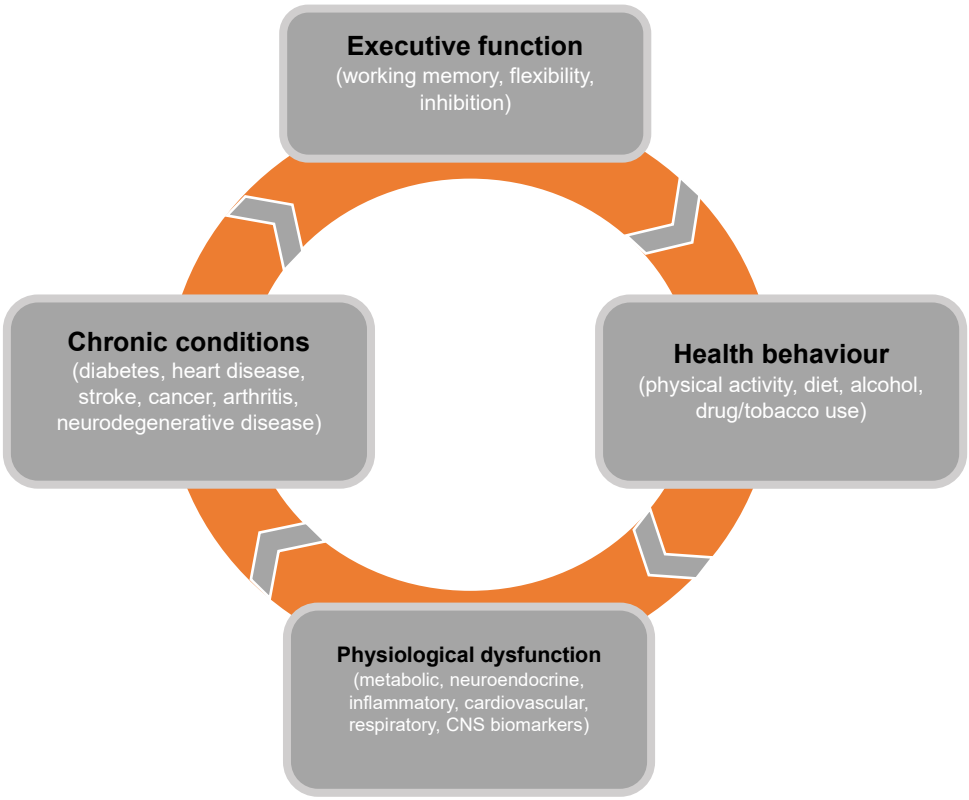


Figure 10
Cyclical Model of the Relationship Between Executive Function, Health Behaviour and Disease Processes (Allan et al., 2016)

Negative health behaviours are viewed as problems in self-regulation (inhibitory control) which lead to metabolic dysfunction and chronic health conditions. This is a parsimonious model which includes cognition but does not consider neurocognitive-emotional factors that could be important pathways and moderators of effects.

Leventhal's Self-Regulatory Model of health behaviours (also known as the Common-Sense Model; Leventhal, Meyer & Nerenz, 1980) takes the

acknowledgement of neurocognitive influence on health a step further, explaining how neurocognitive representations and coping responses in-turn can affect health behaviour (Leventhal et al., 2016). Not only this, but the model differentiates between emotional and cognitive coping responses, and notes that both types of response can influence emotional and health outcomes. This model could therefore be applied to both mental and physical health.

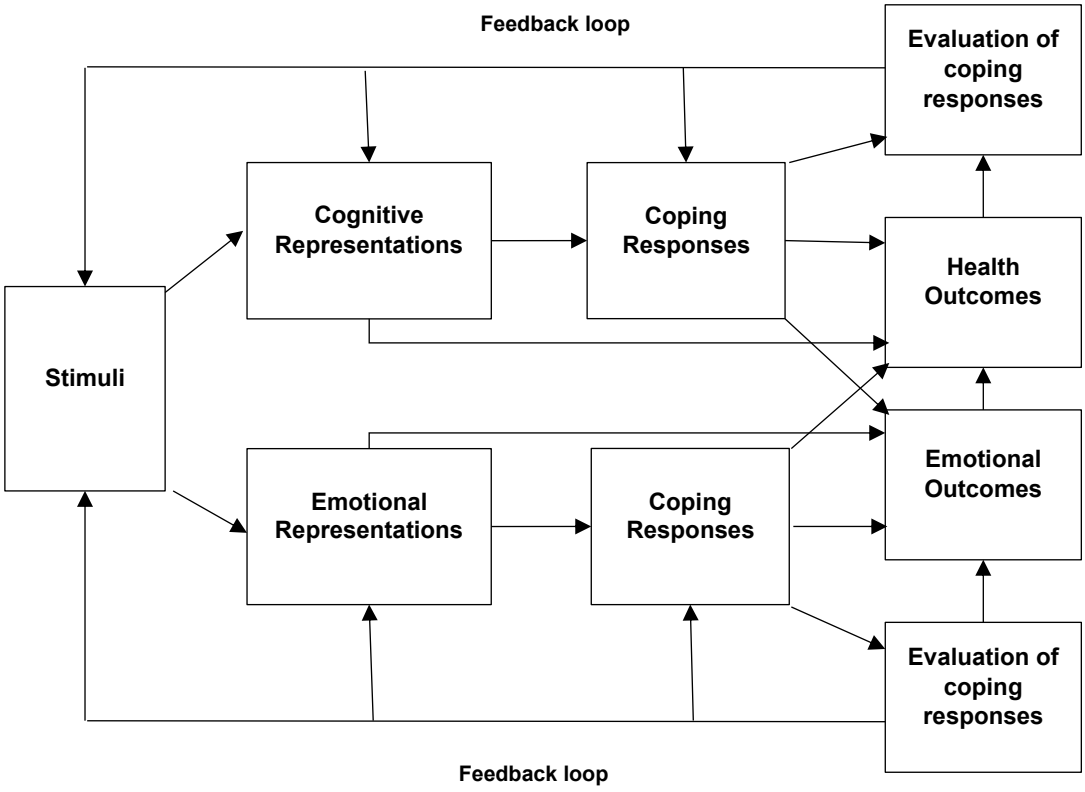


Figure 11
Leventhal's (1980) Self-regulatory Model (SRM)

The SRM explains the influence of attitudes to illness and ways that experience shapes cognition, emotion, and health outcomes through coping responses (cognitive/behavioural). The model was validated by meta-analytic findings (Hagger & Orbell, 2003) which confirmed good support for the impact of the

cognitive-emotional representation of illness (particularly perceived controllability and consequences) on coping strategies and health outcomes.

So how might a predisposition to greater emotional versus cognitive processing (e.g., elevated threat perception) impact self-regulation and health outcomes? And what mechanisms might underlie these individual differences? A further model, the **Social Information Processing (SIP)** model helps explain the influence of faulty cognitions on behavioural outcomes (see Figure 12).

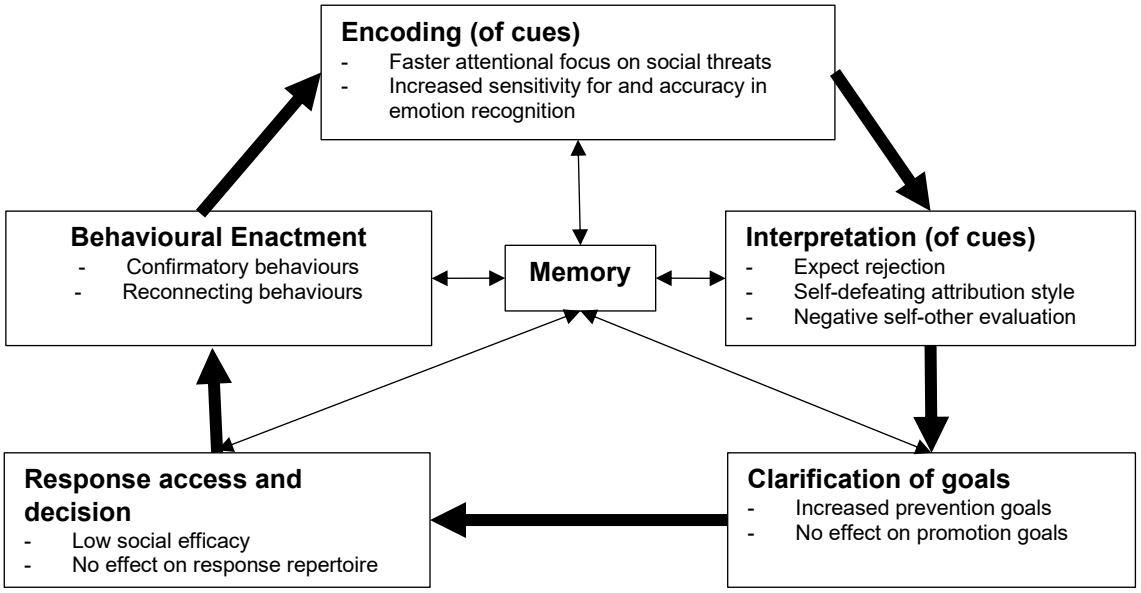


Figure 12
The Social Information Processing (SIP) model.

Crick and Dodge (1994) originally used the SIP to explain cognitive processing biases in childhood aggression, but Spithoven et al. (2017) discussed its utility in relation to self-regulation of health behaviour. The model assumes that the individual is pursuing a goal in line with their motivations (e.g. social connection) based on flawed information due to cognitive biases.

In a model of self-regulatory control, negative affect and excess adiposity could be conceptualised as health issues arising from a failure to regulate our emotions and behaviour based on flawed cues/cognitive biases. The SIP model includes the influence of desired goals, whereas the SRM does not assume that people are actively making top-down decisions for health. Instead, the SRM assumes that people are naturally acting, reacting, and trying to cope with the situation they are in, and that they update their behaviour based on their experience. The SRM does, however, include top-down control in terms of evaluating the outcome of coping responses, which can be used to reassess and alter behaviour. As these models reference self-control or self-regulation, they appear to assume or imply that the goal is healthy behaviour, but this might not be everyone's goal – or at the very least it might not be an overt priority. Additionally, the models do not incorporate the influence of faulty **physiological** signals that might compel a person to act against their own health interests outside of conscious thought.

The SIP model illustrates how biased negative cognitions (including social threat) can affect perceptions and subsequent behaviour through encoding and interpreting cues. These in turn are remembered and the negative effects are strengthened through experiential learning. This model has a clear explanation of cognitive factors, and some consideration of the influence of emotion on behaviour but it does not attempt to link this to physical health outcomes.

Spithoven et al. (2017) applied the SIP to biased cue processing in loneliness, but cue processing also has relevance to obesity. Jane Wardle developed the **behavioural susceptibility theory** to explain the genetic basis for individual differences in eating behaviour and subsequent obesity (Carnell & Wardle, 2009; Llewellyn & Fildes, 2017). The research identified appetite-related traits that put

individuals at higher risk of obesity, specifically, high food cue responsiveness and low internal satiety responsiveness. This means some individuals are genetically predisposed to overeating because they are predisposed to pay more attention to food (greater activation of the brain reward centres) and less likely to notice they are full (Simmons & DeVille, 2017). In support of this, Stevenson et al. (2015) examined research linking fullness, hunger, and thirst with differences in interoception. Findings indicated substantial individual differences in cue responsiveness and a link to obesity in a subset of individuals. Reduced interoceptive awareness was also reported in individuals with depression (but not anxiety) and was found to be systematically affected by attentional distractors such as TV watching while eating.

Summary: Influences on the proposed model. Several existing health models include elements of physical and mental health but do not seek to explain the interaction between them. In contrast to most research that focuses on examining unidirectional relationships between variables, the models depict factors that have a cyclical, **bi-directional influence** on health. This is an important necessity in understanding the complex relationship between health outcomes which will be essential to our understanding of mental and physical health. Models vary on the amount of importance they place on physiological, cognitive, and emotional factors, and the extent to which health behaviour is influenced by overt control, top-down control, or more reactive behavioural processes. The SRM draws a distinction between emotional responses and more thoughtful cognitive responses in influencing behaviour and health. The SIP emphasises ways that cognitions can be biased, providing flawed information as a basis for decisions/ behaviour that will affect health. These key ideas were taken forward in the development of the proposed model.

Like the SRM, the proposed model considers neurocognitive/ behaviour findings and makes a distinction between parallel cognitive and emotional pathways in self-regulation which might have a different impact on behaviour, and a different impact on physical health. Negative emotional pathways are influenced by underlying vulnerabilities, such as hypervigilance to threat which affect approach or avoidance behaviour, cognitive narrowing, and detrimental physiological health effects over time. Like the **CHRQoL model**, (Wilson & Cleary, 1995) and in keeping with a multidisciplinary understanding of health (Forsman et al., 2015; Schumann et al., 2014) the model combines research and concepts that cut across several layers of well-being (see Figure 13).

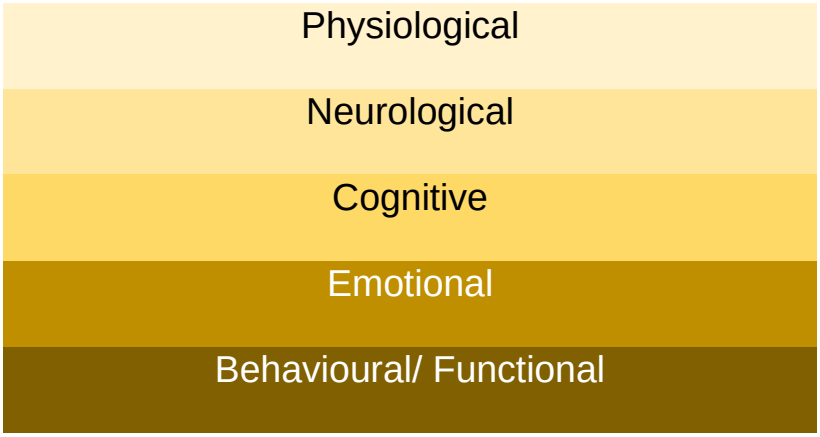


Figure 13
Layers of health and well-being identified by Wilson and Cleary (1995)

Proposed Model: Cognitive-Affective Model of Mental and Physical Health Interaction (CAMMPI)

The first part of this chapter provided a rationale and discussion of important features of existing mental and physical health models that influenced the current model. The CAMMPI model (Figure 14) attempts to provide a framework to understand the complex interaction between negative affect and adiposity, aiding clear explanations and aiming to move toward testable ideas. The model builds on concepts from existing models, and research from physiological, neurocognitive, and psychological approaches. It is hoped that this integrated model of mental and physical health can help to ground negative affect as a somatic issue and aid communication between different disciplines on this important public health matter (see Chapter 1) to facilitate transdiagnostic research and care. The rest of this section will outline the proposed model and explain the suggested mechanism of interaction between mental and physical health conditions.

Components of the model. This section will outline the key definitions and theoretic constructs used within the CAMMPI model with some salient research findings. The main constructs are as follows:

1) Executive function: attention and self-control (via inhibition),

2) Executive function as a limited resource system,

3) Attention funnelling

4) Repetitive negative thinking and impaired disengagement,

5)Separate but linked cognitive and emotional processing systems.

These five components are explained below.

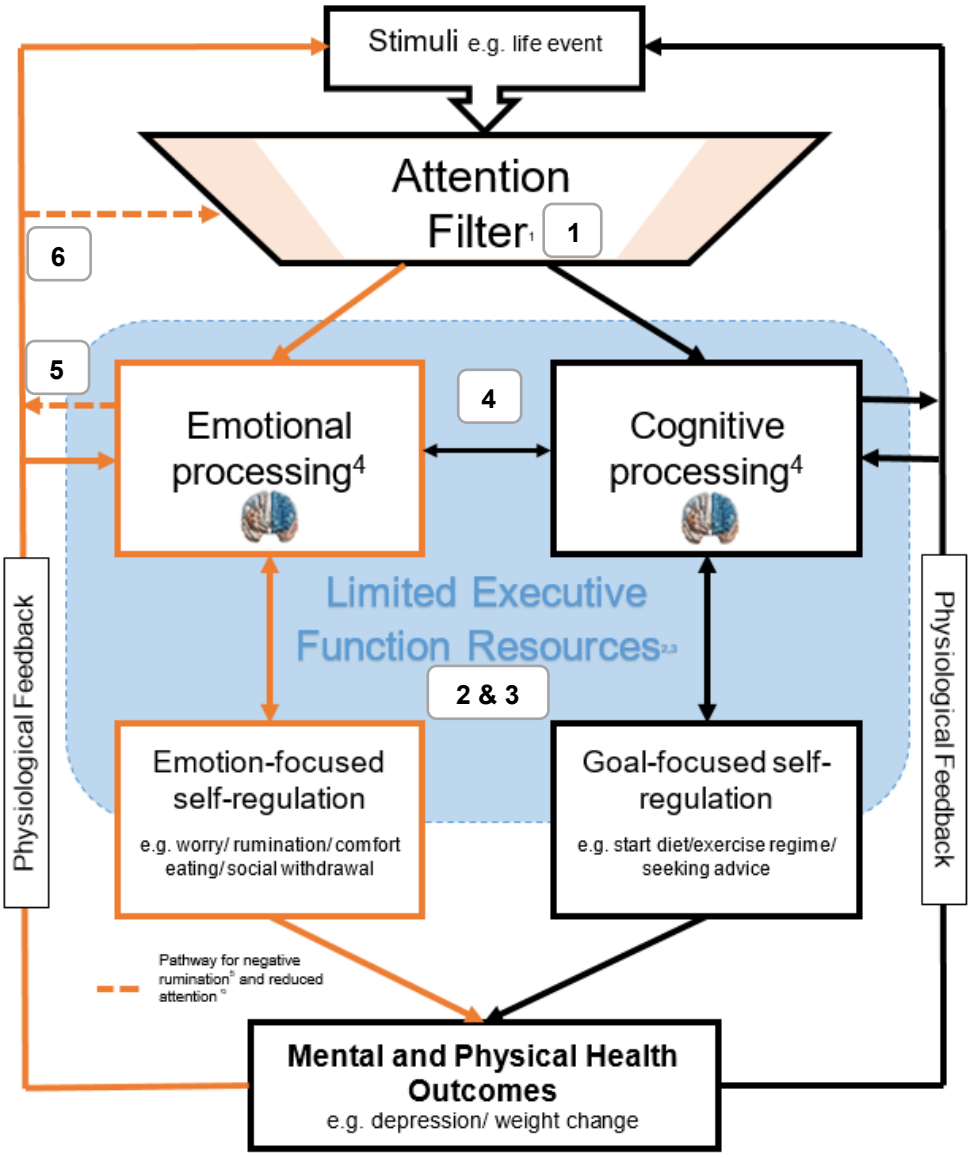


Figure
14 Cognitive-Affective Model of Mental and Physical Health Interaction CAMMPI Theoretical Model
Notes: 1 Attention funnelling in negative mood and affect as information (Clore et al., 2000,2001),

2 Unify and Diversify Model of Executive Function, (Miyake, 2000).

3 Strength Model of Self-Regulation (Baumeister, 1998)**.**

4 Cognitive and affective control (Hare & Casey, 2005).

5 Impaired disengagement hypotheses, (Koster et al., 2011).

6 Resource allocation hypothesis (Levens, et al., 2009)

1)Executive Function, Attention and Self Control. Executive functions are a group of related cognitive (thought) processes that underlie decision-making,

comprising of i) working memory, ii) inhibition and iii) flexibility / shifting (Miyake, Emerson, et al., 2000; Miyake et al., 2001). Hofmann et al. (2012) explain how these separate but related constructs influence self-regulatory behaviour: ***working memory*** helps us multi-task and keep control of our attention (e.g., allowing us to organise and monitor what we eat), ***inhibition*** allows us to suppress our impulses (e.g., to resist eating physiologically rewarding foods because we have set a goal to reduce our calorie intake) and ***flexibility*** allows us to actively switch between different goals (e.g. our ability to switch to a different food choice when considering what we have already eaten). Difficulties with any of these executive function and attention processes can impact our ability to establish and maintain goal-directed behaviour. Self-regulation is also highly dependent on selective attention, which underpins executive function (Diamond, 2013).

Based on neurocognitive research, Petersen and Posner (2012) outline three types of attention systems, which support executive function:

1 Alert system: associated with lateralised activation of the frontoparietal network. The right hemisphere addresses slow signals (tonic; more sustained activation e.g. preparing to respond to an expected stimuli) and left addresses faster signals (phasic; short-lived activation from any warning stimuli, hypothesised to suppress executive activity; Asanowicz and Marzecová, 2017).

2 Orienting systems: the Frontal Eye Fields and interparietal sulcus identify and track movement, and the temporoparietal junction (TPJ) and the ventral frontal cortex which direct switching/reorientation.

3 Executive Attention Systems: the cingulate and-opercular control system maintains sustained whole task attention, and the frontoparietal system relates to initiation and changes of tasks in real time. Difficulties with self-regulation could

therefore arise due to permanent or temporary issues with any one of these three systems which control our ability to sense or perceive a stimulus, the motivational value we place on that stimulus and ultimately, our motivation to approach or avoid stimulus (turning thoughts into action). The relevance of these systems to the regulation of eating behaviour are supported by a reviews of eating behaviour research (French et al., 2012) which strongly implicates physiological individual differences in the perceived reward value of food (stronger sensitivity to food cues and thus greater orientation toward food) and reduced ability to detect internal satiety signals (reduced ability to detect alerts from the body that one is full).

2)Executive function as a limited resource system. **The Strength Model** (Baumeister, Vohs, & Tice, 2007) theorises that self-regulation is a limited resource system that will suffer from 'ego depletion' with overuse (like a muscle). Extra cognitive demands or upregulation of emotional thoughts could increase cognitive load and divert resources away from inhibitory control. As executive function (working memory, inhibition, and cognitive flexibility) is important to our ability to perform basic organisation, taxing this resource could contribute to the difficulties observed in individuals with depression and potentially obesity with everyday tasks such as sticking to health behaviours and routines (giving in to easy rewards), and make it more challenging to switch mindsets toward generating problem-focused coping solutions. Persistent failure to plan and stick to health behaviours and routines would inevitably invite further negative impact on mental health through social comparisons and effects on self-esteem (see Chapter 2). Levens et al. (2009) found support for the impairment of depressed individuals in the controlled ordering and allocation of executive cognitive resources in complex dual process tasks. The

impairments were selective, only affecting complex dual tasks and the authors explain that the effects are likely to be due to impaired disengagement from ruminative thoughts that consume executive processing resources.

3)Attention funnelling. As our attention and cognitive systems have limited resources, we have developed ways of focusing on information that could be important to survival including learned rewards (such as food or social interaction) and indicators of threat. Morales et al. (2016) suggest that affect-biased attention can develop from infancy as individuals orientate toward affect-laden stimuli that represent danger or reward e.g. angry faces or food and this interacts with temperament (cue reactivity) and experience to prime our emotional, and executive attention orientation systems in the brain to initiate avoid or approach behaviour. In addition, in-line with the SIP model, Clore and Gasper (2000) suggest that strong emotions also have a funnelling effect on attention *in the moment,* making information seem more important and more likely to be attributed to (or misattributed to) your current focus. This can exacerbate existing negative biases. Morales suggests that affect-biased attention has a role in the development of mental health disorders (often associated with an overactive threat response) and obesity which is genetically linked to greater 'cue sensitivity' to the reward value of food and reduced sensitivity to satiety signals (Llewellyn & Fildes, 2017).

4)Repetitive Negative Thinking, and Impaired Disengagement. Repetitive negative thinking such as worry, and brooding rumination are hallmarks of internalising conditions such as anxiety and depression. In some individuals these thought processes are very difficult to stop. The **Perseverative Cognition Hypothesis** (Brosschot et al., 2006) explains how negative and repetitive cognitions

can prolong the body's stress response even after a stressful event has ended (or in future anticipation). Continued increase in HPA activation / 'allostatic load' and persistent negative emotions (Renna, 2021; Szabo et al., 2022) appear to result in negative health outcomes over time by causing low grade systemic chronic inflammation (Furman et al., 2019). As well as this increase in cellular 'wear and tear', increased perseverative cognitions could reduce cognitive resources that are available for problem solving and self-regulation (Watkins & Brown, 2002; Whitmer & Gotlib, 2012), especially when paired with the attentional funnelling effect of affect-biased attention (Clore & Gasper, 2000; Morales et al., 2016).

The **Impaired Disengagement Hypotheses** explains that some individuals may be caught in a loop of negative self-focused cognitions due to an attentional bias toward negative material coupled with impaired attentional control (Koster et al 2011). The most severe cases of repetitive negative cognitions include cases of post-traumatic stress disorder (PTSD) where individuals are suddenly cast back or unable to stop thinking about a traumatic event in the past (Nolen-Hoeksema et al., 2008; Wisco et al., 2023). To reinforce the relevance of rumination to health, PTSD is also associated with a greater number of chronic physical health conditions than any other anxiety disorder (including brain, heart, metabolic, autoimmune, and bone/joint related conditions; Sareen et al., 2005 cited in Schumann et al., 2014).

In terms of impaired disengagement, the associative network or frontoparietal task-orientated prefrontal cortex are the brain areas most associated with executive function incorporating on-task activity and decision-making however there are several parts of the brain that are anti-correlated (i.e. negatively correlated) with these areas. Areas of the default mode network (DMN; Fox & Raichle, 2007) show

little activity when a person is engaged in a task but become much more active when a person is at rest. This is because the DMN is focused on self-based or internal thought processing (Stawarczyk et al., 2011). In normal function there is a clear distinction between brain activity of these networks during task and rest whereas failure to down-regulate the DMN during tasks has been observed in mental health conditions such as depression (Bartova et al., 2015; Brzezicka, 2013; Delaveau et al., 2017). DMN impairment has also been noted in relation to increased body mass (Beyer et al., 2017; Figley et al., 2016). The DMN may therefore have a role in impaired disengagement and the executive function difficulties associated with affective mental health conditions (and perhaps obesity).

5)Separate but Linked: Cognitive and Emotional Processing. Petersen and Posner, (2012) assert that neurocognitively, self-regulation (defined as our ability to override a dominant response) is controlled by a network involving the anterior cingulate and anterior insular with links to the prefrontal cortex (for use when inhibitory demands are greater). The anterior cingulate is an area just under the frontal/temporal cortices (see Figure 5 brain diagram) which is subdivided into areas for **cognitive control** (dorsal area shows functional connectivity to sensory areas in cognitive tasks) and **emotional control** (ventral area shows functional connectivity to limbic areas in emotional tasks). These findings are supported by neuroimaging studies (Bush et al., 2003). The dual competition framework details the role of the cingulate in mediating between the emotional areas of the limbic system and the cognitive executive control areas to influence behaviour (Pessoa, 2009).This area would therefore seem a likely area to influence the switch between more emotional or internal processing and task-based executive processing.

6)Neurocognitive Deficits as a Vulnerability Factor for Health Problems.

Neurological research suggests that the brain network most frequently attributed to executive function control (associative network) may compete for attentional resources with the emotional decision-making network (Limbic Loop; Alexander and Crutcher, 1990; Levens, et al., 2009; Watkins & Brown, 2002; Petersen & Posner), making behaviour regulation more difficult. Additionally, a growing body of work suggests that high body fat (adiposity) is itself a risk factor for executive function deficits (Fitzpatrick et al., 2013), but findings are conflicting, and the mechanism of effect is unclear. Of the three areas of executive function, inhibition deficits (motor inhibition and self-control; Scherbaum, et al., 2018) are most consistently related to adiposity and depression (Castaneda et al., 2008; Elliott et al., 2002; Stinson et al., 2018).

Applying the CAMMPI to Negative Affect and Adiposity

Following Study 1 it is conjectured that the link between BMI and negative affect (including loneliness) arises not just from eating to feel better, but from defects or misdirection of brain and bodily processes that allow people to monitor and regulate their behaviour. It is still unclear whether BMI and negative affect share the same pathway to affect health but both conditions are associated with emotion and cognitive problems which in turn can affect health behaviour. Based on available evidence, it is suggested that a propensity for uncontrollable repetitive negative thinking/ impaired disengagement overloads executive function capacity and therefore inhibition and self-regulation behaviour suffers leading to worse mental health outcomes and weight increase. This bias toward repetitive negative thinking

could result from neuro-cognitive processes affecting and perhaps biasing the way we sense and processes information e.g., hypervigilance to threat, individual differences in cue perception/reward processing, or emotional processing drawing attention and inhibition resources away leading to less efficient processing (slower reactions or more errors in tasks).

The combination of not being able to get away from persistent negative thoughts, and interference with problem-solving in daily life leads to a negative spiral of mental health outcomes and negative physical health effects over time. The mode of effect could be physiological e.g., stress-related changes in energy regulating hormones, neurological/neurochemical (changes to the number or activity of brain cells, receptors, or neurotransmitters in the central nervous system), and/ or persistent low-grade inflammation (Furman et al., 2019; Szabo et al., 2022).

Chapter Summary

In summary, there lacks an existing model to explain the interaction between mental and physical health. I therefore developed an integrated multi-disciplinary theoretical model to help explain the complex and novel ideas discussed. The Cognitive-Affective Model of Mental and Physical Health Interaction (CAMMPI) was outlined, followed by an explanation of how the model can help explain the bi-directional relationship between negative affect and adiposity via cognition. The model seeks to provide a parsimonious, joined-up explanation for the link between internalising mental health conditions and physical health conditions that takes account of interdisciplinary evidence across neurological, cognitive emotional and behavioural approaches to health and well-being. This could help to promote the

importance of mental health components in our overall understanding of well-being and research/treatment foci.

Although it would not be feasible to fully test the proposed model within a PhD, Study 1 & 2 seek to identify robust variables and measurement methods that best explain the relationship between adiposity and negative affect, including various facets of negative affect (depression, anxiety, loneliness and repetitive negative thinking), adiposity measurements (such as WC, BMI or direct measures) and measures of neurocognitive function (like cognitive tests, brain scans and self-report). The overall aim is to contribute to a joined-up understanding between mental and physical health and identify which relationships or variables warrant future study, in keeping with the ROAMER Project research goals (Forsman et al., 2015).

4.0 Study 1 Body Mass and Loneliness

My Role	Study Rationale	Method	Results	Discussion	Implications

This chapter provides a rationale for Study 1 methods and discusses the findings in context of the wider aims of the thesis i.e. gaining an understanding of the interaction between negative affect and adiposity. The study was conducted in 2016, and the findings were published in 2018 in the Journal of Youth and Adolescence see Appendix D Study 1 Manuscript (Qualter et al., 2018).

Study 1 built on previous work (Nowland, 2014) within a University of Central Lancashire research team that examined the health and social determinants of loneliness. The cross disciplinary research illustrates the importance of a unified approach to the study of mental and physical health (Forsman et al., 2015; Schumann et al., 2014). Research into the physical effects of loneliness on health implicated negative cognitions as a common cognitive risk factor; a negative thinking vulnerability relevant to internalising mental health conditions and worsening physical health over time (Schweizer et al., 2020). I aimed to find out whether adiposity and loneliness were mutually reinforcing over time as evidence for the possibility of a common mechanism of effect.

With consideration of the research goals set out by Forsman et al. (2015), Study 1 examined the link between body mass and loneliness, a prevalent mental health issue and source of socially mediated negative affect. The study used high quality secondary data to examine whether body mass and loneliness are predictive of one another over time, and whether the two conditions are mutually reinforcing

(reciprocal) in a sample of n = 1042 (Female = 572; Male = 470) from a large epidemiological study.

My Role

As this study was completed as part of a research team, this section clarifies the tasks I undertook in the study. My role consisted of the following: performing background research and proposing the investigation of BMI as a relevant health factor related to loneliness. I requested permission for access to the variables of interest from the Quebec-based research team, translated the questionnaire items and responses from the original French, and cleaned the data. I researched and devised the procedure to calculate the age and gender adjusted childhood BMI scores for an international sample and calculated total scores for loneliness. I compiled the data for analysis including performing checks for missing at random, mean centring the BMI variables and preparing descriptive statistics. I researched appropriate analyses. It was hoped to create a structural equation model, but this was not possible as the variables did not meet the assumption of independence and linearity. I assisted in planning and performing the hierarchical regression analyses and categorical ANOVAs. For the paper itself, I conducted the background research and wrote the initial draft of the introduction and method, helped in writing and interpreting the findings, checking drafts, and answered questions from reviewers.

Study Rationale

As outlined in Chapter 2, Loneliness has been recognised as a prominent cause of ill-health and earlier mortality (Wang et al., 2023). At the time of the study most research had been carried out in elderly populations which neglected the

experience of loneliness in youth and young adults. A systematic review of loneliness and chronic physical health conditions (Petitte et al., 2015) indicated that the relationship with obesity was understudied. They could not draw firm conclusions but found evidence of an indirect relationship with factors such as night-eating, depression, diabetes, poor sleep, and back pain. A more recent systematic review of six studies of obesity, loneliness, and social isolation (Hajek et al., 2021) found mixed evidence. Two longitudinal studies within the analysis found the onset of obesity was associated with increased loneliness, but studies reported opposing effects of gender. The studies relied on self-reported body measurements which are known to be biased.

Longitudinal methods

Health effects in response to variables like stress or loneliness appear over time, leading to different trajectories of health impacts depending on severity and chronicity (the immediate reaction to stress versus dynamic stress accumulation effects on the body; Zapf et al., 1996).

Cross-sectional studies are limited for various reasons. They cannot give the direction of causation between variables, and they have limited ability to control for the influence of additional unmeasured variables. Zapf et al. (1996) cautions awareness of non-constant third variables that vary systematically alongside the IV and DV (synchronous effects) are the most problematic as hidden mediators or moderators of what appear to be lagged effects. These can be especially hard to detect if the IV is not stable over time. They advocate Cook and Campbell's (1979) criteria in the identification of likely causal effects (covariation with ill-health, stressors which appeared before the ill-health developed and other plausible

explanations), emphasising that causal inference can never be proven despite a willingness to accept medical assertions as fact.

In longitudinal studies the time order of effects is used to narrow the direction of causal influences (because a health condition could not be caused by a future level of a variable, i.e. Time 2 IV cannot cause Time 1 DV). Longitudinal techniques are also the most suitable technique for measuring developmental changes over the life course (Pakpahan et al., 2015). Some Third (unmeasured) variable effects can also be controlled for in longitudinal analysis. Occasional variables can be ruled out if an effect is observed over multiple time periods, but background variables such as personality, sex and social status are likely to exert consistent effects over each time point. This can be addressed by controlling for demographic variables in the analysis. Structural equation models have the advantage of a high level of control over error variance. An alternative to a full structural equation model is a cross-lagged analysis. This uses a series of hierarchical regression analysis controlling for variance from the same measure at previous time points. Cross-lagged models would therefore allow the investigation of the influence of BMI and loneliness over time, to help us better understand the nature and direction of relationship between the variables by examining the unique variance explained by each variable at each time point. ANOVA were used to further investigate the cross-sectional association between the variables categorically by BMI cut-offs (see Table 4 and Chapter 2 for a discussion of BMI measurement). For completeness, a similar technique was used to categorically examine loneliness using centiles.

Previous studies have found a tentative association between loneliness and body mass (Lauder et al., 2006), but the area is under-researched, and mechanism/s of effect are unclear. Additionally, studies of body mass may use self-report rather

than robust measurement techniques (Stanley & Bohnert, 2011) or focus on older adults, neglecting the investigation of the link between these variables in adolescent age groups. To further the understanding of the current and prospective relationships between loneliness and BMI, Study 1 used regression analyses to investigate the relationship between these variables over time, using a large longitudinal data sample collected using robust epidemiological methods.

Method

Data for the study was collected as part of the Quebec Longitudinal study of Child Development (QLSCD) which has followed the health of a cohort of children born in Quebec in 1997-1998 (from around 4 months old - see QLSCD website https://tinyurl.com/52fr26m2). Data collections took place every one to two years and included a wide range of measures of health and wellbeing administered by trained researchers (this included anthropometric measurements taken per clinical guidelines).

The current study focused on data for the years 2008 (age 10), 2010 (aged 12) and 2011 (aged 13) when self-reported loneliness was incorporated into the study (mid-way through the second phase). Data for 2013 and 2015 were also requested, but a single item loneliness measure was used at these time points, which was not efficacious for longitudinal comparison.

As well as making good use of participant data that has been collected at great time and expense (in accordance with ethical research practice), the QLSCD used robust data collection practices enabling analysis of a high-quality large longitudinal data set that would not normally be available within the resources and

timescales for an individual doctoral thesis. See Appendix A Ethics & Consent for confirmation of ethical approval.

Measures

Loneliness. Loneliness was measured using a 3-item UCLA Scale (Rotenberg, MacDonald, et al., 2004; Rotenberg, McDougall, et al., 2004) yielding a score between 3 (hardly ever lonely) and 9 (often lonely), see Table 5 for items and scoring.

Table 5

Loneliness Items

Item	Original Items	QLSCD Items
1	"How often do you feel that you lack companionship: 1. Hardly ever, 2. Some of the time, or 3. Often?"	"In the last 2 weeks I have had people to talk to: 1. I have had no one, 2. I have had some people; 3. I have had lots of people" [**reverse scored]**
2	"How often do you feel left out: 1. Hardly ever, 2. Some of the time, or 3. Often?"	"In the last 2 weeks I have felt left out:" 1. Feel really/very left out; 2. I feel a little left out; 3. I do not feel left out." **[reverse scored]**
3	"How often do you feel isolated from others? 1. Hardly ever, 2. Some of the time, or 3. Often?"	"In the last 2 weeks I feel alone: 1. I do not feel alone; 2. I sometimes feel alone, 3. I feel alone all the time."

Body Mass Index. Participants' height and weight were measured while wearing light clothing and no shoes by training research assistants. Each measure was taken twice and if it varied by more than 0.5 cm for height or 0.2 kg for weight, a third measurement was taken. Where multiple measures were recorded for a participant, an average was calculated. For those under 18, BMI is calculated using sex and age-specific growth curves (to take account of child growth). Methods outline by Cole et al. (2007) were followed to convert mean height and weight to BMI - the International Obesity Task Force procedure used BMI in preference to

percentiles as it has better international comprehension and was more relatable to adult body mass (Lobstein et al., 2004).

BMI data was converted to z-scores for the analysis (loneliness data was also mean centred). While reducing BMI to z-scores removes some of the individual variance in the data, it allows more helpful conclusions to be drawn relating to the incremental changes of IV required to produce per unit changes in the dependent variable and helps address skew/normality issues. I therefore examine the difference between the variables rather than the absolute values of the variables themselves. Z-scores were calculated using internal references (sample mean).

Demographic variables. Gender (sex at birth) and income sufficiency were both used within the analysis to control for different rates of growth and material disadvantage. See Qualter et al. (2018) for further detail on the calculation of income sufficiency which is a standard governmental metric of living standards/relative poverty in Canada.

Study 1 Results

Table 6 summarises the mean levels of BMI and loneliness for participants (n=1049) at each time point (Time1, Time2, Time3). Figure 15 shows the frequency of loneliness scores using the response terminology; around 56-59% of respondents were hardly ever lonely, 38-40% were sometimes lonely and 4-5% were often lonely. Table 7 shows the mean loneliness for participants by weight category and Table 8 show the mean BMI for participants by loneliness quartile.

Table 6

Sample Demographics (Mean and Standard Deviations for Age (months), Raw BMI, and Loneliness at Time Point 1,2&3)

Mean (SD)	T1 Age 10 (M=121.70, SD= 3.10)		T2 Age 12 (M= 145.60 SD= 3.05)		T3 Age 13 (M=157.60 SD= 3.12)	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**
BMI	18.30 (3.17)	18.35 (3.20)	20.06 (3.75)	19.83 (3.87)	21.02 (3.89)	20.73 (4.26)
Loneliness	3.83 (1.18)	3.87 (1.24)	3.82 (1.16)	3.72 (1.18)	3.95 (1.31)	3.81 (1.27)

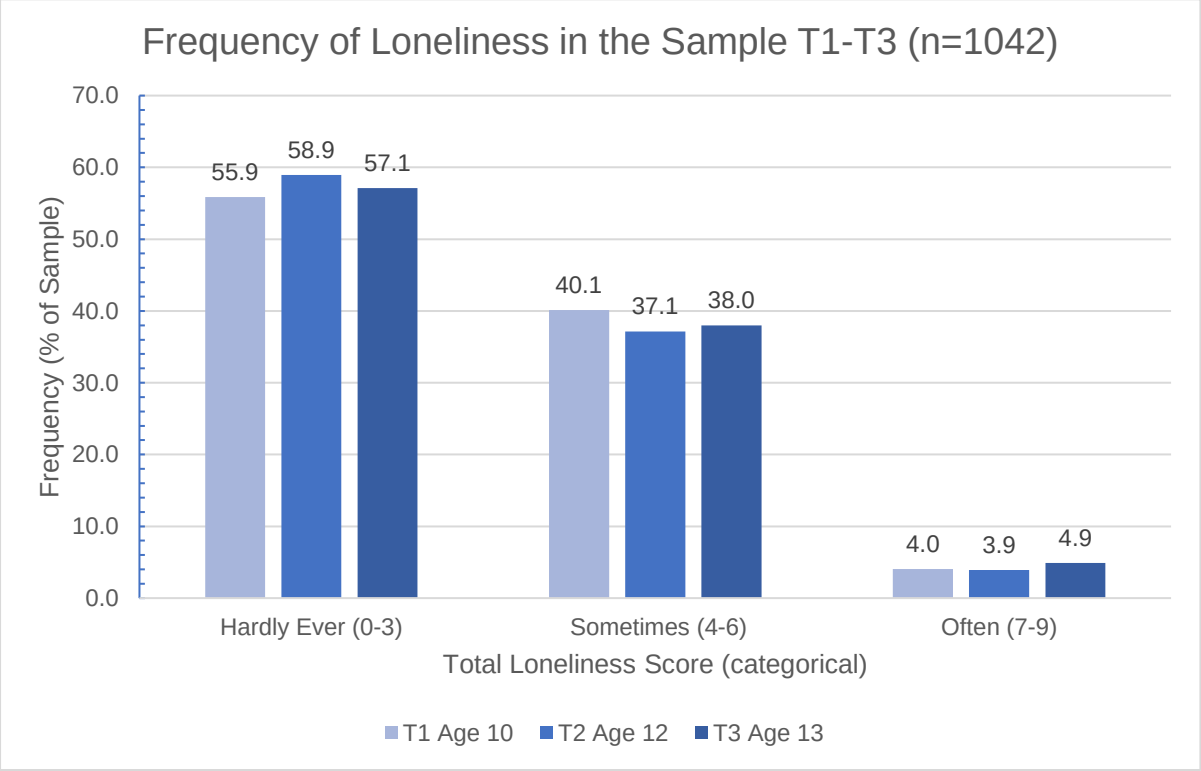


Figure 15

Categorical Loneliness Score T1-T3

Weight Category and loneliness - ANOVA

One-way ANOVA investigating continuous loneliness by BMI category (or class) revealed no significant differences (by BMI category) at any of the three time periods: T1 (age 10 years) females, $F(3, 568) = .99$, $p = .395$, males, $F(3, 466) = 1.0$, $p = .367$; T2 (age 12 years) females, $F(3, 568) = .74$, $p = .528$, males, F (3,

466) = 1.84, p = .140; and T3 (age 13 years) females, F (3, 568) = .01, p = .998, and males, F (3, 466) = .393, p = .76. Thus, in the current population sample, weight category (underweight, normal weight, overweight, obese) was not associated with concurrent reports of higher loneliness during adolescence for males or females.

BMI-z and Loneliness - Hierarchical Regression Analyses (HRA).

A series of HRAs (see Table 8) examined concurrent and cross-lagged effects of BMI-z and mean centred loneliness within timepoints and over time (from one time-point to the next). Baseline levels of the dependent variable, gender and income sufficiency were all controlled at separate steps, as were the quadratic terms to check for curvilinear effects. Bootstrapping was used to verify the reliability of the findings. The variables were very stable from one time-point to the next; BMI coefficients ranged from .89 to .95 (SE=.02), and loneliness from .27 (SE=.03) to .47 (SE=.04). There was no effect of income on either variable over time; BMI β = .00 [SE = .02] to −.02 [SE = .02]) or loneliness (β = .05 [SE = .06] to −.11 [SE = .07]).

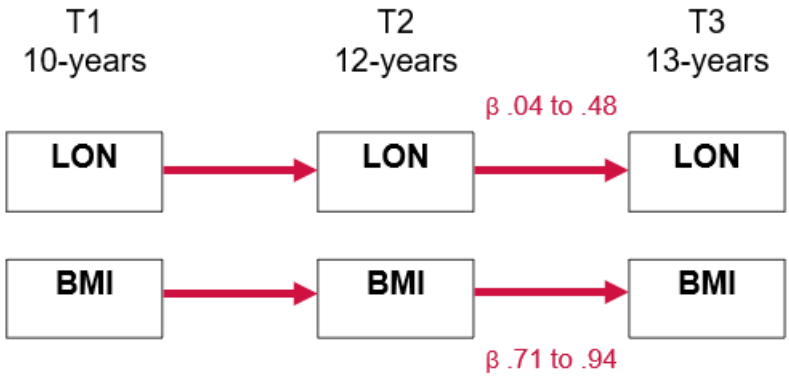


Figure 16
Study 1 Significant Within -Variable Associations between Timepoint 1, 2 and 3
Notes: LON= Loneliness

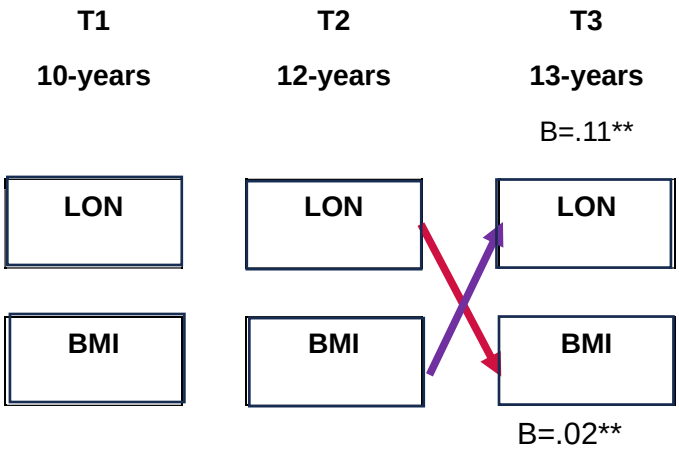


Figure 17
Study 1 Significant Between -Variable Associations between Timepoint 1, 2 and 3
Notes: LON= Loneliness

BMI predicting loneliness. A significant linear effect of weight status at T2 was observed on loneliness at T3 (β = .11, SE = .04, p = .007), suggesting that higher BMI at age 12 predicted higher loneliness at age 13 years.

Loneliness predicting BMI. A significant quadratic effect of loneliness at T2 (age 12 years) on BMI at T3 was also observed. Here loneliness interacted significantly with gender to predict BMI at age 13 years, β = .02, SE = .01, p = .007. Girls who were lonelier at age 12 had greater BMI at age 13, whereas boys who were lonelier at age 12 tended to have lower BMI at age 13.

Table 7

Mean and Standard Deviations for Loneliness in Each IOTF Weight Category at Time Point 1-3

Time	Uw		NR		Ow		Ob	
(age)	BMI <18.5		18.51-24.99		BMI >=25		BMI>=30	
	FEMALES	MALES	FEMALES	MALES	FEMALES	MALES	FEMALES	MALES
T1 (10 yr.)	3.61 (.99)	4.12(1.28)	3.80 (1.15)	3.80(1.37)	4.06(1.34)	4.02(1.37)	3.67(1.12)	3.86(1.36)
N=1042	36	26	470	325	99	90	30	29
T2 (12 yr.)	3.80(1.37)	3.56(1.00)	3.74(1.12)	3.79(1.19)	3.93(1.16)	3.79(1.17)	4.24(1.32)	3.58(1.23)
N=1042	35	25	384	311	116	98	37	36
T3 (13 yr.)	3.97(1.57)	3.50(1.23)	3.86(1.28)	3.73(1.22)	4.05(1.31)	3.92(1.12)	4.55(1.57)	4.29(1.81)
N=1042	30	28	384	299	120	105	38	38

Note. Uw = Underweight (Grade I, II, III were combined due to low numbers), N*R*=Normal Range, Ow= Overweight, Ob = Obesity (Grade I and II combined due to low numbers). Possible loneliness scores ranged from 3 to 9. N= 1042 (Female = 572; Male = 470).

T1=10 years old, T2=12 years old, T3=13 years' old

Table 8

Mean and Standard Deviations for BMI by Loneliness Quartile at time point 1-3

Time	**1st**		**2nd**		**3rd**		**4th**	
(age)	**Lon Quartile**		**Lon Quartile**		**Lon Quartile**		**Lon Quartile**	
	FEMALES	**MALES**	**FEMALES**	**MALES**	**FEMALES**	**MALES**	**FEMALES**	**MALES**
T1 (10 yr.)	22.99 (3.18)	18.17 (.48)	18.45 (2.99)	18.36 (3.20)	18.74 (3.45)	18.30 (3.22)	17.93 (2.45)	18.49 (3.91)
N=1042	3	2	441	347	123	114	5	7
T2 (12 yr.)		17.07	19.87 (3.48)	19.84 (4.00)	20.77 (4.56)	19.73 (3.28)	20.32 (4.97)	20.85 (4.74)
N=1042	0	1	449	373	116	91	7	5
T3 (13 yr.)		15.96	20.78 (3.49)	20.49 (4.13)	21.72 (4.76)	21.37 (4.06)	21.18 (5.60)	23.52 (8.60)
N=1042	0	1	421	359	140	101	11	9

Maximum Loneliness Score 9. Loneliness Quartiles were defined as follows: 1st Quartile 1-2.25; 2nd 2.26-4.50; 3rd 4.51-7.75; 4th 7.76-9

Study 1 Discussion

Study 1 identified the point where physical changes in body sizes and the socially mediated negative feelings of loneliness show an association, between childhood and adolescence. The analyses looked at both predictive directions and found body mass had a larger effect on lonely feelings (negative emotions or cognitions) than loneliness had on body mass. The effect is not observed cross-sectionally but develops over time, becoming notable from 12 to 13 years-old. The study findings do not rule out the possibility of a common mechanism of effect and BMI and loneliness maybe mutually reinforcing, due to the stability of the constructs over time. Individuals who have prolonged trajectories of *both* increased loneliness and high BMI are likely to be a vulnerability group for metabolic health problems and neurocognitive effects in later-life.

Background research revealed that negative affective conditions and excess adiposity both increase inflammation (Creswell et al., 2012; Hawkins et al., 2015; Rodríguez-Hernández et al., 2013). A more recent systematic review of loneliness and inflammation (Smith et al., 2020) found that loneliness was associated with increased levels of Interlukine-6, however there were only two studies that met criteria for inclusion- one was significant and one only approaching significance. Smith et al., hypothesise the mixed findings could be due to indirect effects e.g. that loneliness is indicative of a genetic propensity for more reactive inflammatory response in the presence of a stressor (indicating a difference between acute and chronic presentations), or that loneliness arises because of a stressful experience, so it is a co-occurrence rather than a causal factor. A substantial genetic study examining associations with gene variants that control for loneliness and social interaction (Day et al., 2018) found evidence of a positive predictive relationship for

BMI causing loneliness (larger body size resulting in more lonely feelings) but not the reverse. They found that genes related to loneliness had a lot of overlap with that of depression and that depression was bi-directionally associated with BMI. Depression causing BMI being the dominant direction of effect.

Strengths and Limitations of Study 1

In line with research goals (Forsman et al., 2015), Study 1 examined two conditions of international public health concern: obesity and loneliness. The data was obtained from a major longitudinal epidemiological study which used robust collection and measurement techniques that would not normally be available within the resources and timescales for an individual doctoral thesis. Many other large health studies tend to rely on self-reported body measurement which is subject to bias. The study findings underlined the importance of considering temporal effects (time taken to see the impact of a mental health variable on a physical one), individual differences, the presence of linear and curvilinear effects.

Despite the large initial sample, low numbers of participants at extremes of low weight meant that the association between BMI and very low weight could not be investigated. It is noted however that extreme low weight can be life-threatening, meaning those individuals may be more likely to be found in clinical settings. Although the BMI classifications at the extremes of BMI were reduced to four groups the number of participants in each group was still uneven. The power of the cross-sectional analyses (finding no concurrent association between BMI and loneliness) would therefore have been reduced making it harder to detect between group differences.

While BMI is a common and simple measure it does not directly measure fat mass, and it is recommended that further studies look at additional measures of adiposity to establish whether the effects are related to fat mass or physical size. In an adolescent population, this may present a challenge as there is less research on growth related expectations of different levels of fat mass for age. Gold-standard techniques such as dual-energy X-ray absorptiometry has been used successfully in child and adolescent populations. Recent research has also advocated the use of waist-circumference to height ratio as a closer approximation to fat mass in children (Agbaje, 2024).

In addition to significant differences between health variables, a further issue of interest is whether differences are clinically or subjectively meaningful (to the individuals concerned). While high BMI individuals in the study were lonelier, they did not identify themselves as being *very* lonely. This could mean that even moderate self-reported loneliness can affect health, that participants were downplaying how lonely they felt (e.g., for cultural reasons), or alternatively, that there are other shared variables at play that explain additional variance in the connection. The efficacy of the current loneliness measure could also be considered in future work. The UCLA 3-item loneliness scale (which the current measure was based on) is the most used measure of loneliness (and is recommended by the UK government for loneliness research), however a recent systematic review suggests that it’s psychometric properties are lower with child/adolescent groups than young adults (Cole et al., 2021).

Low beta values are to be expected when examining such multifaceted constructs over time, however loneliness explained less than 1% of variance. One of the reasons for the low variance necessarily arose from the statistical approach to

control for gender and economic sufficiency (as well as measures from the initial time-point). These controls help to show that there was a tangible change in the DV over time (over and above the effects of the demographic control variables) however removing variance may lead to an underestimate of the effect size (as some legitimate variance is removed). Miller and Chapman (2001) question the technique of removing shared variance of covariates on a DV (unless it is being applied to randomised groups).

Additional co-variables that were not considered in this study include depression (data was not available at all timepoints), and this could have been an important co-variable. Research suggests that shared variance with depression is likely to have mediated the effects of the variables of interest. Of particular interest is the shared facets of negative repetitive cognition or rumination and the impact on executive functions that control planning and decision-making in the brain. A transdiagnostic study (Schweizer et al., 2020) modelling the relationship between negative cognitions and several internalizing mental health conditions (depression, social anxiety, separation/panic) indicated a shared common cognitive vulnerability between them and associated physical symptoms (.86).

Impact of the Research

The study was accepted for publication in a good quality journal illustrating its quality and contribution to scientific understanding of the interaction between BMI and loneliness (a source of socially mediated negative affect). The investigation showed further nuances which have relevance for research and measurement of these variables. While there were significant associations between a continuous BMI

measure and loneliness, the effect was non-significant for BMI weight categories. BMI weight categories are purported to be clinical gradings of physiological health risk (NICE, 2014), and as such (considering the significant association) one might expect a relationship between these categories and mental health risk (e.g. a significantly greater risk of loneliness for those categorised as morbidly obesity compared to normal weight), but this was not the case. It seems that the weight categories themselves are not meaningful to mental health risk in the same way as metabolic health risk is purported to be.

Key outcomes of the study include reiteration of the importance of the early adolescence as a key time for support and intervention for socio-emotional and physical health. Also, females with co-morbid moderate to high BMI and loneliness are identified as a potential vulnerability group for future metabolic health problems. The study provides evidence of a reciprocal predictive association between loneliness and body mass and makes an important contribution to our understanding of the temporal relationship between variables for two important physical and mental health conditions. Further study should help identify whether there is a shared underlying mechanism of effect. One way to do this is to consider factors that link mental and physical health conditions. The following section contains a discussion of potential mechanisms of effect.

Implications for the Mechanism Between Adiposity and Negative Affect

Schumann et al. (2014) outlines general factors that could explain the association between physical health conditions and mental health co-morbidities (Table 1, see Chapter 1). These include shared *predispositions* such as genetics,

temperament or personality traits, shared *risk factors* such as trauma, lifestyle, social support, negative cognitive processing, and shared *mechanisms* including health behaviours, repeated or chronic exposure to stress (Hypothalamic-Pituitary Axis activation) which causes adaptations (allostatic load/ wear- and-tear) and inflammation. These predispositions, risk-factors and mechanisms were considered in relation to the explanation of the reciprocal effects between obesity and loneliness. Specifically, the following overlapping areas are considered: role of **health behaviours** (physical activity and emotional eating), a **common vulnerability** (hypervigilance to threat and cue reactivity), **cognitive processing systems** (negative thought processes), and the possibility of a **common psychiatric-somatic comorbidity** that explains the link between the conditions (depression).

Health Behaviours

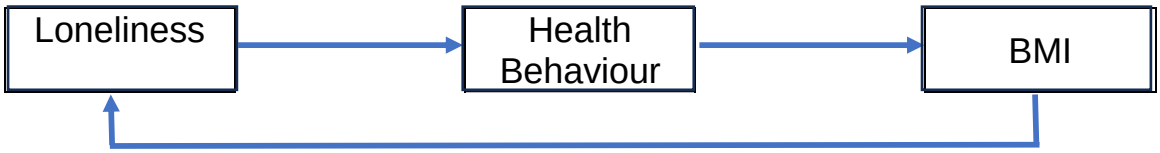


Figure 18
Theoretical Association Between Loneliness and BMI Via Health Behaviour

As loneliness is linked to negative health behaviours (Lauder et al., 2006), Hawkley et al. (2009) indicates that loneliness may affect BMI via behaviours like reduced physical activity or poor eating habits (see Figure 18)., but support for this hypothesis is variable. As a means of comparison of possible effect sizes of health behaviour, in a longitudinal study of BMI it was noted that self-reported health behaviours (healthy diet, and regular exercise) contributed about 20% of obesity variance (Byth et al., 2022) which is slightly less than the variance explained by self-

esteem in the same study (25%). Byth et al. (2022) acknowledge the likely systematic effect of social desirability bias on the accuracy of their self-reported body measurements (studies using researcher measures had lower effect sizes). This underlines the importance of social desirability in body measurements (how we think other people think about our body size) and means the likely effect of loneliness-induced health behaviour is small, however the effects of loneliness on BMI are also small.

Physical Activity. In the initial work on physical activity cited by Hawkley et al., (2009), lonely individuals did not show more sedentary behaviour than a non-lonely group (Lauder et al., 2006). Meta-analytic findings indicate overall physical activity does not have a significant effect on loneliness (Schrempft et al., 2019), and in systematic review, physical activity has been shown to reduce feelings of loneliness but this likely depended on social support during the physical activity (Pels & Kleinert, 2016). Notably the review did not examine the effects in the young to middle adult age range (25-44) but overall, these findings tend to indicate that the link between loneliness and BMI is less likely to be enacted by reduced physical activity.

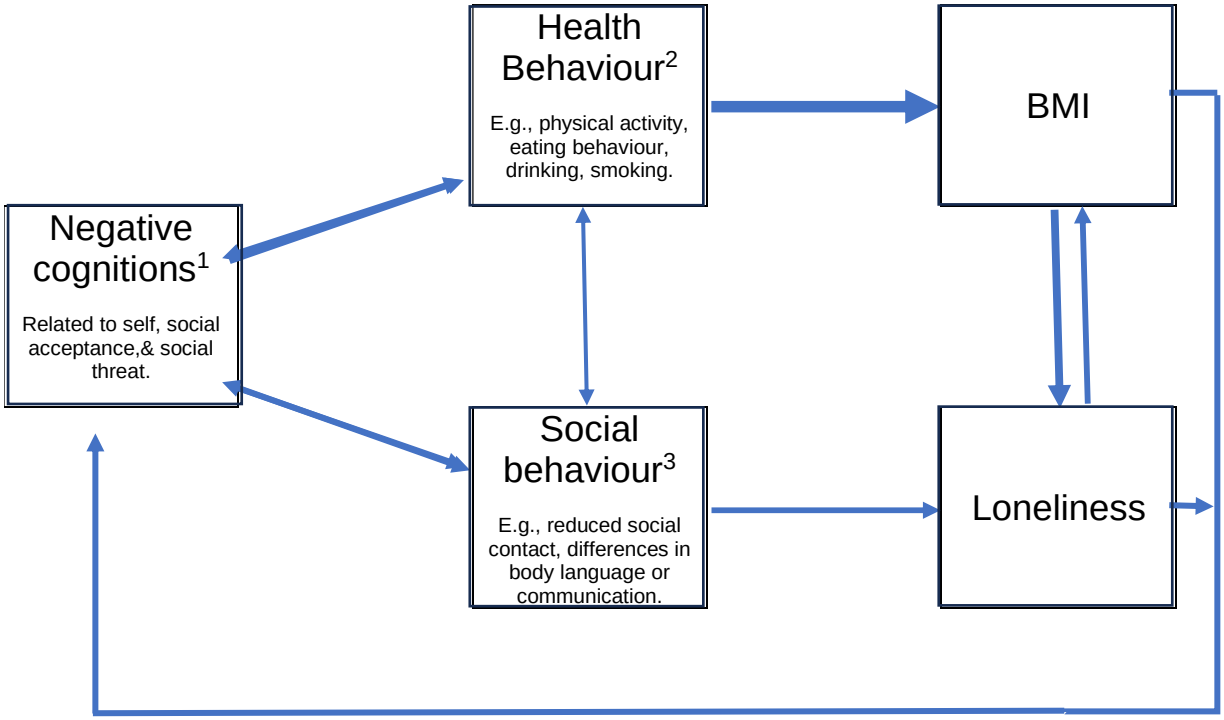


Figure 19

Theorised Interactions Between Loneliness and BMI

Notes: 1= Within this explanation, a predisposition to negative cognitions (and /or physiological weight-gain) is likely to precede (and underly) the behaviour which feeds into loneliness and BMI. Specific negative cognitions could involve e.g., low self-esteem, social acceptance, hypervigilance.

2= Obesogenic behaviour could be encouraged by negative cognitions such as comfort eating to feel better or lack of exercise due to lack of motivation.

3= Reduced social behaviour could be the result of choice (bad experiences reducing motivation to approach others) or being socially excluded by others (this could result from body language, communication issues or violation of social norms). Reduced social behaviour could also affect health behaviours as it may be harder to motivate yourself to exercise without social support.

Emotional Eating. Another potential mechanism offered for the effect of loneliness on BMI is emotional eating behaviour, such as disinhibited eating (Rotenberg & Flood, 1999), or impaired self-regulation (Salvy et al., 2011). Loneliness is implicated in eating disorders such as bulimia along with lack of perceived social support (Makri et al., 2022). In a study of loneliness and dyadic eating patterns a significant association was found between loneliness and emotional eating (e.g. eating in absence of hunger; Mason, 2020), however, despite the apparent association between loneliness and emotional eating (and intuitive

sense that weight gain necessitates positive calorie balance), the evidence for a direct link between self-reported emotional eating and BMI is weak. Bongers and Jansen (2016) question the validity of emotional eating as a concept and argue that purported effects are due to a subgroup of individuals who have an underlying vulnerability, toward low self-control (such as cue reactivity), and high motivation to eat, with little effect of the emotional component. This is supported by genetic studies (Llewellyn & Fildes, 2017) and further supports the possibility that the association between loneliness and BMI could be due to a shared genetic vulnerability. Relating this back to Schumann et al. (2014), this explanation fits with an explanation of shared predispositions, specifically related to both positive valence (greater approach behaviour to food) and reduced attention to self-regulation cues.

A Common Neurocognitive Vulnerability

Reward Learning Vulnerability*.* Leading from this interpretation of a shared genetic vulnerability, an alternative explanation for the findings in Study 1 could be a co-occurrence of negative affect (loneliness) and increased weight due to an underlying propensity for greater cue-reactivity (Tetley et al., 2010), which is relevant to reward sensitivity (affecting reward-based learning) and reduced inhibitory control.

Cognitive Processing Vulnerability. Loneliness and obesity are both implicated in impaired cognitive function, for example, Cacioppo and Hawkley (2009) note that lonely individuals have been shown to have impaired executive function including reduced auditory attentional control, poorer emotional regulation, threat-related cognitive bias, negative affect, and negative thought patterns such as rumination (which is also a core feature of depression; Zawadzki et al. 2013).

Obesity, and over consumption of high fat-sugar diets have also been linked to specific executive function deficits (Yang et al., 2018; Yeomans, 2017). In meta-analysis, Yang et al., (2018) found obesity was related to broad impairments in executive function in tasks of inhibition, cognitive flexibility, working memory, decision-making, verbal fluency, and planning (the only significant moderator was the task type). They also found that inhibition and working memory deficits appear at overweight weight status. Yeomans (2017) suggests that cognitive deficits in obesity may be linked to high fat/ high sugar diets which evidence (from human and animal studies) indicates may lead to structural changes in the hippocampus which impact future appetite control (Vicious Cycle Model). Small (2017) provides evidence that the effects of obesity and nutrition (excess fats/ glucose in the blood; insulin intolerance issues) are linked but separable. They provide a comprehensive and compelling account of the potential mechanism of the neuro-endocrinal effects of dopamine adaptation on neurocognitive impairment. The atrophy and structural changes in the brain associated with obesity outlined (Small, 2017) include reduced brain connectivity in the parietal and prefrontal cortex (PFC; important in executive function), hippocampal atrophy (relevant to learning memory), lower density grey matter and reduced numbers of dopamine receptors (important in reward learning/ sensitivity). Dopamine is essential in regulating multiple neurological processes and the neurological effects of disordered eating have important associations with addiction (Volkow et al., 2013) including shared issues with reward sensitivity (affecting the striatum e.g. with reduced D2 dopamine receptors which are linked to increased reward sensitivity and reduced PFC activity), self-control (affecting the pre-frontal cortex illustrated by reduced blood low and reduced grey matter density in the middle frontal gyrus), increased stress reactivity (increased amygdala/ limbic

system) with links to reduced interoceptive awareness (decreased cingulate and insula activity) which reflects reduced ability to sense when we are full and blunted pleasure when a reward is obtained.

The inflammation is also offered as an explanation for the neurocognitive effects seen in relation to obesity (Rodríguez-Hernández et al., 2013). Inflammation results in release of cytokines which area able to pass through the blood-brain barrier and effect cell death and regeneration and disrupt neuroendocrine systems such as that of dopamine and serotonin (Sullivan et al., 2015; Wilson et al., 2002).

Both loneliness and obesity are also linked to mild cognitive impairment and dementia in older age (Lara et al., 2019; Qu et al., 2020), as well as depression (Lavallee et al., 2021; Lee et al., 2021; Milaneschi et al., 2019; Park et al., 2020). Cognitive impairments could affect decision making processes around food choices, reduce an individual's capacity to monitor their food intake, sense when they feel full (Llewellyn et al., 2014), and monitor their goals/health behaviours (Nolen-Hoeksema et al., 2008; Skinner et al., 2012).

Excess Fats in the Blood. Cognitive effects could be further exacerbated over time by physiological processes caused by the increase in circulating fats in the blood (dyslipidaemia; Nielsen et al., 2012) which is linked to low level inflammation. These also affect cognition through changes in dopamine regulation (Small, 2017), creating or exacerbating neurochemical damage to brain structure, and function (Ward et al., 2010), and further contributing to the bias to threat-perception and negative self-related cognitions (van Reedt Dortland et al., 2010).

Other Psychiatric-Somatic Comorbidities – Underlying Depression

Loneliness and depression have each been shown to have a reciprocal relationship with obesity over time (Luppino et al., 2010; Vittengl, 2018). Loneliness and depression are separate but moderate to strongly correlated negative affect constructs (Cacioppo et al., 2006; Erzen & Cikrikci, 2018; Hawkley & Capitanio, 2015; Weeks et al., 1980) and loneliness frequently develops into depression over time (Lee et al., 2021). The two conditions appear to share core components of negative affect and repetitive cognition with a negative bias (Nolen-Hoeksema et al., 2008; Zawadzki et al., 2013).

In Study 1, consistent measures of depression were not available for the age ranges examined, and we were unable to rule out whether the link between loneliness and obesity was due to underlying depression, so this issue was examined further using existing research. Vittengl (2018) found the relationship between depression and obesity in females is moderated by **emotional eating**, **quality of social relationships** and **physical impairment** in daily activities. The important socio-emotional component in the relationship between these variables indicates that there is likely to be overlap in the variance in obesity which is explained by loneliness and depression. The effect sizes in both directions were small. Depression had a slightly larger effect on obesity over-time (T1 depression to T3 obesity Beta=0.065) than obesity did on depression (T1 obesity to T3 depression Beta = 0.059), however, obesity had a greater influence on the moderating factors than did depression (Vittengl, 2018; see Figure 20). Compared to the findings of Study 1, depression in the Vittengl study appears to explain slightly more variance in obesity than did loneliness.

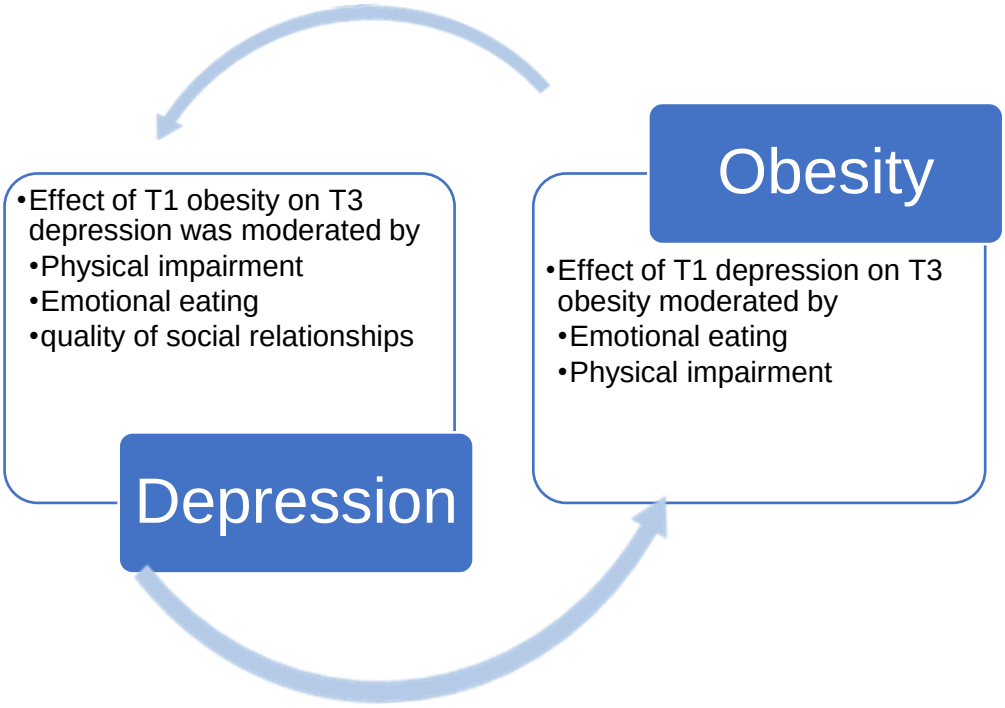


Figure 20
The Suggested Reciprocal Path Relationship Between Depression and Obesity and Vice Versa In Females Over Time, Based on Vittengl (2018).

Importantly, increased eating is only seen in a specific atypical depression subgroup, and it is more typical for depression to result in reduced appetite (Konttinen, 2020). This then implies that if depression ***is*** the link between loneliness and obesity, it relates to a subgroup of people with depression, not a general effect.

A discussion paper by Milaneschi et al. (2019) outlines biological mechanisms that could overlap between obesity and depression. The research indicates that obesity is only predictive of depression where the individuals ***also have an adverse metabolic profile*** (i.e., those with indicators of high inflammation, high blood pressure, insulin resistance etc). This strengthens the argument for a genetic vulnerability to factors such as inflammation and insulin resistance in the mechanism of association, which also impacts cognition.

Repetitive Negative Cognition

Rumination is a cognitive response to emotional distress (Response Styles Theory; Nolen-Hoeksema, 1991), which involves “repetitively and passively focusing” on symptoms and causes of that distress (Nolen-Hoeksema et al., 2008, p400). Nolen-Hoeksema et al. (2008) suggest that rumination increases negative thinking, impedes problem solving, and reduces instrumental action to improve a situation. Trying to inhibit unwanted ruminative thoughts can also make them more pervasive (Wenger, 1994; Whitmer & Banich, 2007), which can give a sense of failure and encourage further avoidant coping strategies. Rumination has prospective links to binge eating, and binge drinking with alcohol abuse (Nolen-Hoeksema & Harrell, 2002) that could be seen as coping strategies used to escape negative ruminative thoughts about self or situation. Rumination could also increase cognitive load (Ward & Mann, 2000) so that would make it more difficult to come up with problem-focused coping strategies, leaving people to use something more impulsive or fast-thinking (Kahneman, 2011), to help regulate their emotions (Aldao, 2012).

Summary

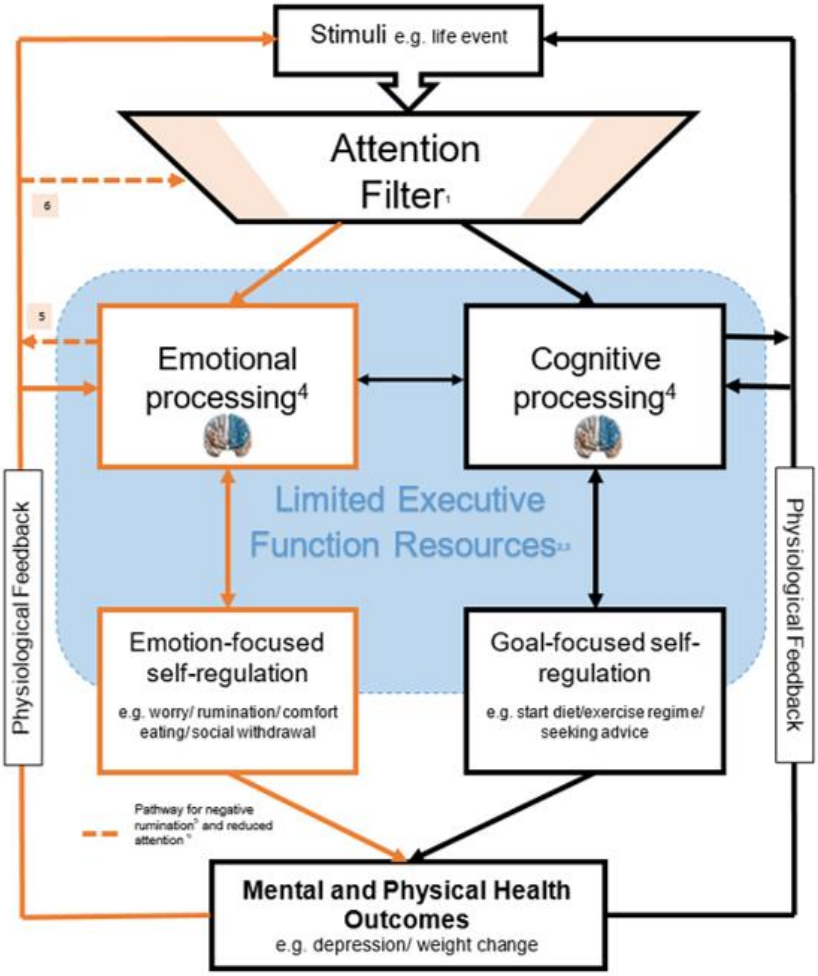
- Obesity to loneliness is the dominant direction of effect although effect sizes are still small (in the region of 1%), however in studies of depression and obesity, depression is the dominant causal variable.
- Obesity and loneliness can feed into a cycle of negative thoughts because they affect self-esteem and perceived social acceptance, meaning individuals may interpret the actions of others more negatively.
- Those prone to negative affect and cognition (e.g., loneliness, depression) may become less healthy over time because:

1)Individuals are focused on negative thoughts and therefore giving less attention to good or neutral things that are happening. This makes it harder for individuals to feel like they are conforming to social norms; it makes the world seem more stressful.

2)Feelings of threat, and increased levels of circulating body fat have physiological effects that increase inflammation, influence brain chemistry and can feed into increased bias toward negative thought processes e.g., reduced dopamine making normally pleasurable experiences (social interaction or food consumption) feel less rewarding.

- Obesity, loneliness, and negative affective disorders have strong components of heritability, and it is likely that there are a sub-group of individuals who share common vulnerability factors in the way they evaluate positive cues (approach e.g., overly reactive to food cues) and negative cues (avoidance e.g., overly reactive to social threat).
- Processing biases can interfere with cognition e.g., more negative threat appraisals, less attention to positive events and bodily signals, such as satiety cues (Llewellyn & Fildes, 2017; Spithoven et al., 2017). Higher cognitive load may leave the individual with less cognitive bandwidth to pay attention to what is happening to them *in the moment* or plan future behaviour.
- All these routes could push vulnerable individuals toward less adaptive coping strategies/behaviour to avoid negative feelings or seek comfort to feel better (avoid/approach).

Explaining the Link Between Negative Affect and Obesity Using the CAMMPI Theoretical Model:



The proposed model (Cognitive-Affective Model of Mental and Physical Health Interaction; CAMMPI see Chapter 3- reproduced above for ease) was synthesised to help explain the findings from Study 1 and the wider association between negative affect and physical health. This included a means to understand i)the longitudinal association between adiposity and loneliness (Study 1), ii) the cross-sectional and longitudinal association between adiposity and depression (Rofey et al., 2009; Vittengl, 2018), and iii) the likely mechanisms of effect. Using the

CAMMPI mode, the link between negative affect and health would be explained as follows:

1)A initial propensity for repetitive negative thinking; propensity (due to genetics or negative life experiences) leads to uprating of emotional processing in the brain (hypervigilance to threat etc.). This would prime the filter mechanism to attend to indicators of threat and mean that positive experiences are less well attended to and less well remembered.

2)Cognitive resources are diverted toward emotional processing and away from task-based activities (executive function, such as self-control/inhibition). This has a subtle negative impact on more complex daily living tasks and situations with high cognitive demands.

3)Experiences are interpreted with a negative thinking bias which reinforces negative interpretations of the behaviour of others (perception of low social support) and efficacy of self-leading to a bias towards greater negative affect (loneliness, depression, anxiety).

4)Experiences such as social interaction feel less rewarding, ‘approach’ behaviour may decrease, and activation of the stress response may increase due to feeling less social support, and/or having less exercise (which is protective against mental health issues).

5) These effects cycle negatively if repetitive negative thinking continues leading to greater allostatic load and persistent low-level inflammation with negative metabolic and neurological effects. Cognitive resources continue to emotional foci leaving fewer resources to deal with tasks and decision-making processes leading to emotion-focused coping behaviours such as avoidance and comfort seeking.

Conclusion

Study 1 found a small predictive reciprocal link between body mass and loneliness over a one-year period that could help to highlight vulnerable groups of adolescents for intervention. In terms of the mechanism of effect we can see that both loneliness and BMI have longitudinal effects on one another. It has also been shown that loneliness and obesity are both linked to negative cognitions related to perceptions of self and acceptance by others as well as a general bias toward negative thinking. The literature outlined in the section 'other psychiatric-somatic comorbidities' shows that over time chronic loneliness can lead to depression, and depression appears to be more predictive of obesity than loneliness (explaining more variance over time). The existence of a chronic trajectory of depression and loneliness in some individuals appears to be maintained by underlying neurocognitive vulnerabilities, such as hypervigilance to threat and repetitive negative thinking. Gaining a better understanding of neurocognitive deficits and the extent that their effects are the same or different could therefore be an important way to disentangle the link between adiposity and negative affect. A better understanding of the mechanism that leads to cognitive impairment linked to adiposity and negative affect could have important ramifications for public health policy in food marketing, social stigma recognition, and the wider understanding of the impact of these conditions on day-to-day tasks. This could also further generally understand of the process whereby mental and physical health interact.

5.0 Study 2 Cognitive Function and Health

Recap from Study 1	Normative Neurological Function	Cognitive Function and Adiposity Research	Cognitive Function and Mental Health Research	Predictions

Study 2 aims to extend the work in Study 1 (Chapter 4) and test key ideas from the proposed theoretical model (Chapter 3) by looking at the association between three areas of interest: adiposity, negative affect, and cognitive function. The model proposes that cognitive function provides an important link between physical and mental health. After an investigation of different ways to measure each construct (adiposity, negative affect, cognitive function; see Chapter 2) this study will also investigate different measures for each construct including both continuous and categorical measures of health. The overall aim is to gauge which areas might be fruitful for future investigations to better understand the link between physical and mental health. The chapter begins by recapping the findings of Study 1 and the proposed theoretical CAMMPI model to explain the importance of cognition in the link between obesity and negative affect. This will be followed by an outline of key research findings between each health area and neurocognition.

Note: More detail about the brain areas mentioned in the following sections can be found in Chapter 2, for example, Figure 5 show a diagram of the areas investigated. In general, we are focused on areas of the prefrontal cortex (PFC) and the medial temporal lobe. These are important areas in the main brain networks that control attention and inhibition (the frontoparietal control network), the network that controls internal thought processes (the default mode network; DMN) and the network that may interface between these areas controlling what we pay attention to (the salience network, including the insula and the cingulate cortex).

Recap Study 1 Adiposity and Mental Health

Study 1 provided evidence of a small reciprocal effect between negative affective conditions such as loneliness and BMI over time, with BMI having a slightly larger predictive effect than loneliness. Studies of depression (a separate but linked negative affective construct) indicate a larger predictive effect on BMI than that of loneliness. Study 2 therefore widens the scope to consider additional measures of adiposity (BMI and waist circumferences) and mental health: depression and anxiety are included with their cognitive symptom of repetitive negative thinking (such as Rumination and Worry). These variables will be examined in relation to one another but also in relation to three areas of cognitive function: executive inhibition and selective attention performance (experiment), executive function in daily living (self-report), and brain function (scan of the neurovascular function of cortical brain areas relevant to inhibition). Study 2 aims to gain a thorough understanding of the relationship between the variables to find out which, if any, relationships show robust effects to inform future study. The nature of the link between these health variables and their neurocognitive effects will also help to evaluate the CAMMPI model (Cognitive-Affective Model of Mental and Physical Health Interaction) in the study discussion. We begin with a summary of key findings from previous literature on the link between mental and physical health and their neurocognitive problems.

Cognitive Function and Mental health

Depression and Cognitive Function Research

Despite clear symptomatic associations with reduced attention and appetite dysregulation, study findings in respect of links to lab-based cognitive deficits, and adiposity are mixed. Although much of the literature in cognition and mental health

focus on clinical populations and older age groups, Castaneda et al., (2007) completed a review of cognitive impairments in young adults (18 to 51) with major depressive disorder (MDD). They found an association between MDD and impaired executive function, attention, short term and working memory (verbal and visual; Fossati et al., 1999), and psychomotor skills (Hill et al 2004), the implication being that depression is causally related to cognitive problems. In contrast, in a population-based sample of 21-35yr olds Castaneda et al.,(2008) found minimal cognitive deficits in those with MDD compared to healthy controls.

Snyder (2013) completed a meta-analysis comparing cognitive function in MDD samples to healthy controls. She concluded that executive function was reliably associated with MDD impairment (effect sizes .32 to .97), however there is some contention as to whether MDD is a cause of cognitive impairment, or a whether cognitive impairment is a vulnerability factor for MDD. A meta-analysis and systematic review examined depression (and anxiety) in relation to inhibition using the classic and emotional Stroop tasks (Epp et al., 2012). They noted previous equivocal findings but found significant differences in performance between clinical populations and controls, albeit with large variation in effect sizes. Their findings challenged the classic conceptions of the negative attention bias, as in the context of emotional words, community depressed participants had poorer performance regardless of whether the words were positive or negative, although those with clinical depression showed some bias to negative stimuli. The authors suggest that depression may cause a general slowing of cognitive processing in the presence of threat which seems to worsen in line with symptom severity. They discuss whether depression affects selective attention (biased) or inhibition and suggest that, per Holmes and Pizzagalli (2008), depression more precisely affects neurocognitive

conflict monitoring. This is shown by significantly reduced activation in the dorsal anterior cingulate cortex (dACC; Brodmann area 24/32) and left dorsolateral prefrontal cortex (Brodmann area 10/46) on incongruent relative to congruent trials (Classic Stroop) in a subgroup of participants with MDD. Participants with the lowest activations in these areas also showed the most Stroop interference (longer reaction times). This makes sense as inhibition tasks like Stroop create conflicting demands (interference) through the participant having to suppress their usual (proponent) responses to select the correct answer (see Chapter 2 on cognitive function).

Neurocognitive findings. A systematic review of MRI studies of MDD (Arnone et al., 2012) indicates reduced brain volume frontal cortex, orbito-frontal cortex, (sensory and affective communication), hippocampus, right cingulate cortex, caudate and putamen. The study findings indicate greater neural activity in the amygdala and enlarged pituitary (processing emotion/ fear and stress responses) but decreased activity in the cingulate, cortex /lateral prefrontal cortex and striatum (reward processing) compared with controls. The study also indicated that there were more white matter lesions in MDD however the mean age was 67 years old, so this is likely to be confounded with age-related cognitive affects (see Chapter 2 & Salthouse 2017).

A more recent meta-analysis of MRI studies of MDD and mild cognitive impairment (Zackova et al., 2021) found reduced volume of more precise regions including the insula and superior temporal gyrus (relevant to communication/ attention/ cognitive performance and language processing), inferior frontal gyrus, amygdala, hippocampus (memory formation), and thalamus.

Depression affects both cognition and negative affect however the main negative emotion centres of the brain (limbic system) are subcortical (see Chapter 2

scanning techniques). Despite this there are fNIRS studies that have investigated MDD using fNIRS. The technique is more widely used in Asia, including Japan where it is used to aid diagnosis and monitoring; haemodynamic activity (blood oxygen or Oxy-Hb) concentrations being negatively correlated with the degree of total depressive symptoms (Ho et al., 2020). A review by Bendall, Eachus & Thompson (2016) give examples of use of fNIRS to examine the cognitive control of emotion which is directly relevant to this study. Bendall et al., note that low activation (lower increases in haemodynamic activity indicating reduced neuronal activity) in the ventrolateral PFC and dorsolateral PFC are related to affective disorders such as depression, and emotional control but it is unclear why the PFC is affected in depression (Pizzagalli & Roberts, 2022).

A further fNIRS study compared prefrontal cortex vascular activity of participants with MDD and healthy controls (Chao et al., 2021). The method used an eyes closed resting state (although this was only for 180 seconds) followed by exposure to four 18-second audio clips portraying different emotions (happy, calm, fear) and white noise. They found significant abnormalities in blood oxygen in the bilateral ventrolateral prefrontal cortex (VLPFC) and bilateral dorsolateral prefrontal cortex (DLPFC) in depressed participants compared to controls. Subsequently, a wide-ranging review of the literature in relation to depression and neurocognitive function was conducted (Pizzagalli & Roberts, 2022). In general, the findings indicate reduced PFC activity and increased activity in the limbic areas (and parts of the ACC) in those with MDD compared to controls (the latter is particularly true in studies using negatively valanced stimuli such as sad faces). As the PFC is activated during executive function tasks and decision-making, the reduced neuronal activity in these areas could indicate that there are fewer neurons in this region (evidenced by

studies showing thinner grey matter), or that the participants are having to divide their cognitive resources between their emotional limbic system (engaged in threat-based processing) and the prefrontal cortex (to attend to tasks or decision making).

Anxiety and Cognitive Function Research. Like depression, although GAD diagnostic criteria clearly report cognitive symptoms, there is little evidence of significant differences in lab-based cognitive test performance compared to controls. Airaksinen et al., (2005) found evidence of anxiety-related impairments in verbal episodic memory and executive function in a population sample of 20–64-year-olds whereas other studies found no association (Castaneda et al., 2007).

Individuals with anxiety disorders or dispositional anxiety have been found to experience deficits of attention control but evidence is mixed (Eysenck et al., 2007). The deficit is thought to be due to due to difficulty in switching away from anxious thoughts or stimuli (i.e. deficits in inhibitory control or switching) however the effect may be overridden in anxious individuals by enhanced effort or other compensatory mechanisms (Eysenck et al., 2007). The deficits of attention control are only observed in individuals with anxious dispositions which underscores the different effects of state and trait anxiety (Robinson et al., 2013).

Anxiety has also been found to be associated with more sensitive cognitive processing for specific feared or threatening stimuli (negative processing bias), and a body of research has examined inhibition when participants are in a fearful state at the time (Robinson et al., 2013). The research reveals the importance of the type of stimuli (its salience to the individual) and the emotional state of the participant during testing. Investigations using the Stroop task indicate that inhibition performance is also affected by age group; older adults having slower performance than young

adults (Kamboureli & Economou, 2021). This reinforces the importance of confounding age-related effects in cognitive testing.

A meta-analysis and systematic review of the effect of anxiety on attention control (Shi et al., 2019) found that anxiety affected inhibition and switching, supporting the ACT Eysenck et al. (2007) explanation of reduced goal orientated attention control and greater stimulus focused attention. Those with higher anxiety symptoms had longer response times in inhibition and switching tasks. The study was not able to establish whether reduced goal-focused attentional control is apparent in tasks which have greater cognitive load demands, and this was noted as an area for future investigation. A further detailed meta-analysis investigating the effects of anxiety on executive function (Majeed et al., 2023) found significant differences based on the type of disorder, task, and the measure (reaction time versus accuracy). Broadly the effect of anxiety disorders was longer reaction times for those with anxiety, lower accuracy on inhibition tasks, but greater accuracy on working memory tasks (updating).

Repetitive Negative Thinking and Cognitive Function. The repetitive negative thinking characteristic of anxiety and depression (worry and rumination) has itself been postulated to arise from a lack of attention or executive function control (ACT; Eysenck et al., 2007). Repetitive negative thinking is associated with more executive function errors (Altamirano et al., 2010; Whitmer & Banich, 2007; Whitmer & Gotlib, 2012). Meta-analyses investigating repetitive negative thinking and executive function have variable findings and explanations for the association, including a greater failure to update working memory (Zetsche et al., 2012), or significant negative associations with inhibition (r between .11 and .23) and set-

shifting (r between .17 and .19) (Vălenaș & Szentágotai-Tătar, 2017; Yang et al., 2017). There is a debate as to whether different types of repetitive negative thinking such as worry (associated with anxiety), and rumination (associated with loneliness and depression) are qualitatively different (see Chapter 2). These repetitive negative thinking constructs could therefore have different effects on cognitive performance and the relationship between mental and physical health.

Although multiple executive function deficits have been noted in relation to physical and mental health, reports of inhibition deficits are quite ubiquitous and appear to be the most consistently reported deficit relevant to both adiposity and mental health. As inhibition deficits can be conflated by attentional deficits, the current study will use one simple and one more complex task paradigm to attempt to disentangle the two constructs. Having a task which measures attention but has consistent features of the main inhibition task to help draw inferences about the role of attention versus inhibitory facets of the construct (Snyder et al., 2015). Due to investigating a complex area with a lot of contradictory findings the study will investigate evidence of cognitive deficits in several different ways - cognitive performance, neurological activity, and self-reported everyday cognitive problems to see if there is evidence of triangulation of findings.

Cognitive Function and Adiposity Research

Studies of obesity that find an association with cognition often highlight deficiencies in inhibition (Stinson et al., 2018). A meta-analysis and systematic review of the effects of overweight and obesity found broad evidence of cognitive impairments in obesity but limited impairment in overweight samples (Yang et al., 2018).

Obesity and Brain Function with fNIRS

Multiple meta-analyses of cognitive and neurocognitive studies (Lavagnino, Arnone, et al., 2016) concluded that lower prefrontal cortex activity affects inhibitory control and BMI, suggesting links through eating behaviour (Vainik et al., 2013). Lavagnino, et al., demonstrated that the right superior PFC was thinner in obese compared to normal weight participants and the effect of BMI on cortical thickness was mediated by inhibition task deficits (Lavagnino, Mwangi, et al., 2016). Stinson et al. (2022) found brain stimulation (tCDS) to the left superior frontal cortex (DLPFC) improved performance on a food-related inhibitory control task. Broadly this ties BMI and food-related inhibition to the superior frontal cortex.

A recent discussion paper (Rebelos et al., 2023) gives examples of fNIRS studies that have been conducted to investigate obesity, diabetes, and weight related outcomes. Obesity studies including the use of inhibitory control measures and fNIRS have been used to assess brain activity in the frontal cortex before and after weight loss or cognitive interventions (to help assess progress). The purported association between obesity, binge eating disorder and inhibition (Rosch et al., 2020) was examined by fNIRS scan (n=40) during a passive viewing task and a Go/No Go inhibition task, each using food image stimuli. During the inhibition task compared to the passive task participants the right inferior frontal gyrus (IFG) and left superior frontal gyrus (DLPFC) displayed greater activity (indicating the role of these regions in inhibition task completion). BMI-related effects were such that the high BMI group (BMI>30) had less activity in the right superior (DLPFC) and left inferior frontal gyrus (VLPFC) compared to the normal weight group.

To draw conclusions about links between the effect of adiposity and negative affect on cognitive function, Study 2 will aim to establish whether these conditions are associated with the same type of executive function deficits. As the extant literature on obesity and depression has often cited inhibition deficits (Stinson et al., 2018; ; Zetsche et al., 2012) this will be the main focus of the cognitive performance investigation using the Simon Task (ST; a measure of inhibitory control performance) and the Continuous Performance Task (CT; a measure of sustained attention that may be conflated with inhibition task performance. See section 2.3.2 and 2.3.3 for more detail about the tasks. Many studies examining health and cognition use older age groups where cognitive effects could be due to age. To fill this gap the sample will be made up of young adults (age 25 to 40) to avoid the confounding effects of younger age (under 25 years) when the prefrontal cortex/inhibitory control structures are still forming and older age (over 45) where cognition may begin to be affected by age-related decline. Investigation of cognitive performance and health will also be complemented with an investigation of neurological function.

fNIRS and Normative Neurological Presentation

fNIRS is flexible and affordable a technique to examine neurological effects. fNIRS can be used at rest and during tasks as it is less affected by movement artefacts than methods such as fMRI (see Chapter 2 for detail about the technique). fNIRS will be used to understand key differences in which areas of the brain are active at rest and during the task. It can also be used to understand the functional connectivity between different areas of the brain to inform discussion about which brain networks are activated. fNIRS is a suitable technique to detect discrimination

between different brain areas by functional connectivity in the frontal cortex during resting state which are stable and reproducible (Geng et al., 2017) and correlate with the fMRI bold signal for wider front to back brain networks (Sasai et al., 2011). To aid in the interpretation of the fNIRS signal the following section details research on normative presentation during the resting state and cognitive tasks. Findings of fMRI and fNIRS studies in participants without health problems (resting state scans and scans during task completion) can help inform about the general pattern of normative neurological function to contrast with cognitive function deficits research.

Normative Function in the Resting State

In resting state analysis, positive frontal mid-right fNIRS signals (Haemodynamic activity; OHb) were found to be associated with the **frontoparietal control network** which includes the bilateral dorsolateral prefrontal cortices, anterior cingulate and inferior parietal lobule (Sasai et al., 2012). Positive frontal superior medial left OHb fNIRS signals were associated with the **DMN** (Default Mode Network) which includes the left and right ventromedial prefrontal cortex (Sasai, 2012). This area is important for reward-based decision-making, regulating negative emotions (i.e., positive, and negative valence) and visual attention to socially relevant stimuli (Hiser & Koenigs, 2018). Evidence includes lesion studies that have revealed decision-making deficits, blunted response to aversive stimuli, and reduced eye gaze. Hypoactivation in this area is also implicated in MDD (Hiser & Koenigs, 2018).

In terms of normative DMN function, Broyd et al. (2009) conducted a systematic review and emphasises that DMN is attenuated or reduced during tasks and not completely extinguished; if a task requires few resources the DMN may

remain active, but more demanding tasks suppress the DMN more strongly. Under the DMN interference hypothesis (Sonuga-Barke & Castellanos, 2007), lapses in attention control are associated with less suppression of DMN activity and this has been observed in the right inferior frontal gyrus, middle frontal gyrus and anterior cingulate cortex. Increased DMN activity has also been associated with task un-related thought attributed to mind wandering. Decreased functional connectivity in the DMN and less anti-correlation between the DMN and task networks are also associated with mental health/cognitive deficits, however even in fMRI a clear picture of the link between health and brain activity is still being established (Broyd et al., 2009; Tozzi et al., 2021).

Normative Function During Attention Tasks

Normative attention function in the brain was investigated using fMRI and fNIRS (Harrivel et al., 2013). Brain activation in areas of the “task positive” network i.e.., **Dorsolateral Prefrontal Cortex** (DLPFC - inferior to F4) and “task negative” i.e., anterior medial frontal gyrus (half-way between FPz and FP2) were examined during an attention task (Multi-Source Interference task; MSIT). Correlation of the strongest haemodynamic signals were compared within and between networks. They found that DLPFC activity increased during task performance and medial frontal activity decreased, surmising that greater DLPFC activity was associated with greater task engagement. Through comparison with fMRI, the study also validated the use of fNIRS for monitoring cognitive states (distinguishing between brain activity during rest and task).

A small fNIRS study utilising non-medicated, older adolescent group (Fishburn et al., 2014) was used to examined whether fNIRS functional connectivity could reliably detect changes in cognitive load and discriminate between task and

resting state in areas of the frontal cortex and parietal lobe. In terms of regional activation, they found that resting state was characterised by greater bilateral functional connectivity in the ventrolateral prefrontal cortex (VLPFC), whereas functional connectivity during cognitive task (N-back) was greater front-to-back (i.e. anterior to posterior between the DLPFC and parietal lobes). The study also indicated that functional connectivity increased with greater cognitive load i.e. larger correlations between activity in areas of the brain was associated with more effortful activity.

Normative function During Inhibition Tasks.

In fMRI studies of the Stroop task, participants show greater activation of the bilateral DLPFC (left BA 10, 9, 6, Right BAs 6 and 9) during interference conditions (Milham et al., 2002). Both hemispheres of the brain maybe recruited when a task is more demanding. An investigation of the nature of inhibition errors by (Garavan et al., 2002) indicated that inhibition performance was dominantly led by the right DLPFC in connection with the parietal areas. They noted that for more difficult inhibition tasks participants relied on the ACC (anterior cingulate cortex) as a fast track to process that relies more on what has happened in previous trials than thinking through the correct response. Participants who self-reported greater cognitive problems were also more likely to recruit this fast track or more 'urgent' alternative pathway which could explain why their responses were less accurate.

In an fNIRS study of Stroop inhibition, young adult participants showed a greater vascular response in the lateral PFC (International 10:20 brain locations F7/8 and F3/4; see Figure 32 & Appendix M) during incongruent compared to neutral trials (Schroeter et al., 2003). As a stronger vascular response is related to greater

neuronal activity this indicates the lateral PFC is important in inhibition task completion. A meta-analysis (Turner & Spreng, 2012) found that young adult participants recruited different sections of the frontal lobe for different elements of the task: the DLPFC during working memory tasks and in inhibition tasks they recruited the right anterior insular, inferior frontal gyrus (BA 9; VLPFC) and the left medial superior frontal gyrus (BA 6). This indicates that the DLPFC i.e. the upper/ superior frontal lobe is likely to be recruited more in tasks that have higher working memory demands.

Study 2 Aim and Predictions

To address the limitations in the current literature and lend support to a cross disciplinary discussion of the interaction of mental and physical health, this study aims to investigate different inhibitory function and attention in young adults, aged 25 to 40 years, when their cognitive ability should be optimal. We will examine whether prominent health measures of adiposity, negative affect and repetitive negative thinking relate to one another, and have comparable relationships to several outcome measures: cognitive task performance, and haemodynamic brain activity and self-reported executive function problems. As fNIRS procedures are still in development investigations of the haemodynamic signal and health are exploratory but it is hoped they will contribute to the on-going scientific exploration of the field.

Based on the extant literature and proposed CAMMPI model (see Figure 14), the following predictions are made:

Adiposity and Negative Affect

a) Larger body measurements are related to greater negative affect and greater repetitive negative cognition.

Key measures: Anthropometry, Negative Affect

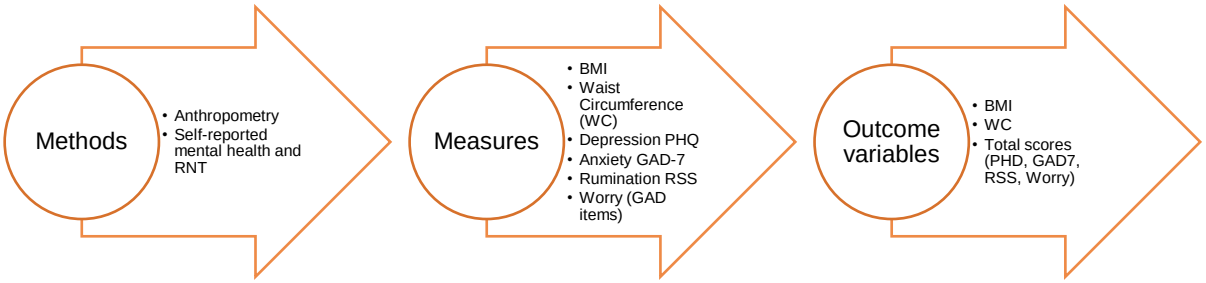


Figure 21
Prediction1 Adiposity and Mental Health
Note: RNT=Repetitive Negative Thinking

Computer-based Performance Tasks of Attention and Inhibition,

2a) Poorer performance will be seen on the more cognitively demanding task (Inhibition) than attention.

2b) Significantly worse cognitive performance for those with greater negative affect, and larger body size.

Key measures: task errors, reaction times, Simon Effect, anthropometry, negative affect and repetitive negative thinking.

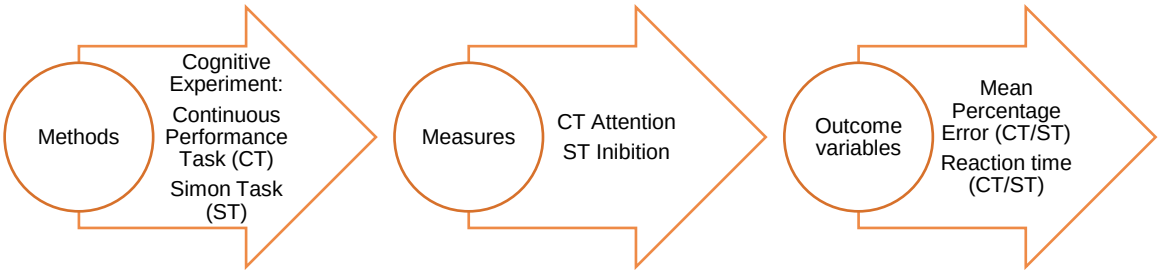


Figure 22
Prediction 2 Attention and Inhibition Performance

Self-reported Executive Function Problems

3a) BRIEF scores will be more strongly associated with negative affect than body size.

3b) Higher BRIEF scores (more self-reported EF problems) will be related to worse performance in computer-based tasks (longer reaction times and more errors of attention and inhibition).

Key measures: BRIEF T-scores; Computer-based cognitive tasks (task errors, reaction times, Simon Effect), Anthropometry, Negative Affect

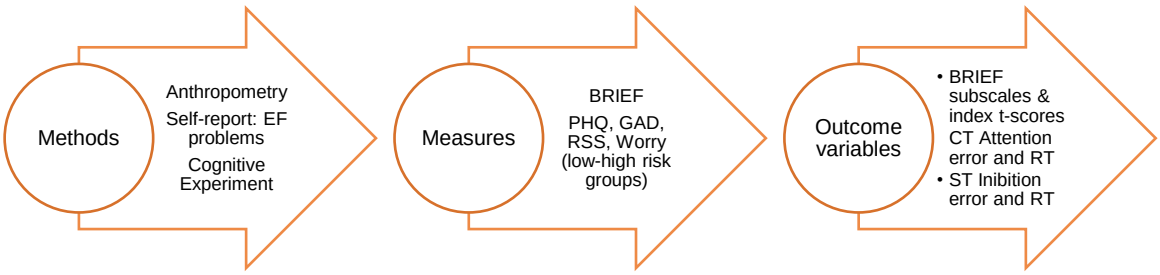


Figure 23
Prediction 3 Executive Function Problems in Daily Living

Resting State Haemodynamic Activity

4a) Temporal lobe areas will show a greater increase in haemodynamic activity than the frontal cortex during the resting state.

4b) Larger body-sizes will be related to significant differences in the pattern of haemodynamic activity during the resting state, in the frontal and temporal lobe, (reflecting significantly different patterns of frontoparietal task control system and Default Mode Network activity compared to participants with normal weight).

4c) Greater negative affect will be related to significant differences in the pattern of haemodynamic activity during the resting state, in the frontal and temporal

lobe (reflecting different patterns frontoparietal task control system and Default Mode Network compared to participants with low levels of negative affect).

Key measures: Haemodynamic activity changes in the resting state; BMI/ WC; Depression, Anxiety, Rumination, Worry

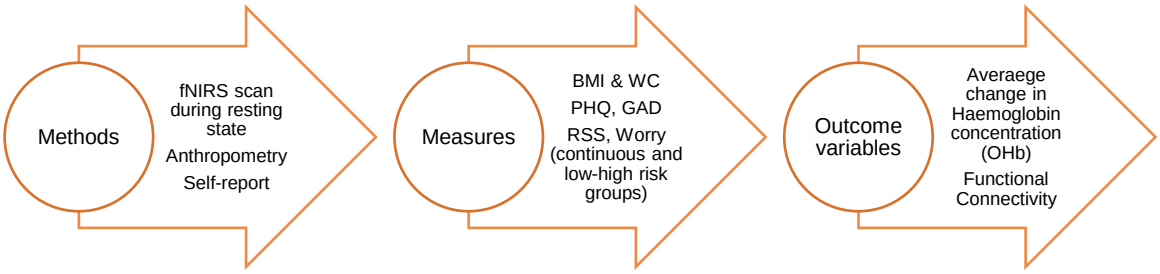


Figure 24
Prediction 4 Resting State Neurovascular Activity

Haemodynamic Activity during Performance tasks

5a) Significant differences in haemodynamic activity in the prefrontal cortex based on obesity (low/high).

5b) Significant difference in haemodynamic activity in the prefrontal cortex based on negative affect (low/high).

Key measures: Haemodynamic activity (change from baseline during computer-based tasks of attention and inhibition); BMI/ WC; Depression, Anxiety, Rumination, Worry.

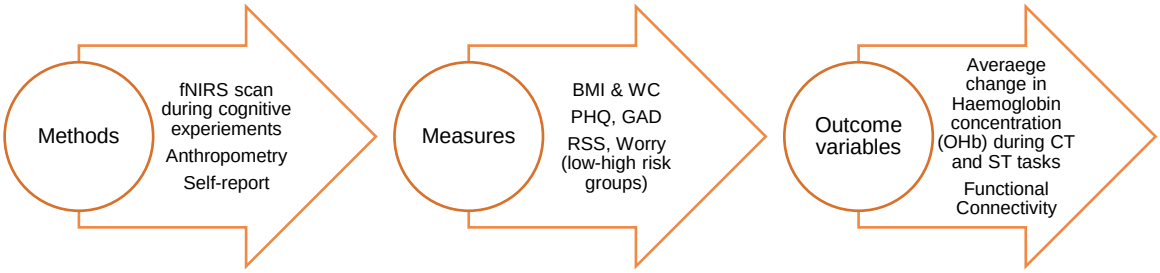


Figure 25
Prediction 5 Task-based Haemodynamic Activity

6.0 Study 2 Methodology

Participants	Measures	Procedure	

Participants

Recruitment

Participants were recruited through online advertisements and posters around university buildings as well as local community spaces (see Appendix G Study 2 Methodology). The sample were therefore a self-selected sample of young adult students and members of the public. The study was approved by the University of Central Lancashire Ethics Committee in July 2018, and written consent was obtained before testing (see Appendix A Ethics & Consent). An a priori power analysis conducted using GPower (Faul et al., 2009) suggested that at least 84 participants would be required to detect small-medium (i.e. effects around .3, based on (Cohen, 1992) correlational effects at 80% power, and 68 participants would be required to detect small (or larger) effects in a multiple regression with up to 5 predictors (see Appendix H).

Inclusion Criteria

Participants contacted the researcher and were provided with a comprehensive information sheet about the study to help them decide if they wanted to take part. The initial requirements were for participants to be young adults (25 to 41 years of age) to prevent confounding effects of brain development and age-related effects, normal or corrected to normal vision and English reading skills sufficient to view understand the surveys and tasks, and not having taken strong

sedative/ stimulant medication or substances in the last 12 hours as these may impact normal cognitive task performance.

Sample Characteristics

90 Participants were recruited aged 25-41 years old. This size is comparable with similar neurocognitive health studies e.g., Rosenbaum (2016), N=84). A range of sample characteristics (demographic variables; see Table 9) were recorded to help describe the normality of the sample and enable comparison with other research studies and interpretation of the findings, including outliers.

Table 9

Demographic Variables and Descriptives (N=90)

Variable	Subgroups	N, %	Mean (SD)	Mode
Age	25 to 41	n=90	M=31 (5.10)	25 (14%)
Gender	Females	57 (63%)	-	Female (63%)
	Males	33 (37%)		
Ethnicity*	WE	75 (83%)	-	WE (83%)
	SA	5 (6%)		
	ME	5 (6%)		
	BA	2 (2%)		
	AC	2 (2%)		
	Other	1 (1%)		
Handedness	Right	75 (83%)	-	Right (83%)
	Left	12 (13%),		
	Ambidextrous	3 (3%)		
Level of Education*	HS	4%	-	PG (36%)
	C	30		
	D	24		
	PG	32		

Measure	Range	Mean (SD)	Mode
Subjective Social Status	3-8	M=5.73 (1.40)	7 (26%)
No. Caffeine drinks (past 12 hrs)	0-4	M=1.18 (1.04)	1 (40%)
Fluid Intelligence (Ravens item 4-15)	1-12	M=6.47 (2.60)	7 (16%)

***Notes*. *Ethnicity**: WE=White European; SA= South Asian; ME= Middle Eastern; BA=Black African; AC= Afro-Caribbean. ; ***Education**: HS= High School; C=College, D=Degree; PG=Postgraduate.

Sex at birth was 63% female. Most of the sample had white European ethnicity (75%). Subjective Social Status (as a proxy for SES) per the MacArthur Scale indicated a normal income distribution. Participants completed the Advanced Progressive Matrices-Short form (APM-SF; Arthur and Day, 1994) to give a baseline measure of general/ fluid intelligence. This measure indicated a normal distribution of FI although those who had attained degree or higher levels of education were over-represented (over 50% of the sample) compared to typical community levels (qualifications of degree and above make up approx. 35% of school qualifications based on 2021 ONS figures (ONS, 2021). Handedness per the Edinburgh Handedness Inventory (Oldfield, 1971) indicated lateralisation (83% right-handed). Participants reported they had consumed an average of one caffeine drink over the 12 hours prior to taking part.

Measures

Table 10 provides an overview of the measures and variables used, scoring and coding. Also see Appendix K Study 2 Treatment of Data for further detail on the calculation of variables and Appendix N Study 2 Scale Reliability (Chronbach's Alpha) for the Cronbach's Alpha of each scale.

Table 10

Summary of Study 2 Measures, Scoring and Coding

Measure	Continuous Variables	Categorical variables
PHYSICAL HEALTH		
BMI. Weight (kilograms)/ height (metres2)	Raw BMI	Coded per International Obesity Task force (IOTF) cut-offs and as a binary risk variable (0=Normal risk/increased risk; 1= Moderate/severe risk).
Waist Circumference (WC). Waist Circumference Risk(WCr)	Raw measure (cm)	The categorical variable is referred to in the thesis as WCr to help distinguish it from the raw score. Coded as per IOTF cut-offs (see Table 4 for male and female cut-offs). 1 =Average risk; 2= Increased risk.
NEGATIVE AFFECT		
Anxiety Generalized Anxiety Disorder scale, GAD-7 (R. Spitzer et al., 2006). Screening and severity measure.	7 items answered on a 0-3 scale based on how often the participant has been bothered by a particular problem (0=Not at all; 1=Several Days; 2= More than half the days; 3= Nearly every day). Total severity scores range from 0-21 with cut offs: 0-5 =mild, 6-10 =moderate, 11-15= moderately severe, 15-21= severe anxiety.	Max score = 21. A score of 10 or greater is considered clinically significant and this cut-off was used to create a clinical variable (0=Nonclinical 1= Clinical anxiety symptoms).
Depression Patient Health Questionnaire, PHQ-8 (Spitzer et al., 1999). Screening and severity measure.	The 8 items were answered on a 0-3 scale based on how often the participant has been bothered by a particular problem (0=Not at all; 1=Several Days; 2= More than half the days; 3= Nearly every day).	Max score = 24. A score of 10 or greater is considered clinically significant and this cut-off was used to create a clinical variable (0=Nonclinical 1= Clinical symptoms).

	The total severity score (0-24) with the following cut offs: 0-5 = mild, 6-10 = moderate, 11-15 = moderately severe, 16-24 = severe depression symptoms.	
REPETITIVE NEGATIVE THINKING		
Rumination 10–item Ruminative Response Scale (RRS-10; Treynor, Gonzales, Nolen-Hoeksema, 2003).	Participants rated how often they think a certain way when they feel 'sad, blue or depressed' (1=Almost never; 2=Sometimes; 3=often; 4=Always). Total rumination score 10 to 40. Subtotals (scoring 5-20) for brooding (items 1,3,6,7,8) and reflection (items 2,4,5,9,10) of between 5 and 20.	Categorical variable used the original coding: (1=Almost never; 2=Sometimes; 3=often; 4=Always). Binary categorical variable: ***1 Low***= combined scores for 1 Almost never and 2 Sometimes; ***2 Moderate/High***= combined scores for 3 often and 4 always.
Worry Ultra-short worry screening using item 2 and 3 of the GAD-7.	Item 2 and 3 of the GAD-7 answered on a 0-3 scale based on how often the participant has been bothered by a particular problem (0=Not at all; 1=Several Days; 2= More than half the days; 3= Nearly every day), Total scores from 0-6	Categorical variable used the original coding from the GAD: (0=Not at all; 1=Several Days; 2= More than half the days; 3= Nearly every day). Binary categorical variable ***1 Low***= combined scores for 0 Not at all and 1 several days; ***2=Moderate/high*** = 2 combined scores for more than half the days and 3 nearly every day
COGNITIVE FUNCTION		
Sustained attention performance. Continuous Performance Task (CT; based on Shalev, Ben-Simon, Mevorach, Cohen & Tsal, 2011) was used to measure sustained attention.	Metrics: average response time (to correct trials), percentage error (commission and omission) Higher scores indicate less efficient attention control.	

Response inhibition performance. Simon Task (Simon, 1990) based on features of tasks used by Scerrati, Lugli, Nicoletti, & Umiltà, 2017). Motor inhibition task (visuospatial signal interference resolution).	Performance is measured by congruency effect (incongruent minus congruent response time (accurate trials); Percentage error (incongruent and congruent). Higher scores indicate less efficient spatial interference control.	
NEURAL ACTIVITY		
Cerebral blood oxygenation. fNIRS Haemodynamic signal during rest and task	Measuring changes in concentrations of oxygenated (OHb) and deoxygenated haemoglobin (HHb) over time in areas of the prefrontal cortex and medial temporal lobe.	
Executive Function IN DAILY LIVING		
Executive Function Problems in Daily Life Behaviour Ratings Inventory of Executive Function for Adults (BRIEF-A; Roth et al. 2005) used to clinically evaluate self-regulatory function. Questions are grouped into nine sub-scales relating to different areas of executive function	75-item self-report scale. Participants were asked to indicate how often they feel that item has caused them problems over the past month (Never=1, Sometimes=2, often=3). Subscale totals are calculated (Appendix K Study 2 Treatment of Data) then looked up in age-dependent standardised tables (Roth et al., 2005) to obtain the relevant T-Score.	A score of 50 represents the standardised population mean (age-dependent T-Score), and 65 (1.5 SD above the mean) is interpreted as 'abnormally elevated' or potentially clinically significant (Roth et al., 2005 pg13). The clinical cut-off was used to create a clinical variable for those below or at/above 65 (0=non-clinical 1= Clinical).

Physical Health (anthropometry)

Body Mass Index (BMI). Body Mass Index is a proxy measure of adiposity (BMI; weight kg/height m^2 or $cm/100^2$). It is often used as a fast screening-tool to identify individuals at risk of cardiometabolic problems more effectively than by observation or by weight alone. BMI can be used as a continuous measure, but often it is summarised as weight categories (underweight, normal weight, overweight, obese, morbidly obese) based on 'cut-offs' that are said to reflect grades of risk of ill-health (see Table 4).

Negative Affect

Depression. The Patient Health Questionnaire (PHQ-8, a variant of the PHQ-9) is a gold standard instrument used internationally to screen for depression. The manual states the scale is used to diagnose and monitor depression as it allows symptom severity to be measured and compared in a brief and reliable scale (Spitzer et al., 2006; Spitzer et al., 1999). The 8 items are rated between 0-3 based on how often the participant has been bothered by the problems listed (not at all – nearly every day) giving a maximum score of 24 (see Table 10 and Appendix G Study 2 Methodology for the questionnaire). Higher scores indicate worse depression and total scores above 10 are deemed potentially clinically significant.

Anxiety. The GAD-7 is a screening and severity measure for Generalized Anxiety Disorder symptoms as well as panic social anxiety and post-traumatic stress disorder (Spitzer et al., 2006) The 7 items are rated between 0-3 based on how often the participant has been bothered by the problems listed (not at all – nearly every day) giving a maximum score of 21 (see Table 10 and Appendix G3 Study 2

Methodology, for the questionnaire). Higher scores indicate worse depression and total scores above 10 are deemed potentially clinically significant.

Repetitive Negative Thinking

Rumination. Rumination was measured using the 10–item Ruminative Response Scale (RRS-10; Treynor et al., 2003). Participants rated how often they think a certain way when they feel 'sad, blue or depressed' (1 almost never to 4 Almost Always) giving a maximum score of 40. Higher scores indicate greater frequency of rumination. Half of the items are relevant to brooding rumination and half reflective rumination (maximum score of 20 for each subscale).

Worry. An ultra-short worry screening measure was taken from item 2 and 3 of the GAD-7, scores ranged from 0-6. Higher scores indicate more worry. The GAD-7 itself has an ultra-short two item scale that can be used as a screener for GAD (Spitzer et al., 1999; Kroenke et al. 2007) and very short and even single item scales have been shown to have reasonably validity and reliability while minimising demands on participant time.

Cognitive Function: Performance Tasks

Experiment-based testing batteries are often used to assess executive function. This is favoured as a reliable and highly standardised way to measure cognitive ability, however there are debates over which constructs are being measured and how the findings relate to lived experience of cognitive difficulties or impairment, especially in milder cases (Vainik et al., 2013). See Chapter 2 on executive function for more detail about response inhibition.

Response Inhibition. The Simon Task (Simon, 1990) is a computer-based spatial inhibition task. Participants must press a button when they see a target shape, but they respond using a different button depending on the colour of the shape. The current task is based on a version used by Scerrati, Lugli, Nicoletti, & Umiltà, 2017), see Procedure for details. This is a signal interference resolution task, similar to the Stroop task (Zhang et al., 2017) but will allow exploration of visuospatial attention/inhibition which is less well studied. Further, the task does not depend on word and audio processing which could be a confounding feature of the task especially for any participants with word processing difficulties such as dyslexia (Mullane et al., 2009). Participants will naturally try to respond using the hand/button on the same side of their body as the shape appears (left or right of the screen). The task therefore measures how effectively participants can inhibit their innate tendency (pre-potent disposition) to press a button based on where the shape appears on screen (left or right side). Performance is measured by the number of errors on congruent and incongruent trials, and effect of congruency/incongruency on reaction time (congruent minus incongruent trials).

Efficacy of the measure. The Simon Task offers moderate test-retest reliability for congruent minus incongruent reaction time (.43), but higher reliability for global RT =.74 (Paap & Sawi, 2016). Increased Intra-individual differences in Simon task RT Standard Deviation have also been observed with participants with conditions that affect RT related to inhibitory functions (Schiff et al., 2014). In a systematic review, Mullane et al., (2009) found that children with Attention Deficit Hyperactivity Disorder (ADHD; known to be associated with reduced attention and inhibition) were slower to respond and made more errors on the Simon Task that typically developing controls.

Inhibition – Simon Task (Hommel, 2011). This spatial motor inhibition task was implemented based on procedure used by Scerrati et al. (2017). Stimuli consisted of blue or red squares that appeared positioned either to the left or to the right of centre of a computer screen on a black background. This signal interference task requires participants to inhibit their prepotent tendency to respond with the same side of the body as the visual field the stimulus appears in (left or right).

Participants were asked to place a finger from each hand on either side of a Chronos button box with two operational buttons (left hand = button 1, right hand = button 5; See Figure 26).

On each trial a single square was presented, and participants had to press the button that corresponded to its colour (e.g. Button 1= red, Button 2= blue). Half of the trials had a congruent presentation (the coloured shape appeared on the same side of the screen as the button assigned to it) and half had and incongruent presentation (the coloured shape appeared on the opposite side of the screen as the button assigned to it). Participants were instructed to ignore the location of the stimuli on the screen and respond as quickly and accurately as possible. The response pattern was counterbalanced between participants (see Figure 26 for response pattern a and b).

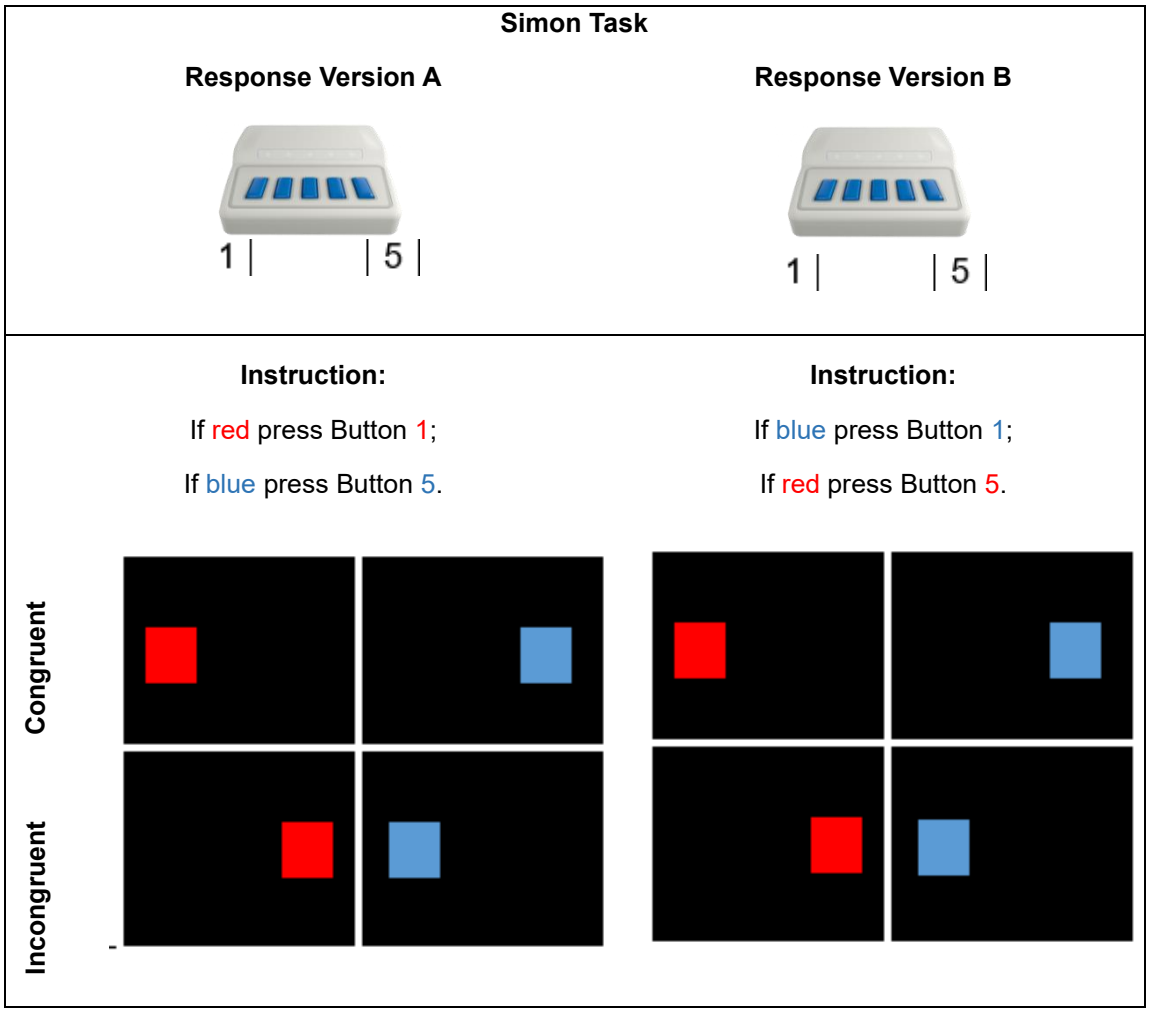


Figure 26 Simon Task Counterbalancing of Stimuli and Response Button Location

Task trials were presented in 4 blocks of 80, plus 16 practice trials (320 in total, see Table 11). Block 1 & 2 consisted of 50% congruent and 50% incongruent presentations.

Table 11

Simon's Task: Number of Trials and Blocks

			All Task Trials	Per Block		
Task Blocks	**Total Trials**	**Practice Trials**	**Total**	**Total**	**Congruent 50%**	**Incongruent 50%**
4	336	16	320	80	40	40

Stimuli were presented for 300ms followed by an ISI of 1500ms. There were no foil trials in this task (every trial required a response), but participants could make errors of omission (failing to press within the required time frame between 100ms and 1500ms of stimuli onset) or commission (pressing the wrong button).

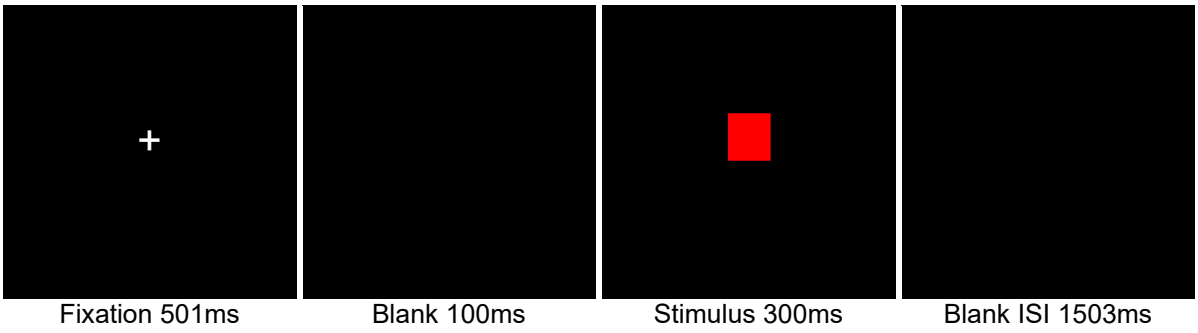


Figure 27
Simon Task Timings Within Each Trial (milliseconds)

Sustained Attention. A Continuous Performance Task (CT; Shalev et al. 2011) was used to measure sustained attention. This is a computer-based task where participants are asked to press a button when they see a target shape appear on screen. The task stimuli and operation were created to be similar to the inhibition task, but it has lower cognitive demands therefore serving as an 'easy' condition. The neurocognitive action of the task is to cue the 'motor preparation phase' of the participant's response so which also occurs within the inhibition task. This means the neurological effects of the of attention (vigilance) and motor preparation phase can be differentiated more clearly from the inhibition response in the Simon Task (ST).

Efficacy of the measure. Shalev et al. (2011) concluded that the CT task had good reliability (.94 split half, .66 test-retest). There are alternative sustained attention tasks (Conners CT and the Sustained Attention to Response Task) however these tasks use a reversed format where participants respond to all stimuli and withhold a response to rare targets. This has the advantage of putting the

habituation effects on the non-target stimuli, but it turns the task in to a withholding inhibition task.

Attention - Continuous Performance Task. (CT; based on Shalev et al., 2011). Task stimuli consisted of coloured shapes that appeared in the centre of a computer screen on a black background. 324 Task trials were presented in 4 blocks of 81. Each block consisted of 30% target and 70% non-target foils (see Table 12).

Table 12
CT Task: Number of Trials and Blocks

			All Task Trials			Per Block		
Task Blocks	**Total Trials**	**Practice Trials**	**Trials**	**Targets** (30%)	**Foils** (70%)	**Trials**	**Targets**	**Foils**
4	343	19	324	96	228	81	24	57

Participants were asked to respond as quickly and accurately as possible whenever they saw the target shape (red square; see Figure 28) by pressing a designated key on a Chronos button box with a finger of their dominant hand. Participants were to ignore non-target shapes (foils). Foils consisted of squares, circles, triangles, or stars in red, blue, purple, or white.

Figure 28
Continuous Performance Task (CT): Example Stimuli and Timings per Trial

Each stimulus was presented for 100 ms. *The interstimulus interval (ISI-time) was set to 1,500 ms.* The maximum reaction time (RT) was limited by the ISI to 1500

ms, and a minimum RT of 100 ms was imposed during data cleaning (to ensure the participant had time to perceptually process the stimulus before they responded). Failures to press in response to a target trial or outside of the time frame were categorised as ***errors of omission***. Responses to a non-target shape (foil trial) were categorised as ***errors of commission***.

Executive Function in Daily Living

Behaviour Ratings Inventory of Executive Function for Adults (BRIEF-A; Roth et al. 2005) is used to clinically evaluate self-regulatory function. Participants are asked to indicate how often they feel that each item has caused them 'problems' over the past month and responses are broken down categorically to give information about nine areas of executive function corresponding to, 2 broad areas of executive function skill: Behaviour Regulation and Metacognition, and a Global Composite score (See Appendix J BRIEF Subscales Items & Definition for details of the sub scales). The Behaviour regulation index includes factors that are more relevant to self-control such as inhibitory control, capacity to switch between tasks and emotional control, whereas the Metacognition index is relevant to less emotional executive function capacities such as working memory, planning and organisation. Performance on the respective areas can be used in clinical settings to gain an overall comparative understanding of an individual in relation to 'normal' function, as well as a more detailed profile of their specific areas of deficit. In this study the BRIEF is relevant to the theoretical model as it can help us understand which executive function capacities are more affected by physical and mental health issues.

Efficacy of the measure. The Behaviour Ratings Inventory of Executive Function (BRIEF) scale was designed with reference to lived experience, clinical reported executive function problems which were organised based on neurological findings and factor analysis. The scale has been well-validated with a range of clinical and community samples (Roth et al., 2005). for self-report normative sample Cronbach's alpha ranged from .73 to .90 for the clinical scales test-retest reliability correlations ranged from .82 to .93, and the scale has good convergent validity with other neurocognitive function measures such as the Frontal Behavioural Scale Executive Dysfunction scale (FrSBe; Grace & Malloy, 2002; *r*=.35 to.74) and the Dysexecutive Questionnaire total score (DEX; Wilson et al, 1996; *r*=.38 to .80). The Clinical Assessment of Depression scale (CAD; Bracken & Howell, 2004) showed moderate to high correlations between Depressed Mood and BRIEF Inhibition (*r*=.44) and Emotional Control scale with Depressed Mood (*r*=.51). The CAD Anxiety/ Worry scale correlated significantly with Inhibition (*r*=.58), Shift (*r*=.47), Emotional Control (*r*=.56), Initiate (*r*=.42) and Working Memory (*r*=..48).

Neurological (fNIRS)

Functional Near Infra-red Spectroscopy was used to measure changes in concentrations of oxygenated (OHb) and deoxygenated haemoglobin (HHb) over time. The haemodynamic signal is used as a proxy measure to examine neurone activity. fNIRs measures are similar to Blood Oxygen Level Dependent (BOLD) response used in fMRI and have been used widely to measure neural activity in inhibition and attention tasks (Tanaka et al., 2013; Cui et al.,2011; Derosière et al., 2015). The effect occurs as brain cells (neurones) demand more oxygen when they are engaged in a task. Monitoring concentration changes of haemodynamic activity

can therefore be used to indicate which areas of the brain are more active. Participants' haemodynamic signal (change in blood oxygen over time) was measured in 8 specific areas of the brain (16 optode channels, see Table 13) over the frontal cortex (important for executive function) and the medial temporal lobe (part of the DMN which is implicated in internal processing e.g. related to the self). See Appendix M Optode Positioning (fNIRS) for further details of the rationale for the chosen measurement locations.

Procedure

The study was conducted in the University of Central Lancashire Brain Imaging Lab (see Figure 29 for an overview of the procedure, see Appendix G Study 2 Methodology for the study information and questionnaire pack). Each participant testing session took between 90 minutes and 2 hours. Initially the participant was settled and given a written and verbal overview of the study and completed written consent to proceed (see Appendix A2).

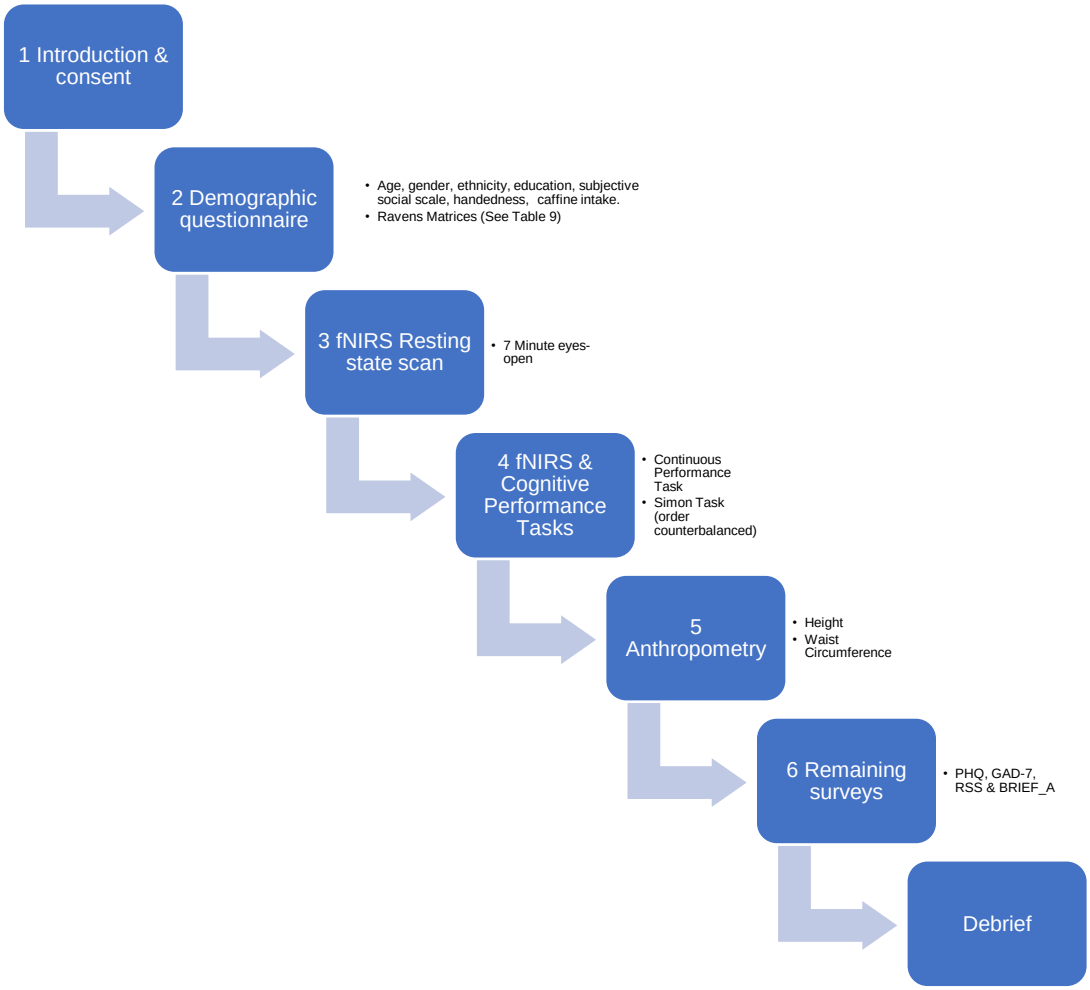


Figure 29
Overview of Study 2 Procedure
Notes: RSS= Ruminative Response Scale (RRS-10); GAD= General Anxiety Disorders (GAD-7); PHQ=Patient Health Questionnaire (PHQ-8); BRIEF= Behaviour Ratings Inventory of Executive Function

Participants completed the paper-based demographics and the Raven's Advanced Progressive Matrices-Short form as a proxy for general intelligence to check if we had a normal distribution in the sample. They were then fitted with an fNIRS cap and completed a 7-minute resting state scan, followed by task-based scans during computer-based cognitive testing. The order of the cognitive tasks was counterbalanced. This was followed by the anthropometry (see Appendix G4 Anthropometric Procedures). They then completed the remaining paper-based scales: the PHQ, GAD-7, RSS-10 and the BRIEF_A (see Appendix G Study 2

Methodology for the questionnaire pack). After the measurements, participants were debriefed (see Appendix B) and given their choice of either SONA points (available to students only towards use of the psychology participant pool), or a £5 gift card for their time (available to students and community participants).

Anthropometric Procedure

Measures were taken in socks and light sports clothing. The height and weight measures (to the nearest 0.1cm/0.1kg) were taken twice, if measurements varied by more than 0.2 cm for height or 0.2 kg for weight, a third measurement was taken. NB. The final measures were averaged, in line with WHO (2017) guidance (see Appendix G4).

Cognitive testing

E-Prime 3.0 software was used to present the cognitive (behavioural) tasks and record participant response time and accuracy in button presses. fNIRS data was recorded simultaneously and automatic triggers recorded the onset of each task, each rest period and each stimulus. The cognitive tasks were designed to a similar format. The stimuli were coloured geometric shapes presented on a black background and participants had to respond to target shapes using a button press.

The presentation order of the CT and ST were counterbalanced. Task trials were presented in blocks (see Figure 30 Task Timings) and each block was followed by a 20 second fixation (‘Rest’) where participants were instructed to remain still and look at the screen. This was to allow the fNIRS signal to return to baseline (Tanaka et al.,2013; Cui et al., 2011). Each ‘rest’ period was followed by the option to take a comfort break (to allow participants to move and rest their eyes and allow the

researcher to make any adjustments). Participants were instructed to press a button when they were ready to continue.

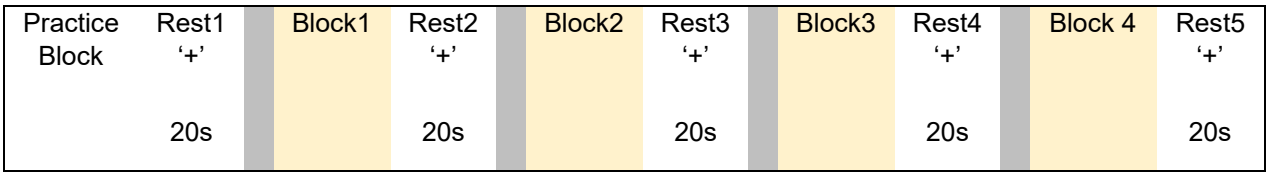


Figure 30
Task Block Structure and Timing for CT & Simon Task
Notes: ■ = Optional comfort break

Neurological Procedures

fNIRS Instrumentation and Software. The scan was performed using the Artinis Oxymon (MKIII) and Oxysoft 3.0. The system had 16 optodes which measure chromophore concentration changes in oxygenated and deoxygenated haemoglobin (collected at 760 and 850nm wavelengths, sampling rate 10hz) over targeted areas of the cortex. Measurement was corrected for the age dependency of the differential pathlength factor (DPF) per Duncan et al. (1996). The modified Beer Lambert Law (Cope et al., 1988) was applied using Oxysoft software. This takes account of the relative intensity of light emitted and returned whilst factoring in scattering and absorption (based on wavelength of the light and the distance between the transmitter and receiver).

Optode placement. The positions of the haemodynamic measurements were decided after interrogation of the fNIRS Optode Location Decider (fOLD) and the literature on inhibition (highlighting the importance of the dorsolateral prefrontal cortex) and consultation with Artinis (the fNIRS equipment manufacturer). To examine the DMN (medial temporal lobe) and the frontal cortex with a 16 optode

configuration, the optodes were arranged in cross-section across the frontal lobe (superior, medial; inferior), and medial temporal lobe (just above the ear).

Eight optodes were placed on each hemisphere (each pair of transmitter and receiver optodes created a measurement channels). The optodes pairs were positioned at 3cm using pre-cut holes in the cap which corresponded to the international 10:20 (i10:20) system (see Figure 31 of the fNIRS cap setup).

Figure 31
Image of fNIRS Cap Optode Arrangement (Right Hemisphere)
Notes: Participants were seated in front of a 17inch monitor. The distance between the eyes and the monitor was measured adjusted to be 70 cm.

Participant preparation. Participants were asked to remove any headwear or hair styles that would interfere with the cap placement or later height measure. Sensitivity and privacy were afforded towards anyone wearing head scarves or veils. A neoprene cap with pre-punched holes and marked with 10:20 reference points was used. To select the appropriate cap size, participant head circumference was measured with a non-elastic tape measure. Cap sizes were as follows: Small = 53-55cm, Medium= 55-58cm, L =>59cm. The cap was positioned per the International

10:20 (i10:20) System, by centring it on the head using measures front to back (nasion to inion) and left to right (between the two pre-auricular points). The Cz marking on the cap being lined up with the intersection of these two measurements.

Areas of the brain investigated. See Appendix M Optode Positioning (fNIRS) for rational of fNIRS optodes positioning to examine nodes of the Default Mode Network (DMN; medial temporal lobes) and prefrontal frontal cortex. Figure 32 highlights the spatial location of the optodes on the International 10:20 system to illustrate the position of the optodes and the brain areas examined on a side view. Table 13 gives more detail about the location of these optodes in relation to regions of interest in common brain atlases (I10:20; Talairach and anatomical atlas) per Okamoto et al., 2004. Figure 33 gives a top-down view of the position of the optodes in-relation to the International 10:20 locations, and Table 14 lists the receiver-transmitter pairs for each measurement channel in the study, and the associated Brodmann's areas.

Table 13

Name Labels and Locations of the Regions of Interest from different Brain Atlases including Percentage Coverage adapted from Okamoto et al. (2004)

i10:20	Anatomy Talairach Daemon	% Coverage	BA	% Coverage	Anatomy Manual Labelling	% Coverage	BA	% Coverage
	Frontal Lobe (FL): Superior/Inferior Associated with Attention and Inhibition							
F3	L FL Superior frontal G	(56)	10	(47)	L FL Middle frontal G	(81)	**9**	(63)
	L FL Middle frontal G	(44)	**9**	(43)	L FL Superior frontal G	(19)	10	(31)
F4	R FL Middle frontal G	(60)	10	(49)	R FL Middle frontal G	(98)	**9**	(52)
	R FL Superior frontal G	(40)	**9**	(34)	R FL Superior frontal G	(2)	**46**	(25)
F7	L FL Inferior frontal G	(84)	47	(81)	L FL Inferior frontal G	(88)	47	(63)
	L FL Middle frontal G	(16)	**45**	(13)	L FL Middle frontal G	(13)	**45**	(19)
F8	R FL Inferior frontal G	(94)	47	(94)	R FL Inferior frontal G	(100)	47	(60)
	R FL Middle frontal G	(6)	**45**	(6)	R FL Middle frontal G		**45**	(29)
	Medial Frontal & Temporal Lobe: Associated with the Default Mode Network							
T3	L TL Middle temporal G	(94)	**21**	(94)	L TL Middle temporal G	(88)	**21**	(88)
	L TL Superior temporal G	(6)	22	(6)	L TL Superior temporal G	(12)	22	(12)
T4	R TL Middle temporal G	(96)	**21**	(95)	R TL Middle temporal G	(85)	**21**	(85)
	R TL Superior temporal G	(4)	22	(5)	R TL Superior temporal G	(15)	22	(15)

Notes: BA= Brodmann's Area; i10:20= International 10:20 System Labels; Superior frontal gyrus = DLPFC; Inferior frontal gyrus =VLPFC

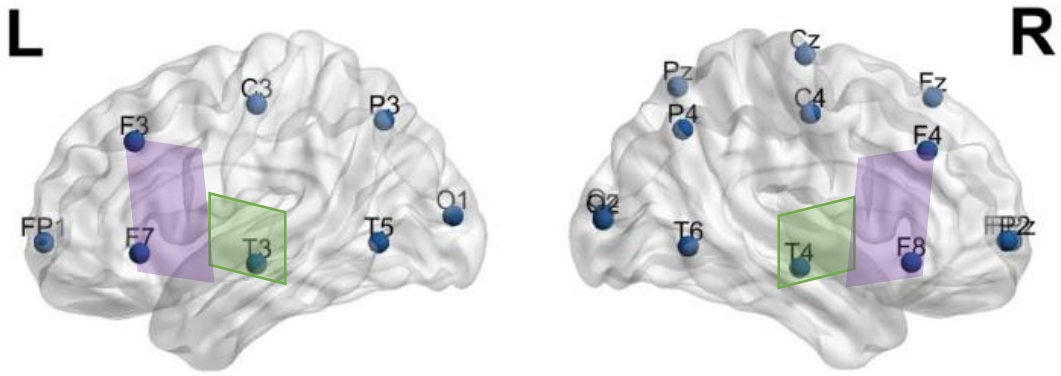


Figure 32

Brain Areas Investigated in Relation to the i10:20 System – side view

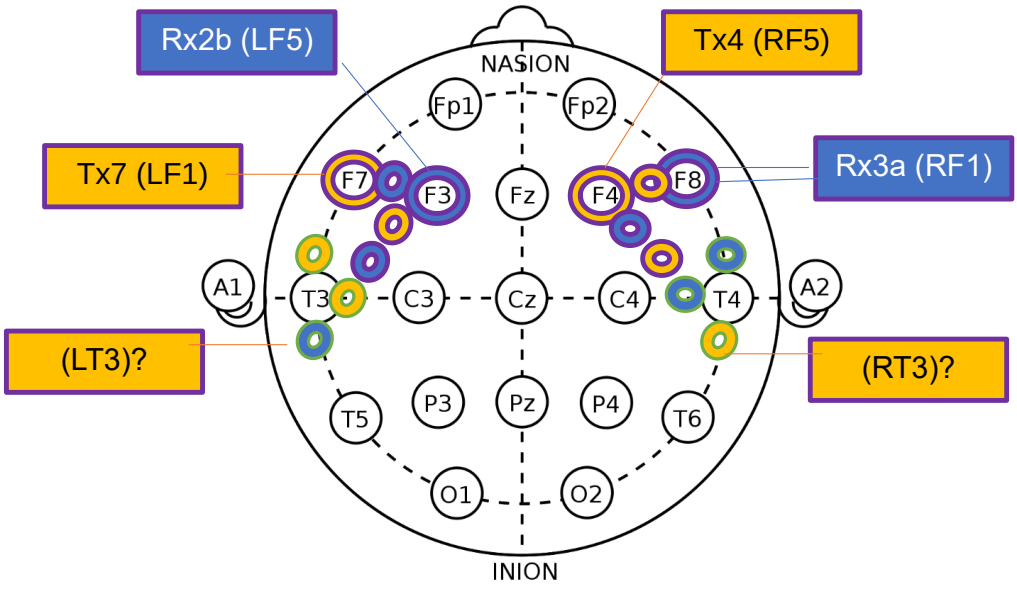
Notes: frontal areas near F3/F4 Superior/DLPFC and F7/F8 Inferior/VLPFC shown in purple, and temporal area around T3/T4 shown in green.

Table 14

Optode Channels (Receiver to Transmitter Pairs) and Brain Regions

i10:20 /location	Left Channels		Relative position				Right Channel		Relative position			
Behind/ above ear	**LT1**	Rx4b - Tx6	LH	TL	SU	PO	**RT1**	Rx1a - Tx1	RH	TL	SU	PO
Above ear	**LT2**	Rx4b - Tx8	LH	TL	IN	PO	**RT2**	Rx2a - Tx1	RH	TL	IN	PO
T3/T4	**LT3**	Rx3b - Tx8	LH	TL	SU	AN	**RT3**	Rx2a - Tx3	RH	TL	SU	AN
F8 Inferior PFC front	**LF1**	Rx3b - Tx7	LH	FL	IN	AN	**RF1**	Rx3a - Tx3	RH	FL	IN	AN
F8 Inferior PFC (lower)	**LF2**	Rx3b - Tx6	LH	FL	IN	PO	**RF2**	Rx1a - Tx3	RH	FL	IN	PO
Inferior PFC	**LF3**	Rx1b - Tx6	LH	FL	MI	PO	**RF3**	Rx1a - Tx2	RH	FL	MI	PO
Medial PFC	**LF4**	Rx1b - Tx5	LH	FL	SU	PO	**RF4**	Rx4a - Tx2	RH	FL	SU	PO
F3/F4 Superior PFC	**LF5**	Rx2b - Tx5	LH	FL	SU	AN	**RF5**	Rx4a - Tx4	RH	FL	SU	AN

Notes: LH= Left Hemisphere RH= Right Hemisphere TL=Temporal Lobe, FL= Frontal Lobe; Relative Position in the lobe group: Superior= SU, Inferior= IN, Posterior =PO, Anterior = AN



Left Optode and Channels **Right Optodes and Channels**

Figure 33

Top-Down Visual Arrangement of the Optodes in Relation to i10:20 Positions.

7.0 Study 2 Results

Sample Characteristics	Treatment of Data	Results	Summary

This chapter begins with an overview of the sample characteristics and treatment of data followed by descriptive statistics and inferential results. The analyses are organised into sections related to the study variables and predictions:

Results1

Physical Health: BMI and WC

Mental Health: depression and anxiety

Repetitive Negative Thinking: Rumination and Worry

Results2

Cognitive Function Performance: Sustained Attention and Inhibition Tasks

Results3

Executive function in Daily Living: BRIEF self-reported problems

Results4

Neurological: fNIRS Hemodynamic activity in the resting state

Results5

Neurological: fNIRS Hemodynamic activity during cognitive tasks.

Results6

Neurological: fNIRS Functional Connectivity

Treatment of Data

See Table 10 for a summary of measures and variables. Data from all demographics, questionnaire and anthropometric measures were entered into an SPSS document and then rechecked. Data were coded and SPSS was used to calculate total scores. Further variables were created based on the clinical cutoffs for each measure (categorical scores – see Appendix K Treatment of Data for a summary of the variables calculated). An Excel spreadsheet was made detailing completeness of measures for each participant.

Physical Health Data

Raw BMI was calculated [kg/m^2]. Categorical variables for BMI and WC were calculated using the IOTF cut-offs see Table 27. These categories are used to inform clinical assessments of the degree of health risk posed by adiposity per NICE guidelines (NICE, 2014). As BMI is normally distributed the number of participants at the very high and low end of the scale was small. To obtain sufficient power for the categorical analyses, a binary split was used: BMI 1 Low/increased (BMI<30) and 2 BMI high/very high risk (>=30). Waist Circumference risk already binary (1 low/normal, 2= Increased).

Mental Health Data

Raw scores for anxiety and depression were each summed to give total symptom scores (Anxiety 0-22, depression 0-24). Categorical variables were calculated using the symptom severity cutoffs (mild, moderate, moderately severe, severe per (Spitzer et al., 1999; Spitzer et al., 2006) see Table 10. Binary scores

were calculated reflecting whether the participant's symptoms were clinically significant (defined as a score of 10 or greater).

Repetitive Negative Thinking Data

Total rumination (score 10-40) was calculated with subtotals for brooding (items 1,3,6,7,8) and reflective (items 2,4,5,9,10) items (score 5-20). Worry items (2&3) from the GAD-7 were summed to give a total worry short-form scale. A similar method of creating a 2-item short form of the GAD and PHQ is detailed in the PHQ and GAD-7 instruction manual (Spitzer et al., 2006). Binary categorical repetitive negative thinking scores were also calculated based on rumination frequency (***Low***= 1 Almost never & 2 Sometimes; ***High***= 3 often & 4 Always) and worry severity (***Low***=0 none & 1 Mild; ***High***= 2 moderate & 3 severe). These followed the same procedure as for the GAD-2 and PHQ-2, but they are not clinically validated measures.

Cognitive Function Performance Task Data

For each task (attention and inhibition) participant reaction times and error data for block 1 and 2 of each task were extracted. This included error rates for total error, omission error and commission error. Validation checks were performed by comparing data for block 1 and 2 of the task. Overall mean reaction times for accurate trials and mean error (over both blocks) for each participant were used as task performance measures. For the Simon Task the difference in reaction time between accurate responses on incongruent minus congruent trials was calculated (also see Appendix K Treatment of Data for a table of variables calculated).

Executive Function in Daily Living (BRIEF) Data

Per Roth et al., (2005) response items were grouped into nine sub-scales and summed to give subtotals for each area of executive function (see Appendix J BRIEF Subscales Items & Definition). Selected items of the raw subscales were summed to obtain 2 Indexes: Behaviour Regulation Index (BRI) and Metacognition Index (MI) which are further summed to give the raw Global Executive Composite score.

Age-standardised T-scores and Confidence Intervals (90% CI based on standard error of 1.65) were established for each participant in relation to the reference population (see Roth et al., 2005) and applied to the subscales, indexes, and the global composite scores. A score of 50 represents the standardised population mean (T-score), and 65 (at 1.5 SD above the mean) are interpreted as 'abnormally elevated' or potentially clinically significant (Roth et al., 2005 pg13). This cut-off was applied to the T-scores to create categorical variables indicating executive function problem severity (whether participants difficulties were at, or below. the clinical level for each area of executive function and index).

Neurological (fNIRS) Data

fNIRS measurements were taken during the resting state scan, the CT task (sustained attention) and the SIMON task (motor inhibition). A Moving Gaussian smoothing filter was applied to all traces (the signal from each channel). During recording, automatic markers were placed within the trace to code the onset of each task, each block of experimental task and each block of rest (20 seconds). These markers were used to automatically label the haemodynamic signal for total, oxygenated and deoxygenated haemoglobin concentration over time during task and rest. The labels were therefore used to extract average fNIRS measurements within epochs of rest and task. Where the automatic markers failed, stimuli onset timings were used to calculate the epoch start and finish times (see Appendix L Neurocognitive Task Measurement Epochs).

The Beer Lambert Law was applied and means and standard deviations of the haemodynamic signal (during the resting state, and rest and task epochs for the cognitive tasks) for each optode pair (8 per hemisphere) were extracted from Oxysoft for each participant (Appendix L Neurocognitive Task Measurement Epochs). for each participant MS Excel was used to calculate the mean change in oxygenated and deoxygenated haemoglobin for each measurement epoch (resting baseline concentration minus task concentration) for each measurement channel. A PCA was carried out to verify the relative associations between measurement channels revealing a two-factor structure which broadly confirmed a distinction between the activation of frontal and temporal channels (see Results 4 PCA Findings). Average activity for each brain region (left temporal, right temporal, left frontal, right frontal) was also calculated for each participant (see Results 4 Resting State Descriptive Statistics) and examined in relation to continuous and categorical health.

Results 1 Physical Health, Mental Health, and Repetitive Negative Thinking

Prediction 1

a) Larger body measurements are related to greater negative affect and greater repetitive negative cognition.

Results 1 Descriptive Statistics

Table 15

Mean Continuous Physical, Mental Health and Repetitive Negative Thinking Scores for the Final Sample

N=88	Minimum	Maximum	Mean	SD	Skew (SE .26)	Kurtosis (SE .51)
Waist Circumference (cm)	62.35	130.73	89.11	15.60	.77	.13
BMI	17.6	52.65	27.44	6.92	1.12	1.24
Depression (PHQ-8)	0	22	6.73	5.72	.99	.20
Anxiety (GAD-7)	0	21	6.43	5.09	.84	.09
Rumination (RSS)	10	35	21.45	6.32	.15	-.82
RSS Brooding	5	19	10.75	3.91	.45	-.88
RSS Reflective	5	19	10.70	3.62	.27	-.86
Worry (GAD Q2&3)	0	6	2.17	1.91	.64	-.68

Notes: RSS= Ruminative Response Scale (RRS-10); GAD= General Anxiety Disorders (GAD-7); PHQ=Patient Health Questionnaire (PHQ-8)

Anthropometric Descriptive Statistics. Continuous raw BMI ranged from 17.60 to 52.65. Per International Obesity Task force BMI classification (see Table 15 above) this reflected 2% Underweight (UW=<18.50), 40% Normal range (N*R*=18.50-25.99), 28% Overweight (OW=26.00-29.99), 23% Obese (Ob>=30.00-39.99) and 4% Morbidly Obese (Mb>=40.00). See Figure 34 below BMI Category Frequencies.

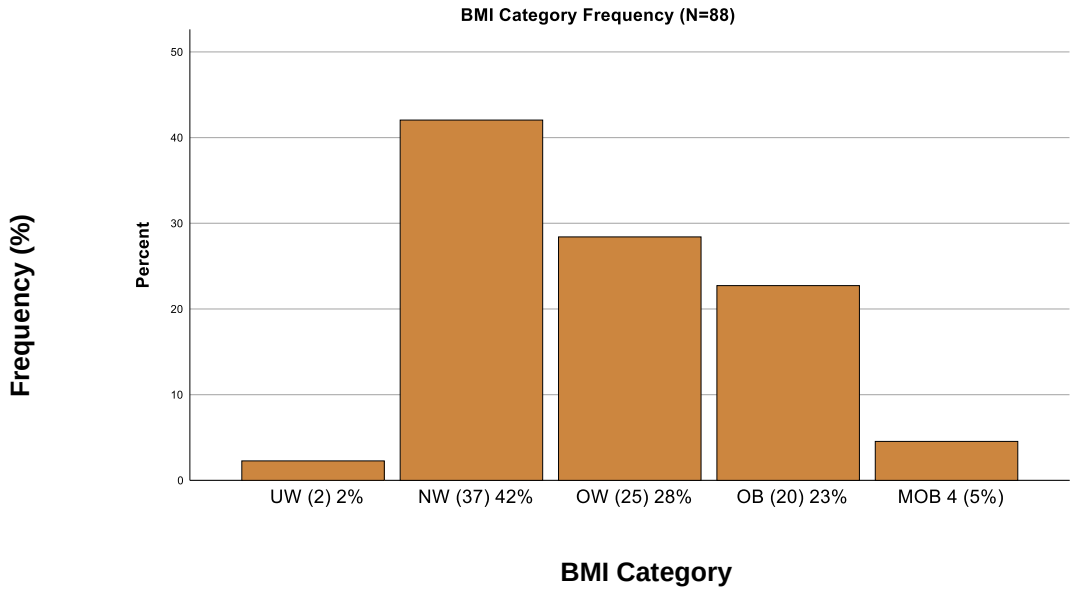


Figure 34
BMI Weight Category Frequency (N=88)

Waist circumference ranged from 62.35 to 130.73cm reflecting 41% in normal risk and 47% at increased risk of developing metabolic diseases.

Mental Health Descriptive Statistics. Anxiety and depression symptoms in the sample were categorised as : mild (0-5), moderate (6-10), moderately severe (11-15), severe anxiety symptoms (15-21). Depression scores ranged from 0 to 22, and anxiety scores ranged from 0 to 21. Figure 35 to Figure 36 illustrate the frequency and percentage of participants in each category.

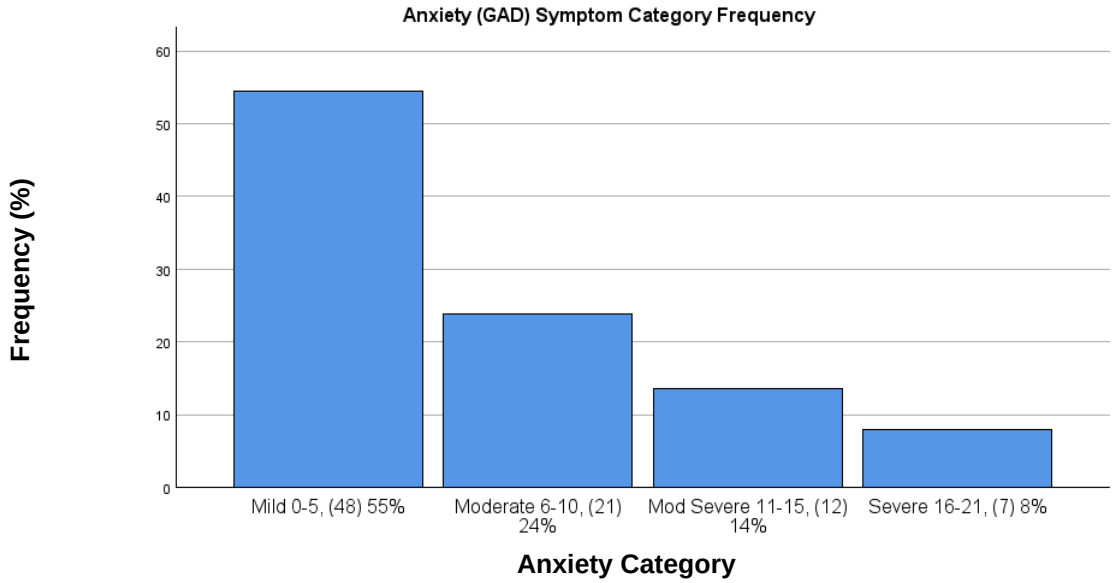


Figure 35
Anxiety (GAD) Symptom Category Frequency (N=88)
Notes: GAD= General Anxiety Disorders (GAD-7)

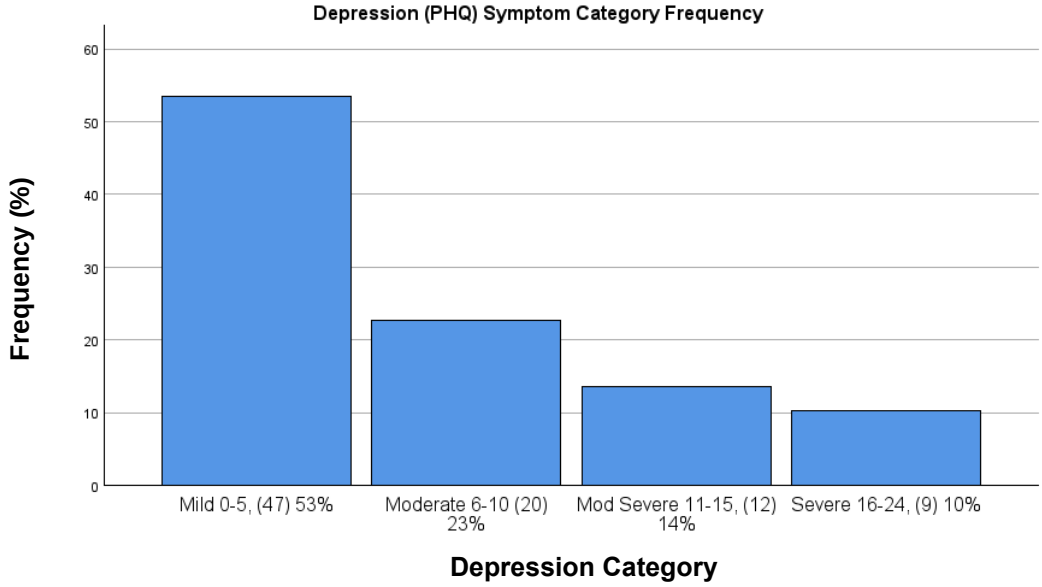


Figure 36
Depression (PHQ) Symptom Category Frequency (N=88)
Notes: PHQ=Patient Health Questionnaire (PHQ-8)

Using the GAD/PHQ clinical cutoff score of 10 and above, 22% (anxiety) and 24% (depression) of the sample experienced internalising/negative affective symptoms.

Repetitive Negative Thinking Descriptive Statistics. Total rumination scores for the RSS ranged between 10 and 35. Categorically 51% reported mild rumination, 42% moderate and 6% high levels (see Figure 37). The RSS is not a clinical scale, but a binary split (mild versus combined moderate and high rumination) indicated that 51% of the sample reported low rumination and 49% reported moderate-high levels.

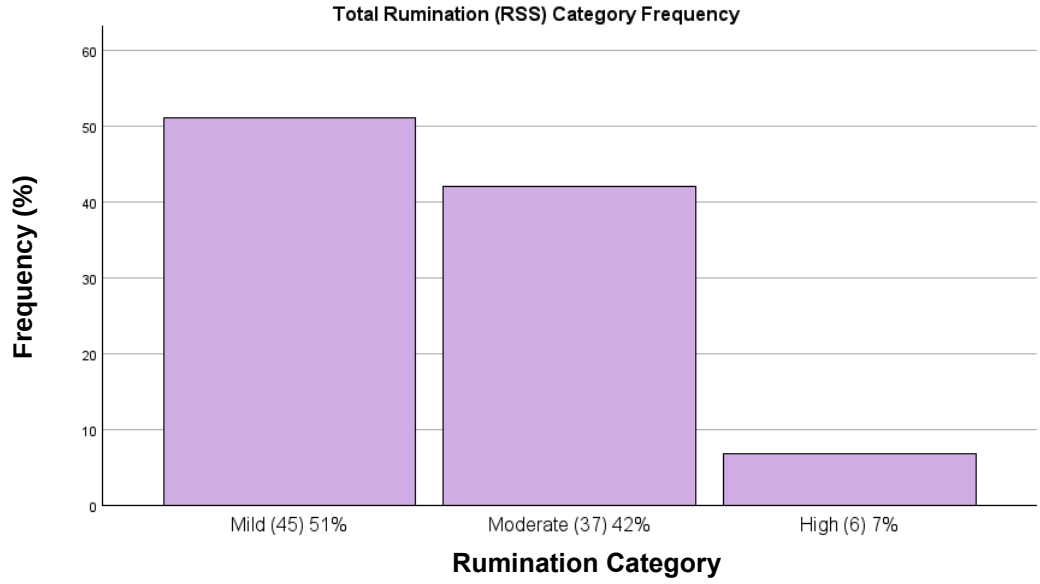


Figure 37
Total Rumination (RSS) Frequency by Category (n=88)
Notes: RSS= Ruminative Response Scale

Worry scores (extrapolated total of item 2&3 of the GAD as a short form) ranged from 0-6. The GAD categories were retained (see Figure 38 for the categorical frequencies). The binary scores indicated that 66% of the sample experienced low or no worry and 35% experienced moderate to high levels.

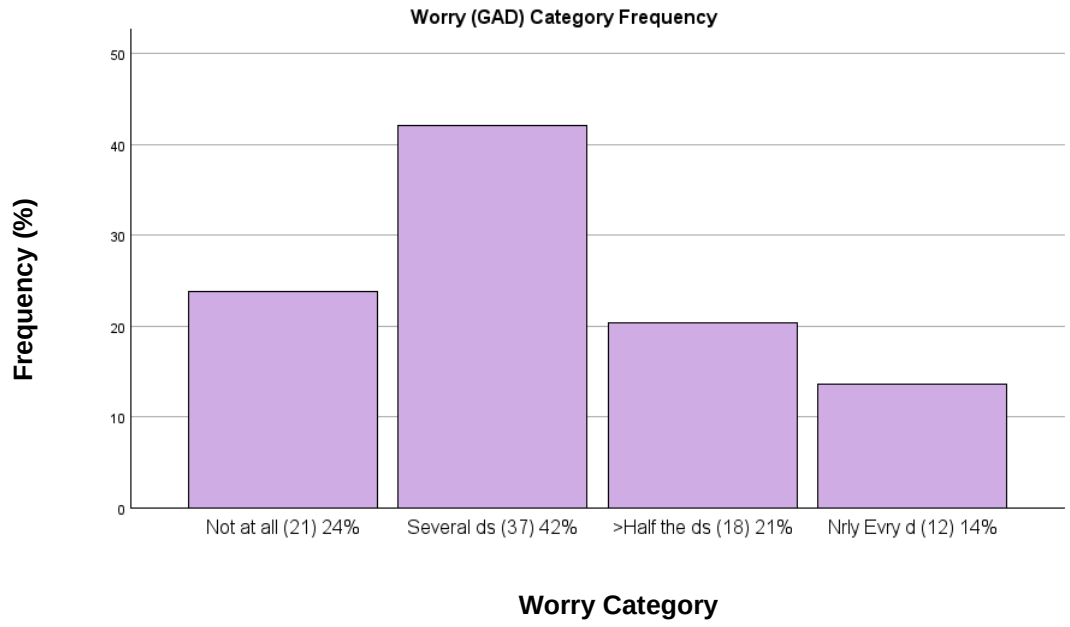


Figure 38
Worry (GAD Items 2&3) Category Frequency in the Sample (N=88)
Notes: GAD= General Anxiety Disorders (GAD-7)

Results1: Analyses

Measure Correlations	Physical and Mental health Correlations	Brooding and Physical Health Regressions	

Analytical Approach. Correlational analysis was used to examine the associations between different types of health measures (physical and mental health). Regressions were performed to help identify which physical measures were *most* predictive of mental health outcomes, and which mental health measures were *most* predictive of physical health outcomes. This analysis was done in line with the aims of the thesis, to help identify which measures might be more important to include in future studies of the interaction between physical and mental health. Understanding the limitations of the technique for creating models (e.g. differentiating a large list of potential predictors from nuisance variables without good theoretical reasoning per Smith, 2018), a forward stepwise method was used to gain a better understanding of measurement variables that were already show to be related to the outcome variable in previous studies, and were correlated with the outcome variable in the current study. Specifically, the technique was used to gain a quantitative estimation of how much of the variance explained by each measure overlapped, and how much variance in the outcome variable was contributed by each predictor. The theoretical rational for the order of entry of variables is explained for each regression.

Associations with Similar Measures. A series of Pearson correlations were first used to examine associations between measures of the same health category (see Table 16). Both physical health measures were in the expected direction with strong positive correlation between measures (WC and BMI r=.91, p<.001) providing criterion validity. Mental health measures also had strong highly significant

associations (depression and anxiety r=.72, p<.001) and moderate to strong relationships with repetitive negative thinking (depression and rumination r=.50, p<.001; depression and worry r= .58, p<.001) with anxiety and rumination (r=.44, p<.001) having a slightly smaller relationship than depression and rumination (r=.50, p<.001). For the rumination subscales, brooding rumination (RUMb r=.50 to.58, p<.001) had moderate-high correlations with mental health. Reflective rumination had small correlations with depression and anxiety (RUMr; with Depression r=.27, p=.013; with Anxiety r=.23, p=.032).

* ***Note****:* the worry measure was derived from the anxiety scale hence the very high correlation between these two variables (r=.90 p<.001*).*

Associations between Mental and Physical Health. To address the main prediction, correlation between physical and mental health measures (see Table 16) indicated that raw WC and BMI had small significant positive correlation with RUMb (r=.22, p=.043; r=.22, p=.041). This was the only mental health measure that was significantly correlated with physical health. The correlation between BMI and Depression was approaching significance (r=.20, p=.056). Proxy measures of adiposity (BMI and WC) therefore seem to be more relevant to rumination than negative affective conditions (depression, anxiety, or worry). To check this assumption regressions were performed to see whether depression added further variance over RUMb in predicting physical health.

Table 16

Pearson Correlation: Continuous Physical and Mental Health Variables

N=88		2 BMI	3 Dep	4 Anx	5 Rum(t)	6 Rum(b)	7 Rum(r)	8 Wor
1. WC *cm*	r	**.91****	.18	.04	.06	**.22***	-.13	-.01
	p	<.001	.097	.733	.586	.043	.224	.901
2. BMI *cm*	r		.20	.11	.07	**.22***	-.11	.05
	p		.056	.304	.493	.041	.324	.667
3. Depression	r			**.72****	**.50****	**.57****	**.27***	**.58****
	p			<.001	<.001	<.001	.013	<.001
4. Anxiety	r				**.44****	**.50****	**.23***	**.90****
	p				<.001	<.001	.032	<.001
5. Rumination (Total)	r					**.85****	**.82****	**.39****
	p					<.001	<.001	<.001
6. Rumination (Brooding)	r						**.40****	**.43****
	p						<.001	<.001
7. Rumination (Reflective)	r							**.22***
	p							.039
8. Worry	r							
	p							

Notes: *. Correlation is significant at the .05 level (2-tailed). **. Correlation is significant at the .01 level (2-tailed). Dep = Depression; Anx = Anxiety; Wor=Worry; RUMb= Brooding Rumination; RUMr= Reflective Rumination

Regression Brooding Rumination and Physical Health.

DV=Waist Circumference. A forward stepwise regression was conducted to see whether depression explained any variance in addition to that explained by brooding rumination (RUMb) in predicting Waist Circumference (WC). As the health risk conferred by WC is different depending on sex, sex was controlled for in the first step of the regression. RUMb and then depression were entered on subsequent

steps (see Table 17 for the r^2 change and beta coefficients). Gender (Beta=-.22) and RUMb (Beta=.23) added significant variance to the model, and each contributed around 5% variance. The association with gender was negative reflecting that males had larger WC (males code 1, females coded 2). The overall model was significant: $F(1,86) = 3.42$, $p = .021$, R^2 .11, explaining around 11% of variance in WC. Depression did not add significant variance (r^2 change = .01, $p = .421$) and the beta was not significant. This indicates that brooding rumination (a symptom of depression) is a better predictor of WC than depression.

Table 17

Regression of Mental Health Predictors on Waist Circumference

Step	Predictors of WC	R^2	Adjusted R^2	R^2 Change	df1	df2	Sig. F Change
1	Gender	.05	.04	.05	1	88	.040
2	Gender, RUMb Total	.10	.08	.05	1	87	.026
3	Gender, RUMb Total Depression Total	.11	.08	.01	1	86	.421

Step	Predictors of WC	Standardized Beta Coefficients	t	Sig.
1	Gender	-.22	-2.08	.040
2	RUMb Total	.23	2.26	.026
3	Depression Total	.17	.81	.421

DV: BMI. As with WC the Gender, RUMb and Depression were entered on separate steps (see Table 18 for the r^2 change and beta coefficients). Gender was not a significant predictor, RUMb contributed around 5% variance to the model (R^2 .06, $p = .030$). Depression contributed around 1% but this was not significant ($p = .314$). The overall model was not significant (F (1,87) =2.02, $p = .118$, $r^2 = .07$). This indicates that brooding rumination (a symptom of depression) is a better predictor of BMI than depression.

Table 18

Regression of Mental Health Predictors on BMI

Step	Predictors of BMI	R^2	Adjusted R^2	R^2 Change	df1	df2	Sig. F Change
1	Gender	.00	-.01	.00		88	.671
2	RUMb Total	.06	.03	.05	1	87	.030
3	Depression Total	.07	.03	.01	1	86	.321

Step	Predictor	Standardized Beta	t	Sig.
1	Gender	.05	.43	.617
2	RUMb Total	.23	2.30	.030
3	Depression Total	.13	.99	.321

DV: Brooding Rumination. A final regression was run to check the predictive relationship in the reverse direction. WC was a significant predictor of RUMb explaining around 5% of the variance (Beta = .23) however BMI did not add significant additional variance (Beta = .13), and the overall model was not significant (F1,87) =2.55, *p*=.084, R^2=.06, 6%). This indicates that WC (central adiposity) is a better predictor of RUMb than BMI. Additionally, the lower R^2 indicates that rumination is a better predictor of adiposity than adiposity is of rumination.

Table 19

Regression of Waist Circumference and BMI on Brooding Rumination

Step	Predictors of RUMb	R^2	Adjusted R^2	R^2 Change	df1	df2	Sig. F Change
1	Waist Circumference	.05	.04	.05	1	88	.030
2	BMI	.06	.03	.00	1	87	.617

Step	Predictor	Standardized Beta	t	Sig.
1	Waist Circumference	.23	2.22	.030
2	BMI	.13	1.01	.642

Results1 Summary

There were small, significant correlations between continuous measures of BMI and WC and brooding rumination (RUMb). There was a small correlation between BMI and depression, but this was not significant. Regression analysis found that RUMb explained 5% variance in WC and BMI; depression added around 1% additional variance but did not add significantly to the models. A separate regression (to check the predictive relationship in the reverse direction) found that WC was a significant predictor of RUMb, but BMI did not add significant variance to the model. These findings indicate that brooding rumination and waist circumference could be useful variables to consider in future investigations of the interaction between mental and physical health, because these measures may explain more variance than more commonly used depression and BMI measures.

Results 2 Cognitive Performance

Prediction 2

2a) Poorer performance will be seen on the more cognitively demanding task (Inhibition) than attention.

2b) Significantly worse cognitive performance for those with greater negative affect, and larger body size.

Cognitive Performance Descriptive Statistics

The experimental cognitive function tasks were the Continuous Performance Task (CT) which primarily measured attention and the Simon Task (ST) which primarily measured inhibition. The format of the tasks was similar to aid comparability of effects as attention capacity influences inhibition. The main measures were mean percentage error (MPE), mean reaction time (RT), and the Simon Effect (incongruent minus congruent reaction time/errors as a measure of inhibition efficiency). Table 20 outlines the descriptive results across the different conditions of each cognitive task. See section 7.2.4 for Treatment of Data and Appendix K for Coding and Calculation of Variables.

Table 20

Cognitive Performance Descriptive Statistics Mean Reaction Time (RT) and Mean Error Frequency (MEF), Standard Deviation, Skew and Kurtosis for Attention(CT) and Inhibition (CT)

Task	Mean RT	RT	SD	Skew	Kurtosis	Total error	MEF	SD	Skew	Kurtosis
Attention (CT)		353.92	53.91	1.03	1.09		1.29	1.68	1.46	1.61
						Omission	.14	.55	5.12	29.94
						Commission	1.15	1.48	1.38	1.42
Inhibition (ST)		409.73	76.48	1.51	2.89		11.78	9.79	1.64	3.26
	Congruent	395.16	80.51	1.57	3.15	Congruent	3.89	3.84	2.32	7.77
	Incongruent	425.98	73.87	1.39	2.44	Incongruent	8.49	6.93	1.45	2.02

Note: CT commission errors were incorrect responses i.e., failure to withhold a response, omission errors were incorrect withholding of response. ST congruent trials had the stimuli and response button on the same lateral side of the body, incongruent were on different sides of the body. Red font=skew/kurtosis present Skew standard error = .26; Kurtosis standard error = .51.

Results2: Analyses

Mean Percentage Error and Health T-test	Mean Reaction Time and Health T-test		

Analytical Approach. Initial analyses (t-test) sort to find out whether the attention task was more challenging than the inhibition task. Cognitive performance differences between binary high/low health groups (based on clinical cutoffs where available) were then examined (independent t-tests) for each of the cognitive outcome measures: mean percentage error, reaction time. For the Simon Task, differences in the Simon Effect (incongruent minus congruent trial reaction time) were also examined; longer reaction times and greater number of errors are indicators of less efficient inhibitory control.

Task Difficulty. Paired t-tests were used to compared performance on the attention task (CT) and Inhibition Task (ST). The ST had more errors than the CT, and longer reaction times than the CT. This appeared to confirm that the inhibition task was more challenging than the attention task (see Figure 39a&b Comparison of Error and Reaction Time by Task; Paired t-test). The mean reaction time (MRT) for the ST task was significantly longer than the CT (t (86) =-8.27, p<.001) indicating that the ST required more time to think and respond. The mean percentage error (MPE) for the ST task was significantly higher than the CT (t (86) =10.45, p<.001) indicating that participants found it more challenging to give an accurate response.

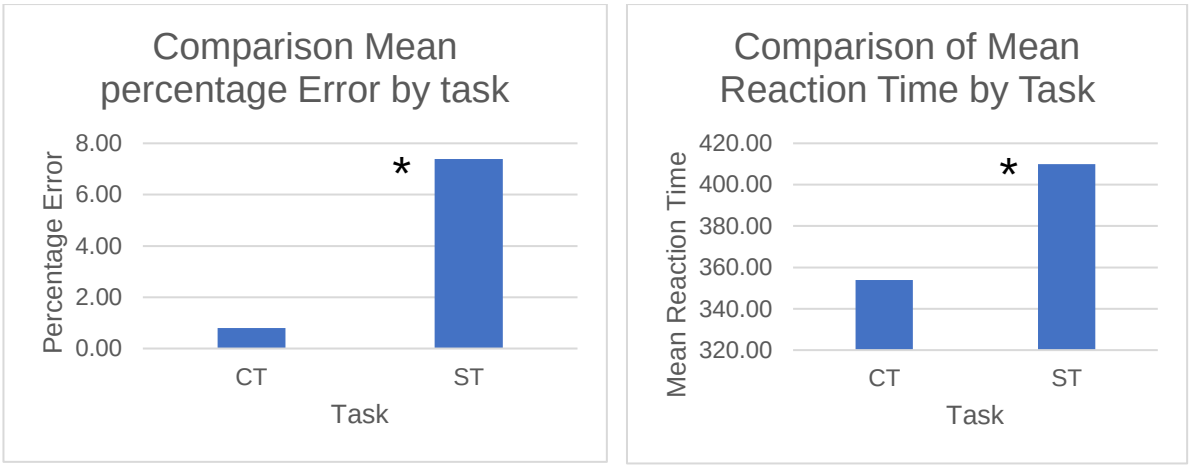


Figure 39a&b
Comparison of Error and Reaction Time by Task (Paired t-test)

Mean Percentage Error (MPE) and Physical Health. There was a significant effect of BMI risk group (see Table 21) on MPE in the CT task (*t* (85) =-1.81, *p*=.037 1-tailed), and a highly significant effect in the ST (*t* (85) =-2.58, *p*=.006, 1-tailed). Findings were in the expected direction, i.e., moderate/severe weight risk was associated with more errors. In the ST those with moderate/severe weight risk had significantly more errors on congruent trials than those of normal weight risk group (*t* (27) =-2.51, *p*=.018, 2-tailed) . This means that in addition to the general effect of task complexity (significantly more errors on the incongruent trials than congruent for the participants as a whole) the high body mass group had a more problems in the congruent trials compared to the low body mass group. The effect of WCr on error was not significant (see Table 22).

Mean Percentage Error and Mental Health / Repetitive Negative Thinking. Those with depression symptoms at clinical levels experienced more errors in the ST (*t* (85) =-2.35, *p*=.011, 1-tailed) but not the CT compared to those with lower depression scores (see Table 23). This indicates that depression may impact inhibition more than sustained attention. Those with high depression were

disadvantaged in all trial types, but there were more substantial effects in the incongruent trials (t (85) =-2.28, p=.025, 2-tailed). Spatial response inhibition appears to be less efficient in those with depression. There was no significant effect of anxiety symptom group on mean percentage error in either task (see Table 24).

Rumination group (see Table 25) had a significant effect on errors in the ST (t (67) =-2.37, p=.010, 1-tailed) with significant effects on both congruent (t (62) =-2.18, p=.033, 2-tailed) and incongruent trials (t (71) =-2.21, p=.030, 2-tailed). Investigation of the rumination subgroups indicates the effect on errors is linked to brooding rather than reflective rumination (see Table 26 & Table 27). Brooding rumination is associated with depression, but it impacted errors on both incongruent and congruent trials (both effects were significant). Those high in rumination therefore appear to be experiencing an effect on inhibition, plus an additional cognitive effect. This additional effect is leading them to make errors where no spatial conflict exists, and it is not likely to be due to sustained attention because there was no effect of rumination or depression on CT errors. There was no significant effect of worry on errors in either task (see Table 28).

Table 21

BMI Risk (Comparing Normal/Increased- Moderate/Severe) ***Mean Percentage Error*** *(MPE)*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
Group	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Normal/ Increased	64	**MPE**	.68	.96	t(85)=	-1.81	.037		-.44	64	**MPE**	6.37	5.13	t(85)=	-2.58	.006		-.62
Moderate/ Severe	23		1.13	1.20						24		10.03	7.70					
Normal/ Increased	64	**OM**	.20	.81	t(85)=	-1.25≠		.216	-.30	64	**CONG**	3.87	3.37	t(27)=	-2.51		.018	-.80
Moderate/ Severe	23		.54	1.80						24		7.50	6.78					
Normal/ Increased	64	**COM**	.88	1.26	t(85)=	-1.59		.115	-.39	64	**INCON**	9.84	8.20	t(85)=	-1.36		.176	-.33
Moderate/ Severe	23		1.37	1.34						24		12.66	9.67					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed; Normal/Increased BMI= BMI<30; Moderate/Severe BMI risk = BMI>=30.

Table 22

Waist Circumference Risk Group (Normal/Increased) ***Mean Percentage Error*** *– Non-sig*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
Group	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Normal/ Increased	41	MPE	.80	1.11	t(85)=	.03	.489		.01	41	MPE	6.83	6.00	t(86)=	-.77	.223		-.16
Moderate/ Severe	46		.79	.98						47		7.83	6.25					
Normal/ Increased	41	OM	.20	.78	t(85)=	-.64		.524	-.14	41	CONG	4.02	3.89	t(86)=	-1.54		.129	-.33
Moderate/ Severe	46		.36	1.41						47		5.59	5.40					
Normal/ Increased	41	COM	1.05	1.46	t(85)=	.27		.787	.06	41	INCON	10.49	9.39	t(86)=	-.12		.902	-.03
Moderate/ Severe	46		.97	1.14						47		10.72	8.07					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 23

Depression Group (Comparison Above and Below Clinical Cutoff) ***Mean Percentage Error*** *(MPE)*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
	N		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
NonClin	66	**MPE**	.77	.98	t(85)=	-.44	.331		-.11	67	**MPE**	6.53	5.59	t(85)=	-2.35	.011		-.59
Clinical	21		.88	1.23						21		10.03	7.08					
NonClin	66	**OM**	.22	.83	t(85)=	-.95		.344	-.24	67	**CONG**	4.37	4.25	t(85)=	-1.74		.085	-.44
Clinical	21		.50	1.85						21		6.43	6.08					
NonClin	66	**COM**	1.00	1.31	t(85)=	-.15		.885	-.04	67	**INCON**	9.46	7.88	t(85)=	-2.28		.025	-.57
Clinical	21		1.04	1.26						21		14.29	10.14					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed; NonClin= below the PHQ8/ GAD7 clinical threshold of 10.

Table 24

Anxiety Group (Comparing Above and Below Clinical Cutoff) ***Mean Percentage Error*** *– Non-sig*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
	N		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
NonClin	69	MPE	.77	1.10	t(85)=	-.44	.330		-.12	69	MPE	7.12	6.34	t(85)=	-.72	.238		-.19
Clinical	18		.89	.80						19		8.26	5.29					
NonClin	69	OM	.30	1.25	t(85)=	.23		.819	.06	69	CONG	4.91	5.20	t(85)=	.19		.849	.05
Clinical	18		.23	.67						19		4.67	2.97					
NonClin	69	COM	.97	1.36	t(85)=	-.59		.556	-.16	69	INCON	10.05	8.59	t(85)=	-1.15		.253	-.30
Clinical	18		1.17	1.00						19		12.63	8.86					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed; NonClin= below the PHQ8/ GAD7 clinical threshold of 10.

Table 25

Total Rumination group (Comparing Low – High) ***Mean Percentage Error*** *(MPE) Independent t-test*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
	N		**MPE**		**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Low	44	**MPE**	.62		.91	t(85)=	-1.63		.108	-.35	45	**MPE**	5.88	4.26	t(67)=	-2.37		.021	-.51
High	43		.98		1.14						43		8.92	7.32					
Low	44	**OM**	.09		.63	t(56)=	-1.58[≠]		.120	-.34	45	**CONG**	3.78	3.03	t(62)=	-2.18		.033	-.47
High	43		.48		1.50						43		5.99	5.95					
Low	44	**COM**	.84		1.24	t(85)=	-1.25		.214	-.27	45	**INCON**	8.64	6.52	t(71)=	-2.21		.030	-.48
High	43		1.18	1.34							43		12.67	10.11					

Note: [≠]= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 26

Brooding Rumination Group (Comparing Low-High) ***Mean Percentage Error*** *Independent t-test*

	Attention (Continuous Performance Task)									Simon Task (Inhibition)								
	N		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Low	49	MPE	.64	.91	t(85)=	-1.56		.122	-.34	49	MPE	5.82	4.25	t(57)=	-2.60≠		.012	-.59
High	38		.99	1.17						39		9.31	7.48					
Low	49	OM	.21	.88	t(85)=	-.68		.495	-.15	49	CONG	3.70	3.05	t(53)=	-2.45≠		.018	-.56
High	38		.38	1.44						39		6.31	6.08					
Low	49	COM	.82	1.21	t(85)=	-1.52		.131	-.33	49	INCON	8.67	6.38	t(59)=	-2.29≠		.025	-.52
High	38		1.25	1.38						39		13.04	10.46					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 27

Reflective Rumination (Comparing Low-High Groups) ***Mean Percentage Error*** *–Independent t-test Non-sig*

	Attention (Continuous Performance Task)									Simon Task (Inhibition)								
	N		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Low	44	**MPE**	.80	1.01	t(85)=	.05		.964	.01	45	**MPE**	6.47	4.58	t(70)=	-1.39≠		.168	-.30
High	43		.79	1.08						43		8.30	7.34					
Low	44	**OM**	.09	.63	t(56)=	-1.58≠		.120	-.34	45	**CONG**	4.22	3.55	t(85)=	-1.28		.205	-.27
High	43		.48	1.50						43		5.52	5.79					
Low	44	**COM**	1.10	1.39	t(85)=	.64		.523	.14	45	**INCON**	9.64	7.04	t(75)=	-1.07≠		.288	-.23
High	43		.92	1.19						43		11.63	10.07					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 28

Worry (Comparing Low / High Groups) ***Mean Percentage Error*** *– Independent t-test Non-sig.*

	Attention (Continuous Performance Task)									Inhibition (Simon Task)								
	N		**MPE**	**SD**			**1t p**	**2t p**	**d**	**N**		**MPE**	**SD**			**1t p**	**2t p**	**d**
Low	58	MPE	.79	1.11	t(85)=	-.09	.464		-.02	58	MPE	7.16	6.59	t(86)=	-.45	.329		-.10
High	29		.81	.91						30		7.77	5.17					
Low	58	OM	.32	1.34	t(85)=	.41		.684	.09	58	CONG	4.91	5.56	t(86)=	.15		.880	.03
High	29		.22	.65						30		4.75	2.87					
Low	58	COM	.98	1.39	t(85)=	-.26		.799	.06	58	INCON	10.19	8.70	t(86)=	-.63		.533	-.14
High	29		1.06	1.11						30		11.42	8.67					

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 29

Physical Health (Comparing Normal/Increased and High Adiposity Risk) ***Mean Reaction Time*** *Descriptives and Independent t-test*

RT		Attention (Continuous Performance Task)						Inhibition (Simon Task)					
		Mean	SD	Skew	SE	Kurtosis	SE	Mean	SD	Skew	SE	Kurtosis	SE
BMI	Normal/ Increased Risk (N=64)	354.40	57.6	1.01	.30	.91	.60	404.48	66.18	.94	.30	.65	.60
	High Risk (N=23)	352.60	43.08	1.03	.50	1.18	.90	423.73	99.23	1.73	.50	2.69	.90
						NS t(85)=.14 *p*=.893						NS t(31)=-.88≠, *p*=.386	
Waist Cr.	Normal/ Increased Risk (N=41)	357.60	58.14	.70	.40	-.35	.70	407.43	72.58	.81	.40	.27	.70
	High Risk (N=46)	350.60	50.26	1.46	.40	3.61	.70	411.74	80.45	1.98	.40	4.5	.70
						NS t(85)=.60, *p*=.550						NS t(86)=-.26, *p*=.793 (2t)	

Note: ≠=Equal Variances Not Assumed; Red font=skew/kurtosis present

Table 30

Mental Health (Comparing Groups Above and Below the clinical cutoff) Mean Reaction Time Descriptives and Independent t-test

RT		Continuous Performance Task						Simon Task						
		Mean	SD	Skew	SE	Kurtosis	SE		Mean	SD	Skew	SE	Kurtosis	SE
Depression	NonClin (N=66)	352.10	53.51	1.24	.30	1.91	.60	NonClin (N=67)	403.56	67.89	1.5	.30	3.74	.60
	Clinical (N=21)	359.80	56.07	.47	.50	-.63	1.00	Clinical (N=21)	429.44	98.47	1.25	.50	1.06	1.00
						t(85)=-.57≠, *p*=.572 (2t)							NS t(26)=-1.12≠, *p*=.271(2t)	
Anxiety	NonClin (N=69)	355.70	53.52	1.14	.30	1.43	.60	NonClin (N=69)	411.78	74.53	1.74	.30	4.11	.60
	Clinical (N=18)	347.30	56.44	.77	.50	.24	1.00	Clinical (N=19)	402.29	84.91	1.06	.50	.33	1.00
						t(85)=.58, *p*=.561(2t)							NS t(86)=.48, *p*=.635 (2t)	

Note: ≠ =Equal Variances Not Assumed; NonClin= below the PHQ8/ GAD7 clinical threshold of 10; Red font=skew/kurtosis present

Table 31

Repetitive Negative Thinking (Comparing low/high groups) and Mean Reaction Time Descriptives and Independent t-test for Each Task

RT		Continuous Performance Task						Simon Task						
		Mean	SD	Skew	SE	Kurtosis	SE		Mean	SD	Skew	SE	Kurtosis	SE
Rumination (total)	Low (N=44)	360.37	53.25	1.5	.40	2.6	.70	Low (N=45)	406.36	69.65	1.93	.40	5.26	.70
	High (N=43)	347.32	54.40	.68	.36	-.39	.71	High (N=43)	413.26	83.72	1.24	.36	1.76	.71
		NS t(85)=1.13, *p*=.261 (2t)							NS t(86)=.42, *p*=.675 (2t)					
Brooding Rumination	Low (N=49)	358.06	54.48	1.20	.34	2.05	.67	Low (N=49)	405.71	71.73	1.56	.34	4.09	.67
	High (N=38)	348.59	53.40	.86	.38	-.13	.75	High (N=39)	414.79	82.73	1.47	.38	2.17	.74
		NS t(85)=.81, *p*=.420 (2t)							NS t(86)=.-55, *p*=.583 (2t)					
Reflective Rumination	Low (N=44)	347.24	41.27	1.37	.36	2.97	.70	Low (N=45)	396.76	55.40	1.31	.35	1.41	.69
	High (N=43)	360.76	64.13	.71	.36	.02	.71	High (N=43)	423.31	92.36	1.23	.36	1.61	.71
		NS t(71)=.-1.17$^{\neq}$, *p*=.247 (2t-)							NS t(86)=.-16, *p*=.104 (2t)					
Worry	Low/none (N=58)	357.25	52.08	1.29	.31	2.04	.62	Low/none (N=58)	410.53	69.13	1.56	.31	3.70	.62
	High (N=29)	347.25	57.73	.77	.43	-.13	.85	High (N=30)	408.20	90.27	1.49	.43	2.22	.83
		NS t(85)=.81, *p*=.417							NS t(86)=.14, *p*=.893(2t)					

Note: $^{\neq}$=Equal Variances Not Assumed; Red font=skew/kurtosis present

The Simon Effect

Paired samples t-tests found a significantly longer reaction times and greater mean percentage errors for incongruent compared to congruent trials [Reaction time: t (87) =11.33, p<.001; Mean percentage errors t (87) =8.03, p<.001]. The Simon Effect (an indication of inhibitory control deficit) was calculated as the reaction time for incongruent trials minus the reaction time for congruent trials (only accurate trials were included), and the Simon Effect for error (number of incongruent errors minus congruent errors) was also investigated.

Simon Effect and Health. Binary health groups were analysed to see if there were significant differences in the Simon Effect based on health. There was a significant effect of BMI risk on the Simon Effect (t (86) =2.01, p=.048 2-tailed, d=.48, small to medium effect), indicating that reaction time was significantly affected by the spatial congruency effect of the task. Those in the high BMI risk group had longer MRT on incongruent trials and congruent trials (see Table 32), but the *difference* between incongruent trials and congruent trials was more pronounced in the normal/increased BMI risk group (MD 34.10) compared to those with high BMI risk (MD 22.06); those with low/increased BMI risk were faster on congruent trials (than incongruent), so this group showed a more obvious Simon Effect. This indicates the effects on BMI were due to more than inhibition alone. Further, there was no significant Simon Effect in relation to the mental health groups. This may indicate less robust effects on spatial response inhibition for depression and rumination.

Table 32

Simon Effect on Mean Reaction Time (incongruent minus congruent trials; ms) Group Means and Independent t-tests based on Binary Health Groups

			Group Means (ms)				MRT Incongruent-Congruent (ms difference)	
	Health Groups	N	Incongruent Mean(SD)	Congruent Mean(SD)	Mean Difference	SD	t	2t p
BMI RISK (Binary)	Normal/increased Risk	64	422.47 (64.74)	388.36 (69.22)	34.10	24.65	t(86)=2.01	.**048**
	High Risk	24	435.35 (95.05)	413.29(104.50)	22.06	26.22		
WC RISK(Binary)	Normal	41	424.23 (70.08)	392.61 (76.56)	31.63	26.15	t(86)=.28	.783
	Increased risk	47	427.50 (77.75)	397.39 (84.57)	30.12	25.21		
DEPRESSION (BINARY)	NonClin	67	419.30 (64.63)	389.28 (72.91)	30.02	25.62	t(86)=-.52	.601
	Clinical	21	447.30 (96.57)	413.92(100.85)	33.38	25.63		
ANXIETY (BINARY)	NonClin	69	428.32 (71.05)	396.83 (79.47)	31.50	26.42	t(86)=.47	.639
	Clinical	19	417.47 (84.88)	389.11 (86.13)	28.37	22.38		
BROODING RUMINATION	Low	49	420.50 (68.35)	392.17 (76.59)	28.34	24.43	t(86)=-1.02	.309
	High	39	432.86 (80.65)	398.92 (86.04)	33.94	26.81		
REFLECTIVE RUMINATION	Low	45	412.58 (52.72)	382.51 (60.02)	30.07	24.76	t(86)=-.28	.779
	High	43	440.00 (89.44)	408.40 (96.45)	31.61	26.55		
WORRY	Low	58	427.18 (67.31)	395.45 (86.36)	31.72	26.01	t(86)=.46	.648
	High	30	423.67 (86.36)	394.59 (95.41)	29.08	24.87		

Notes: NonClin= below the PHQ8/ GAD7 clinical threshold of 10; 2t P = 2-tailed significance.

Table 33

Simon Effect on Percentage Error (incongruent minus congruent trials) Group Means and Independent t-tests based on Binary Health Groups – Non Sig.

			Group Means		% Error Incongruent-Congruent (difference)			
	Health Groups	N	Incongruent	Congruent	Mean Difference	SD	t	2t p
BMI RISK (Binary)	Normal/Increased Risk	64	9.84 (8.20)	3.87 (3.37)	5.98	6.71	t(86)=.51	.613
	High Risk	24	12.66 (9.67)	7.50 (6.78)	5.16	6.86		
WC RISK(Binary)	Normal	41	10.49 (9.39)	4.02 (3.89)	6.46	7.64	t(86)=.93	.357
	Increased	47	10.72 (8.07)	5.59 (5.40)	5.13	5.81		
DEPRESSION (BINARY)	NonClin	67	9.46 (7.88)	4.37 (4.25)	5.09	5.89	t(86)=-1.66	.100
	Clinical	21	14.29 (10.14)	6.43 (6.08)	7.86	8.71		
ANXIETY (BINARY)	NonClin	69	10.05 (8.59)	4.91 (5.20)	5.14	6.30	t(86)=-1.63	.106
	Clinical	19	12.63 (8.86)	4.67 (2.97)	7.96	7.84		
BROODING RUMINATION	Low	49	8.67 (6.38)	3.40 (3.05)	4.97	5.70	≠t(68)=-1.18	.242
	High	39	13.04 (10.46)	6.31 (6.08)	6.73	7.78		
REFLECTIVE RUMINATION	Low	45	9.64 (7.04)	4.22 (3.55)	5.42	6.32	t(86)=-.48	.634
	High	43	11.63 (10.07)	5.52 (5.79)	6.10	7.17		
WORRY	Low	58	10.19 (8.70)	4.91 (5.56)	5.28	5.94	t(86)=-.92	.362
	High	30	11.42 (8.67)	4.47 (2.87)	6.67	8.05		

Note: ≠=Equal Variances Not Assumed; 2t P = 2-tailed significance; NonClin= below the PHQ8/ GAD7 clinical threshold of 10.

Results2 Summary

As anticipated, the error rate for the CT was low, with slightly more errors for commission (pressing when the target was not present i.e., failure to withhold a press) than omission. Being the more complex task the reaction time for the ST was slightly longer than the CT. One reason for the difference in performance between the CT and ST could therefore be task difficulty (e.g. greater cognitive load) as well as the effects of the construct being investigated (sustained attention and response inhibition). More errors and longer reaction times were observed on the incongruent trials than congruent indicating a successful 'Simon Effect' (spatial response conflict) was produced.

Binary health groups (high/low based on clinical cutoffs where available) indicated a those with high BMI had significantly more attention errors and those with high BMI, high brooding rumination, and high depression had significantly more inhibition errors. There was no significant effect of health group on reaction time, however there was a significant effect of BMI on incongruent verses congruent reaction time (Simon Effect); those with a normal BMI had a larger mean difference in reaction times between incongruent and congruent trials compared to the high BMI group. There were no significant differences in Simon Effects (reaction time or error) between the binary mental health groups.

Results 3: Executive Function in Daily Living (BRIEF)

Prediction 3

a) Self-reported executive function problems will be more strongly associated with negative affect than body size,

b) Self-reported executive function problems will be related to worse performance in computer-based tasks (attention and inhibition)

Executive Function in Daily Living Descriptive Statistics

T-scores and clinical frequency scores are summarised in Table 34. The most common executive function problems reported were for working memory, (which were potentially clinically relevant in 22% of the sample), emotional control (20%), shifting (18%) and initiating tasks (17%). 12% reported clinical levels of problems in inhibitory control, planning and task monitoring. Overall, there was an even distribution of problems between the two indexes with slightly more problems reported in behaviour regulation than metacognition.

Table 34

BRIEF Mean T-Scores for Subscales and Indexes with Frequency of Scores At/Above and Below the Clinical Cut-off (65) [Participant 22 and 31 removed N= 90]

	BRIEF sub-scale N=90	Mean	SD	Kurtosis (SE=.50)	Skew (SE=.25)	Frequency Non Clin (%)	Frequency Clin (%)
Behaviour Regulation Index	Inhibitory Control	51.66	10.54	.70	0.87	79 (87.80%)	11 (12.20%)
	Emotional Control	53.34	12.33	-.16	0.75	72 (80.00%)	18 (20.00%)
	Self-Monitor	49.39	11.44	.73	0.99	81 (90.00%)	9 (10. 00%)
	Shifting	53.93	10.69	-.22	0.66	74 (82.20%)	16 (17.80%)
Metacognitive Index	Initiation	51.09	11.23	-.65	.64	75 (83.30%)	15 (16.70%)
	Organisation of Materials	46.08	10.19	.65	.70	82 (91.10%)	8 (8.90%)
	Planning	51.14	10.13	.34	.90	79 (87.80%)	11 (12.20%)
	Task Manage	52.06	10.46	-.25	.44	79 (87.80%)	11 (12.20%)
	Working Memory	55.73	11.60	-.11	.62	70 (77.80%)	20 (22.20%)
	Beh Reg Index T-score	52.77	11.25	.71	.89	78 (86.70%)	12 (13.30%)
	Met Cog Index T-score	51.52	10.77	.50	.83	80 (88.90%)	10 (11.10%)
	Gen Exec T-score	52.03	10.98	.38	.76	79 (87.80%)	11 (12.20%)

Results 3: Analyses

Executive Function Daily Living and continuous Physical Health	Executive Function Daily Living and continuous Mental Health & Repetitive Negative Thinking	Executive Function Daily Living and Performance Tasks

Analytical Approach. An exploratory approach was taken to examine how the BRIEF subscales and indexes related to measures of health and cognitive performance. Correlation matrices were used to help understand which aspects of self-reported EF problems were related to each health measure. Regression analyses were performed to see which (if any) of the health measures were most predictive of self-reported inhibition and emotional control (aspects of inhibition that relate to ideas presented in the CAMMPI model). Finally, to establish the level of agreement between self-reported EF and cognitive performance (mean percentage errors, reaction time and Simon Effect), independent t-tests comparing high/low participant groups based on the BRIEF clinical cutoffs (T Score >=65) were conducted.

Executive Function in Daily Living and Physical Health Correlation. Due to the large number of BRIEF subscales and indexes (see Chapter 2 and Appendix J BRIEF Subscales Items & Definition for a summary) a Pearson correlation matrix was used to examine associations between self-reported executive function and health. Continuous BMI and WC were primarily associated with the behaviour regulation index (r=.30; r=.32; see Table 35).

The strongest associations between adiposity and self-reported executive function problems were with ***self-monitoring*** (awareness and understanding of the

social behaviour of self and others; BMI r=.35; WC r=.40, p<.001), ***emotional control*** (both BMI & WC r=.28, p=.008) and ***inhibitory control*** (BMI r=.24, p=.024; WC r=.25, p=.019). Waist circumference had additional significantly and positive associations with the initiation, working memory and task monitor (error awareness) sub scales (p<.05).

Table 35

Pearson Correlations BRIEF Subscale T-Scores with Physical Health (Continuous Variables)

	BRIEF sub-scale		BMI *cm*	Mean Waist Circumference *cm*
Behaviour Regulation Index	**Inhibitory Control**	r	.24*	.25*
		p	.024	.019
	Shifting	r	.08	.12
		p	.454	.252
	Emotional Control	r	**.28****	**.28****
		p	.008	.008
	Self-Monitor	r	**.35*****	**.40*****
		p	<.001	<.001
Metacognition Index	**Initiation**	r	.20	.25*
		p	.060	.017
	Working Memory	r	.20	.22*
		p	.057	.034
	Plan/organise	r	.12	.19
		p	.260	.069
	Task-Monitor	r	.12	.22*
		p	.095	.036
	Organising Materials	r	.12	.153
		p	.273	.149
	Behaviour Regulation Index	r	**.30****	**.32****
		p	.004	.002
	Metacognitive Index	r	.177	.22*
		p	.095	.037
	General Executive score	r	.26*	**.30****
		p	.014	.004

Note: Significance ***=<.001; **= at <.01; *=Significant at <.05; BRIEF=Behaviour Ratings Inventory of Executive Function

Executive Function in Daily Living, Mental Health, and Repetitive Negative Thinking. Both BRIEF indexes (Behaviour Regulation and Metacognitive) had moderate to strong correlations with mental health and repetitive negative thinking. The only exception to this was the RUMr (reflective rumination subscale). To correct for multiple correlations significance was accepted below .01. Figure 40 shows a profile of daily living problems for each mental health measure. The width of each coloured band reflects the strength of relationship with each brief subscale (see Table 36 for the correlations). Shifting, emotional control, and planning/organising, appear to be the most salient executive function problems reported by those with greater mental health symptoms.

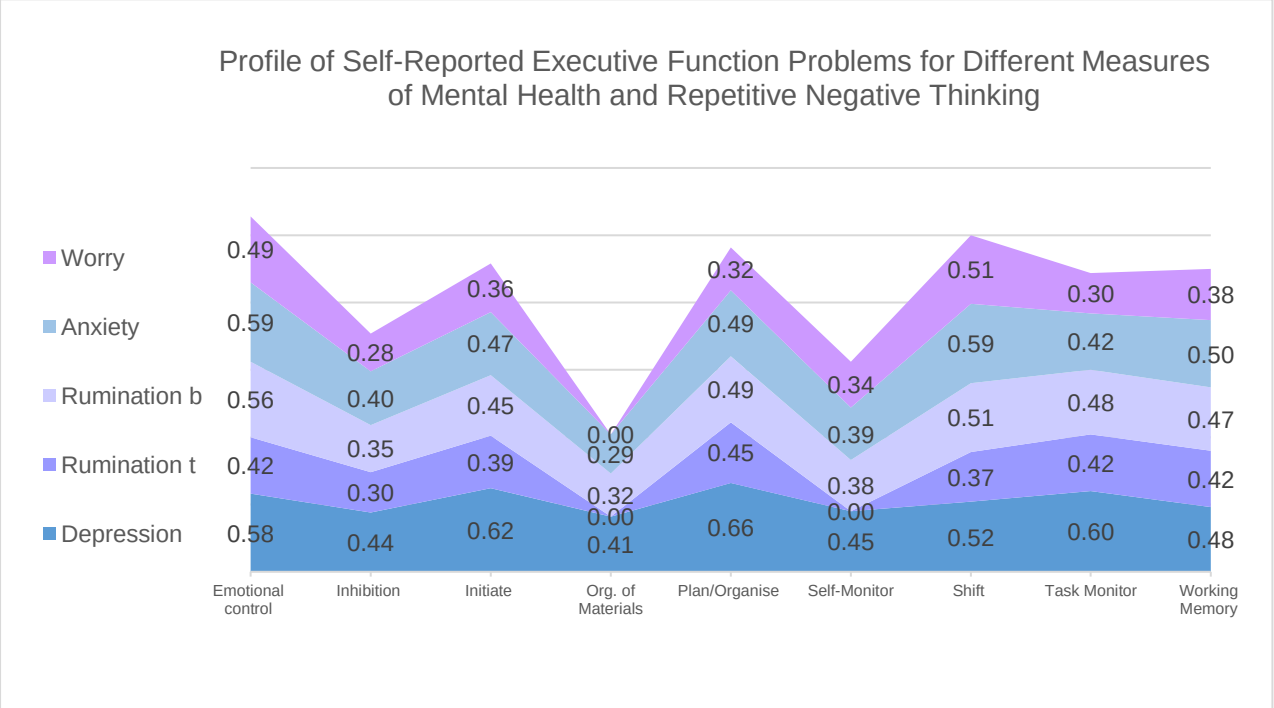


Figure 40

BRIEF Subscale Profile for Different Measures of Mental Health and Repetitive Negative Thinking Based on Pearson Correlations

Note: This diagram shows the profile of which executive function problems are associated with different mental health conditions (depression and anxiety) and repetitive negative thinking (worry and rumination). The thickness of each coloured band relates to the strength of correlation between the mental health measure and the BRIEF subscale. We can see that Organisation of Materials has a very weak relationship with worry, but a stronger relationship with depression. ; Rumination b= brooding rumination; Rumination t= total rumination See Table 34 for the Pearson Correlations

Table 36

Significant Pearson Correlations (p<.001) between Executive Function in Daily Living (BRIEF T-Score), Continuous Mental Health and Repetitive Negative Thinking.

BRIEF (Self-Reported Executive Function problems)	DEP	RUMb	Anxiety	Worry
Behaviour Regulation Index	.65 (<.001)	.67 (<.001)	.51 (<.001)	.51 (<.001)
Emotional Control	.58	.58	.60	.49
Shift	.52	.50	.60	.52
Inhibition	.44	.33	.44	.28
Self-Monitor	.45	.35	.40	.33
Metacognitive Index	.59 (<.001)	.51 (<.001)	.51 (<.001)	.36 (<.001)
Plan/Organise	.66	.50	.48	.31
Initiate	.62	.45	.47	.36
Task Monitor	.60	.48	.41	.29
Working Memory	.48	.47	.51	.38
Org. Materials	.41	.29	.29	*NS*

Note: BRIEF= Behaviour Ratings Inventory of Executive Function; **Green** = BRI Behaviour Regulation Index; **Orange** = MCI Metacognition Index; RUMb= Brooding Rumination; Reflective Rumination was not significantly correlated with any subscale except Plan/Organise (*r*=.22, *p*>.01)

The highest correlations for each of the mental health measures were as follows:

Depression. Depression (continuous and categorical) was positively correlated with all the 9 BRIEF subscales (*r*=.66) and all correlations were highly significant (*p*<.001). The largest correlations are with Plan/Organise (*r*=.66), Initiate (*r*=.62) and Task Monitor (*r*=.60).

Anxiety. Anxiety variables correlate with all BRIEF subscales (*r*=.59). The largest correlations with anxiety were: Emotional Control (*r*=.60) , Shifting (*r*=.60) and Working Memory (*r*=.51).

Rumination. Correlations with the RUMb subscale were slightly higher than depression. The largest correlations were emotional control (*r*=.58), shifting (*r*=.50) and planning/organising (*r*=.50). Reflective rumination (RUMr) scores were not significantly correlated with the BRIEF at a p<.01 significance level except the

positive Plan/Organise correlation was approaching significance. If reflective rumination is involved positively in planning and coping strategies, we would have expected a negative correlation with some of the MCI subscales, but this was not the case.

Worry (GAD7 Item 2&3). Worry was correlated with most subscales except Organisation of materials. The largest correlations between BRIEF subscales and worry were Shifting (*r*=.52), Emotional Control (*r*=.49) and Working Memory (*r*=.38).

Backward Regression of Physical and Mental Health Predictors on Self-Reported Inhibition Problems. Backward regression analysis was performed to examine to see which (if any) mental and physical health variables predicted inhibition and emotional control problems. Unlike the previous regression analyses there was a larger number of potential variables and there was not a clear theoretical basis for the order of entry, hence a Backward regression was selected as an appropriate technique to try and exclude variables that explained the least variance in the predictor in a more exploratory context. In backward regression all of the variables are entered in the first instance, and variables that explain little or no variance are removed on subsequent steps. In line with the aims of the study this technique was to help exclude variables with least predictive association to inform future studies.

Table 37
Backward Regression Physical and Mental Health Predictors of Daily Living Inhibition Problems

Step	Predictors:	R^2	R^2 Change	F Change	df1	df2	Sig. F Change
1	Worry, WC, RUM, Depression, BMI, Anxiety	.33	.33	6.71	6	81	.000
2	Worry, WC Depression BMI, Anxiety	.33	.00	.10	1	81	.756
3	Worry, WC, Depression, Anxiety	.33	.00	.55	1	82	.462

4	Worry, WC, Anxiety	.31	-.02	2.18	1	83	.143

Depression, RUMb and BMI were removed from the model on subsequent steps with no significant change in F. The final model (see Table 37, Step 4) was significant and explained 31% of the variance in self-reported inhibition (*F* (1,83) =12.52, *p*<.001, R^2=.31, 31%), however the only significant predictor of inhibition problems was anxiety (Beta=.75, *t* =3.01, *p*=.003).

Backward Regression of Physical and Mental Health on Emotional Control Problems. A second backward regression (see Table 38) was used to identify which if any health variables were significant predictors of Emotional Control problems (another facet of inhibition that is relevant to the CAMMPI model).

Table 38

Backward Regression Physical and Mental Health Predictors of Emotional Control Problems

Step	Predictors	R^2	R^2 Change	F Change	df1	df2	Sig. F Change
1	Worry , WC, RUMb , Depression , BMI, Anxiety	.52	.52	14.67	6	81	.000
2	Worry , WC, RUMb , Depression , Anxiety	.52	.00	.39	1	81	.535
3	Worry , WC, RUMb , Depression , Anxiety	.51	.00	.70	1	82	.407
4	Worry , WC, RUMb , Anxiety	.50	-.01	1.97	1	83	.164

Notes: WC= Waist Circumference; RUMb= Brooding Rumination; df= degrees of freedom; Sig= Significant

BMI, Worry and Depression were removed from the model on subsequent steps (see Table 38) as they did not add significant variance. The final model was significant and explained 51% of the variance in Emotional Control (*F* (1,83) =28.33, *p*<.001, R^2=.50, 51%). The only significant standardised betas predicting Emotional Control problems were anxiety (Beta = .52, *t*=2.36, *p*=.021) and RUMb (Beta .28, *t*=2.92, *p*=.005).

Executive Function in Daily Living (BRIEF) and Cognitive Performance

Mean Percentage Error (see Table 39 to 43). A series of Independent t-tests on binary BRIEF groups (those with scores above and below the clinical cutoff of 65) found a significant effect of self-reported Inhibition group on the inhibition task errors. Those with high self-reported inhibition had higher mean percentage error in the ST task (*t (*86) =-1.81, *p*=.037 1-tailed, d=.56) however this did not survive correction for multiple comparisons (alpha = .016). No other self-reported EF scales had a significant effect on mean percentage error in the CT and ST tasks.

Mean Reaction Time *(see Table 45 & 46).* There was no significant association between BRIEF executive function group (clinically significant/ non-clinically significant T score groups) and reaction time on either the sustained attention or inhibition performance task. This means that despite some participants having potentially clinically significant self-reported executive function problems, their responses in the attention and inhibition tasks were not significantly slower. This could be because their thought processes while completing the task were not much different across the two groups (i.e. the task did not tap into the source of their self-reported problems). For most self-reported EF problems, there was no significant effect on task accuracy (percentage errors) so if participants did use any strategies used to answer more quickly (e.g. responding without thinking), they did not appear to show between group differences or substantially enhance accuracy.

Table 39

Behaviour Regulation Index (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error*** *(MPE) – Non-Sig*

	Continuous Performance Task (Attention)							Simon Task (Inhibition)						
	N		**MPE**	**SD**		**t**	**1t p**	**N**		**MPE**	**SD**		**t**	**1t p**
Non Clin<65	75	MPE	.80	1.07	t(85)=	.08	.467	76	MPE	7.03	5.73	t(86)=	-1.29	.100
Clin >=65	12		.77	.84				12		9.48	8.17			
Non Clin<65	75	OM	.31	1.22	t(85)=	.37	.358	76	CONG	4.69	4.28	t(86)=	-.84	.202
Clin >=65	12		.17	.60				12		5.94	7.45			
Non Clin<65	75	COM	1.01	1.33	t(85)=	-.04	.483	76	INCO	10.25	8.32	t(86)=	-.99	.162
Clin >=65	12		1.02	1.11				12		12.92	10.72			

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 40

Metacognitive Index (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error*** *(MPE) – Non-Sig*

	Continuous Performance Task (Attention)							Simon Task (Inhibition)						
	N		**MPE**	**SD**		**t**	**1t p**	**N**		**MPE**	**SD**		**t**	**1t p**
Non Clin<65	77	MPE	.72	.97	t(85)=	-1.85	.034	78	MPE	6.81	5.56	t(10)=	-1.74≠	.056
Clin >=65	10		1.36	1.42				10		11.69	8.63			
Non Clin<65	77	OM	.22	.80	t(9)=	-.74≠	.240	78	CONG	4.33	3.97	t(10)=	-1.79≠	.052
Clin >=65	10		.83	2.64				10		9.00	8.12			
Non Clin<65	77	COM	.93	1.27	t(85)=	-1.49	.070	78	INCO	9.98	7.99	t(10)=	-1.39≠	.097
Clin >=65	10		1.58	1.42				10		15.50	12.18			

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 41

Inhibitory Control (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error*** *(MPE)*

	Continuous Performance Task(Attention)							Simon Task (Inhibition)							
	N		**MPE**	**SD**			**p**	**N**		**MPE**	**SD**			**p**	**d**
Non Clin <65	76	MPE	.77	1.05	t(85)=	-.54	.295	77	MPE	6.92	5.62	t(86)=	-1.81	.037	-.56
Clin >=65	11		.95	.97				11		10.45	8.58				
Non Clin <65	76	OM	.33	1.23	t(85)=	.88	.190	77	CONG	4.48	4.09	t(11)=	-1.23≠	.123	
Clin >=65	11		.00	.00				11		7.50	8.00				
Non Clin <65	76	COM	.96	1.28	t(85)=	-.95	.172	77	INCO	10.06	8.03	t(86)=	-1.58	.059	
Clin >=65	11		1.36	1.38				11		14.43	11.98				

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 42

Shifting (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error*** *(MPE) – Non-Sig*

	Continuous Performance Task(Attention)							Simon Task (Inhibition)						
	N		**MPE**	**SD**		**t**	**1t p**	**N**		**MPE**	**SD**		**t**	**1t p**
Non Clin <65	71	MPE	.80	1.07	t(85)=	.10	.461	72	MPE	6.77	5.51	t(18)=	-1.55≠	.069
Clin >=65	16		.77	.94				16		10.04	8.02			
Non Clin <65	71	OM	.32	1.25	t(85)=	.60	.275	72	CONG	4.51	4.20	t(86)=	-1.44	.077
Clin >=65	16		.13	.52				16		6.41	6.84			
Non Clin <65	71	COM	1.00	1.30	t(85)=	-.11	.455	72	INCO	9.83	7.74	t(18)=	-1.42≠	.087
Clin >=65	16		1.04	1.29				16		14.14	11.62			

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 43

Emotional Control (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error****(MPE) – Non-Sig*

	Continuous Performance Task(Attention)							Simon Task (Inhibition)						
	N		**MPE**	**SD**		**t**	**1t p**	**N**		**MPE**	**SD**		**t**	**1t p**
Non Clin <65	69	MPE	.73	1.01	t(85)=	-1.07	.143	70	MPE	6.99	5.65	t(86)=	-1.13	.130
Clin >=65	18		1.03	1.16				18		8.82	7.71			
Non Clin <65	69	OM	.18	.78	t(18)=	-1.06$^{\neq}$	.152	70	CONG	4.71	4.27	t(86)=	-.55	.291
Clin >=65	18		.69	2.02				18		5.42	6.59			
Non Clin <65	69	COM	.97	1.35	t(85)=	-.59	.278	70	INCO	10.25	8.23	t(86)=	-.77	.222
Clin >=65	18		1.17	1.08				18		12.01	10.30			

Note: $^{\neq}$= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 44

Self-Monitoring (Comparing Groups Above and Below Clinical Cut-off) ***Mean Percentage Error****(MPE)*

	Continuous Performance Task(Attention)							Simon Task (Inhibition)						
	N		**MPE**	**SD**		**t**	**1t p**	**N**		**MPE**	**SD**		**t**	**1t p**
Non Clin<65	78	MPE	.81	1.04	t(85)=	.33	.371	79	MPE	7.08	5.87	t(86)=	-1.30	.099
Clin >=65	9		.69	1.13				9		9.86	8.00			
Non Clin<65	78	OM	.32	1.21	t(85)=	.79	.216	79	CONG	4.54	4.05	t(8)=	-1.02	.167
Clin >=65	9		.00	.00				9		7.64	8.98			
Non Clin<65	78	COM	1.01	1.27	t(85)=	.08	.467	79	INCO	10.28	8.64	t(86)=	-1.05	.149
Clin >=65	9		.97	1.61				9		13.47	8.77			

Note: $^{\neq}$= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial error; INCON= incongruent trial error; 1t p = 1-tailed; 1t p = 2-tailed

Table 45

*Behaviour Regulation Index (Comparing Groups Above and Below Clinical Cut-off) and **Mean Reaction Time** (RT) – Non-sig*

Mean Reaction Time	Non-Clin T Score <65			Clin T Score >=65					
	N	Mean	SD	N	Mean	SD	t-test		2t p
CT	75	356.30	53.40	12	339.07	57.05	t(14)=	.98	.344
ST	76	407.46	71.55	12	424.16	105.30	t(13)=	-.53	.605
CONG	76	393.05	75.56	12	408.51	110.06	t(13)=	-.47	.647
INCON	76	423.46	69.20	12	441.95	100.94	t(13)=	-.61	.551

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial; INCON= incongruent trial; 1t p = 1-tailed; 1t p = 2-tailed; CLIN= potentially clinically significant.

Table 46

*Metacognitive Index (Comparing Groups Above and Below Clinical Cut-off) and **Mean Reaction Time** (RT) – Non-sig*

Mean Reaction Time	Non Clin T Score <65			Clin T Score >=65					
	N	Mean	SD	N	Mean	SD	t-test		2t p
CT	77	353.24	53.44	10	359.21	60.13	t(85)=	-.33	.744
ST	78	404.52	69.65	10	450.43	113.81	t(10)=	-1.25≠	.241
CONG	78	390.39	74.53	10	432.39	115.72	t(10)=	-1.12≠	.290
INCON	78	420.12	66.21	10	471.66	112.40	t(10)=	-1.42≠	.187

Note: ≠= Equal Variance Not Assumed; OM= Omission Error; COM=Commission Error; CONG= Congruent trial; INCON= incongruent trial; 1t p = 1-tailed; 1t p = 2-tailed; CLIN= potentially clinically significant.

Self-Reported Executive Function and The Simon Effect

The following analyses examined the relationship between self-reported executive function problems in daily living and the Simon Effect (incongruent minus congruent performance on a spatial inhibition task). Specifically, Independent t-test were used to see if there were between group differences in the Simon Effect based on BRIEF executive function problem scores (above or below the cut-off for potential clinical significance). Table 47 shows the Simon Effect on mean percentage error

(incongruent minus congruent %Err) and Table 48 shows the Simon Effect for mean reaction times (MRT), along with the Independent t-test results. In general, self-reported EF problems appeared largely unrelated to the Simon Effect. There was a significant difference in Simon Effect reaction time between high/low groups for Organising Materials (t (86) =-2.01, p=.048 (2-tailed). The group that reported high problems in organising materials had a larger difference between their incongruent and congruent reaction times than the group with low problems. This indicates that those who self-reported high levels of organisational problems had less efficient spatial inhibition. This could indicate that spatial inhibition problems affect perceptions of being able to organise materials which makes logical sense, however the effect size was very small (d=-.08). There were no other significant differences in the Simon Effect between groups based on clinical cutoffs indicating no obvious differences in spatial response inhibition.

Table 47

Between Group Differences (Self-Reported Cognitive Problems) in the Simon Effect on Mean Percentage Error (Incongruent Minus Congruent Trials) with Independent t-test – Non-Sig

DV=Simon Effect (%Err)	Non-Clinical <65			Clinical >=65					
	N	Mean %Err In-co	SD	N	Mean %Err In-co	SD	t	2t *p*	Cohen's *d*
Behaviour Regulation *Index*	76	5.56	6.37	12	6.98	8.86	t(86)= -.68	.499	-.12
Metacognitive *Index*	78	5.66	6.27	10	6.50	9.94	t(86)= -.37	.711	-.37
Inhibition	77	5.58	6.34	11	6.93	9.24	t(86)= -.62	.537	.13
Shifting	72	5.31	5.79	16	7.73	9.90	t(17.35)= -.94	.358	-.27
Emotional Control	70	5.54	6.35	18	6.60	8.16	t(86)= -.60	.553	-.18
Self-Monitor	79	5.74	6.94	9	5.83	4.55	t(86)= -.04	.970	-.43
Initiation	73	5.10	5.84	15	8.92	9.59	t(16.20)= -1.48	.157	-.18
Working Memory	68	5.70	6.42	20	5.94	7.83	t(86)= -.14	.890	-.28
Planning	77	5.52	6.14	11	7.39	10.13	t(11.07)= -.60	.563	-.06
Organising Materials	80	5.70	6.21	8	6.25	11.18	t(7.44)= -.14	.895	-.74
Tasking Monitor	77	5.93	6.32	11	4.55	9.33	t(86)= .64	.527	-.15

Notes: 2t *p*= 2-Tailed Significance ; Non-Clinical= below the BRIEF cutoff of 65; Clinical = at or above the BRIEF cutoff of 65 for potentially clinically significant scores.

Table 48

Between Group Differences (Self-Reported Cognitive Problems) in the Simon Effect on Mean Reaction Time (Incongruent Minus Congruent MRT for Accurate Trials)

DV=Simon Effect (MRT)	Non-Clinical <65			Clinical >=65					
	N	Mean RT In-co	SD	N	Mean RT In-co	SD	t	2t *p*	Cohen's *d*
Behaviour Regulation *Index*	76	30.41	25.64	12	33.44	25.67	t(86)= -.38	.704	-.21
Metacognitive *Index*	78	29.74	24.72	10	39.27	31.25	t(86)= -1.11	.269	-.12
Inhibition	77	31.24	25.07	11	27.90	29.61	t(86)= .40	.688	-.20
Shifting	72	29.56	25.06	16	36.48	27.58	t(86)= -.98	.329	-.36
Emotional Control	70	29.90	25.86	18	34.40	24.52	t(86)= -.67	.508	-.16
Self-Monitor	79	29.71	25.51	9	40.55	24.87	t(86)= -1.21	.229	-.01
Initiation	73	30.04	25.96	15	34.63	23.69	t(86)= -.63	.529	-.58
Working Memory	68	29.22	23.96	20	36.27	30.27	t(86)= -1.09	.279	-.04
Planning	77	30.63	25.10	11	32.15	29.57	t(86)= -.18	.854	-.28
Organising Materials	80	29.12	25.01	8	47.78	25.87	t(86)= -2.01	.048	-.08
Task Monitor	77	30.34	25.21	11	34.20	28.63	t(86)= -.47	.642	.20

Notes: 2t *p*= 2-Tailed Significance ; Non-Clinical= below the BRIEF cutoff of 65; Clinical = at or above the BRIEF cutoff of 65 for potentially clinically significant scores.

Results3 Summary

Continuous BMI and WC had small to medium correlations with the BRIEF subscales in the Behaviour Regulation Index (highest correlations with self-monitoring, emotional control and inhibitory control). Depression and all mental health measures, (except reflective rumination) had moderate to strong correlations with both BRIEF Indexes (i.e. most subscales). Shifting, Emotional Control, and the Plan Organise subscales (depression) had the highest correlations with mental health outcomes. Backward regressions found that anxiety predicted self-reported Inhibition problems and anxiety, and brooding rumination together predicted Emotional Control problems.

Using high/low participant groups (based on the BRIEF clinical cutoffs) there was a significant difference in cognitive performance (mean percentage errors) based on self-reported Inhibitory Control problems. On the ST (inhibition task) the clinical group had more errors, but this did not survive multiple correction.

Participants who reported high problems in Organising Materials displayed less efficient spatial inhibition (based on the Simon Effect for reaction time). The effect size was very small, and the Simon Effect was not significantly different for any other groups.

Results4: fNIRS In the Resting State

Prediction 4

4a) Temporal lobe areas will show a greater increase in haemodynamic activity than the frontal cortex during the resting state.

4b) Larger body-sizes will be related to significant differences in the pattern of haemodynamic activity during the resting state, in the frontal and temporal lobe, (reflecting significantly different patterns of frontoparietal task control system and Default Mode Network activity compared to participants with normal weight).

4c) Greater negative affect will be related to significant differences in the pattern of haemodynamic activity during the resting state, in the frontal and temporal lobe (reflecting different patterns frontoparietal task control system and Default Mode Network compared to participants with low levels of negative affect).

fNIRS Measurement Channel Visualisation

for easy reference in upcoming sections the following diagram illustrates which areas were measured in relation to nearest 10:20 positions

Left Hemisphere **Right Hemisphere**

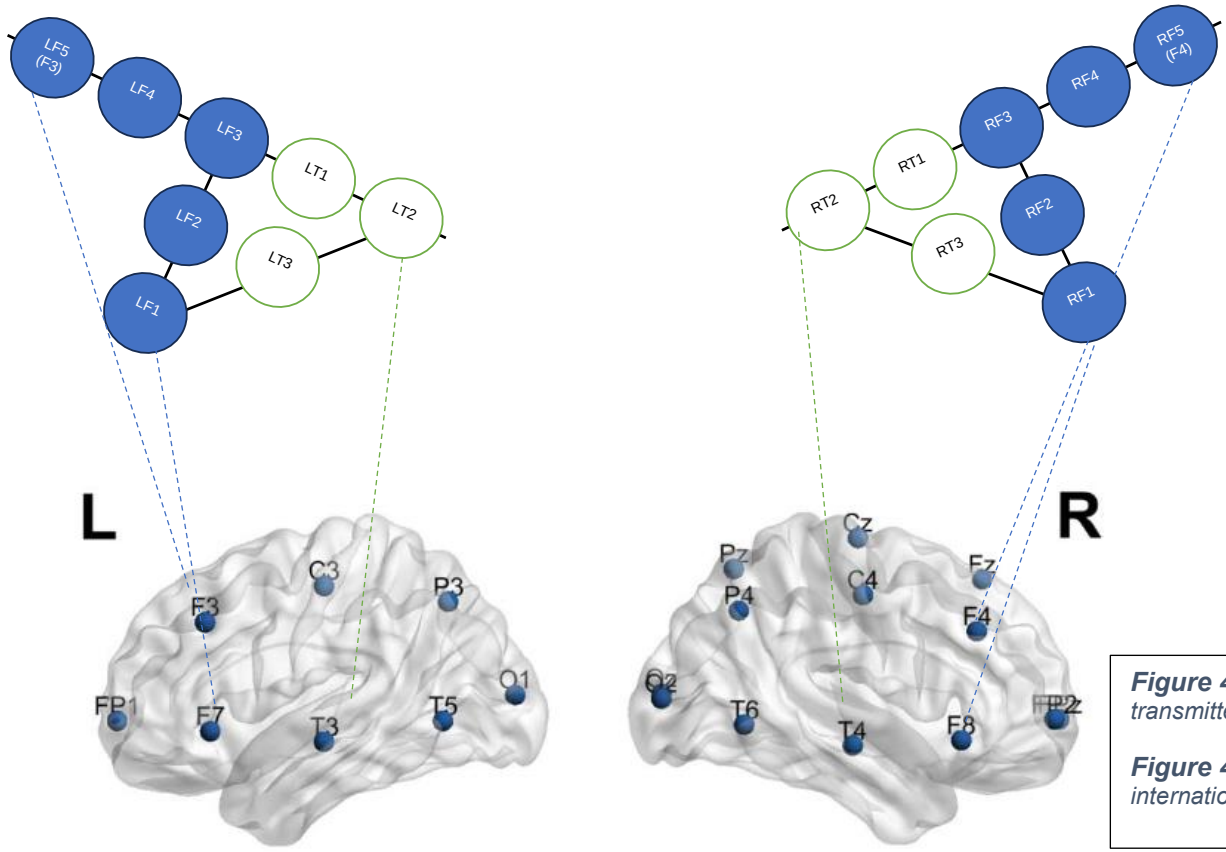


***Figure 41 Above**: fNIRS cap right hemisphere transmitters/ receiver locations.*

***Figure 42 Left**: Measurement channels in relation to the international 10:20 System.*

Resting State fNIRS Descriptive Statistics and Exploration of General Trends

All-Channel Means	fNIRS All-channel PCA	Regional Means	Regional fNIRS differences ANOVA

Analytical Approach. A great deal of data was generated from the 16 measurement channels hence the analysis strategy aimed to be parsimonious and reduce the number of comparisons. Table 49 shows the mean oxygenated and deoxygenated haemoglobin(OHb and HHb) concentration values as well as locations of the channels in relation to the i10:20 system. The rest of the results section focuses on the findings for oxygenated haemoglobin to be comparable with most fNIRS literature. The relative merits of each measure are debated, for example deoxygenated values could be considered a more reflective of the BOLD signal (Huppert et al., 2006), but they are not often reported as the main analysis. As this investigation was exploratory the general trends in the channel data were first explored using one-way ANOVA to examine regional differences and a Principal Components Analysis (PCA) to establish whether there were functional differences in the neural activity measures for the frontal and temporal lobe areas (per research question 4a). This was followed up with an exploration of regional differences in activity by lobe and hemisphere using ANOVA (to reduce the number of comparisons). Correlation and regression were then used to see if there was a significant association between neurovascular activity and health (per predictions 4b and 4c).

Table 49

Means and Standard Deviations for Average Concentrations of Oxygenated and Deoxygenated Haemoglobin during Resting State

	Channel	Oxy Mean (SD)	Deoxy Mean (SD)	Receiver (Rx)-Transmitter(Tx)	Relative position		Nearest 10:20		Channel	Oxy Mean (SD)	Deoxy Mean (SD)	Receiver (Rx)-Transmitter(Tx)	Relative position		Nearest 10:20
Right Temporal (RT)	**RT1**	-11.33 (19.99)	-9.87 (16.91)	Rx1a - Tx1	SU	PO	T3	Left Temporal (LT)	**LT1**	-12.69 (24.29)	-10.96 (18.45)	Rx4b - Tx6	SU	PO	T4
	RT2	-11.03 (21.85)	-8.74 (14.98)	Rx2a - Tx1	IN	PO	T3		**LT2**	-12.92 (22.98)	-9.06 (16.28)	Rx4b - Tx8	IN	PO	T4
	RT3	-3.70 (9.48)	-3.31 (8.86)	Rx2a - Tx3	SU	AN	T3		**LT3**	-5.20 (13.24)	-4.41 (11.88)	Rx3b - Tx8	SU	AN	T4
Right Frontal (RF)	**RF1**	-2.91 (12.53)	-2.04 (9.55)	Rx3a - Tx3	IN	AN	F8	Left Frontal (LF)	**LF1**	-5.32 (20.19)	-3.96 (12.23)	Rx3b - Tx7	IN	AN	F7
	RF2	-5.56 (14.14)	-5.41 (12.99)	Rx1a - Tx3	IN	PO			**LF2**	-7.09 (17.34)	-5.27 (12.29)	Rx3b - Tx6	IN	PO	
	RF3	-7.97 (20.59)	-5.62 (14.39)	Rx1a - Tx2	MI	PO			**LF3**	-9.68 (23.06)	-7.59 (16.72)	Rx1b - Tx6	MI	PO	
	RF4	-4.15 (13.63)	-3.11 (9.10)	Rx4a - Tx2	SU	PO	F4		**LF4**	-5.89 (15.89)	-5.96 (13.95)	Rx1b - Tx5	SU	PO	F3
	RF5	-3.35 (12.55)	-3.56 (11.40)	Rx4a - Tx4	SU	AN	F4		**LF5**	-2.76 (11.95)	-3.35 (12.24)	Rx2b - Tx5	SU	AN	F3

Notes: (Optode Channel Receiver to Transmitter pairs, relative position to one another and Nearest I10:20 Positions) H=Hemisphere Left=L, Right=R; L=Lobe: Temporal=T, Frontal=F; Relative Position in the lobe group: Superior= SU, Inferior= IN, Posterior =PO, Anterior = AN; F3/F4 = Frontal Lobe: Dorsolateral (PFC); F7/F8 = Frontal Lobe; T3/T4 = Temporal Lobe: Middle temporal lobe (MTL) [see Talairach atlas Lancaster et al.,2000 http://www.talairach.org/)

Resting State Mean Haemodynamic Activity Principal Components Analysis (PCA)

A Principal Components Analysis (PCA) was conducted to examine the general pattern of haemodynamic activity across all channels. This aimed to establish whether the channels were measuring similar functional activity in each lobe and hemisphere. The PCA confirmed a two-factor structure in the activity of the channels measured in the frontal lobe and temporal lobe during the resting state scan. Spatial plots of the PCA solutions are presented for each hemisphere Figure 43. Solutions had some variation but broadly confirmed spatial/ functional differences between the channels in different regions and the distinct brain activity they were measuring. The highest loadings (indicating similar patterns of haemodynamic activity) mapped on to the following regions of interest: the most closely associated ***frontal regions*** were channel 4 and 5 (positioned near to the F3/4 region, the superior and medial frontal gyrus see Figure 41 **Left**: Measurement channels in relation to the international 10:20 System.) and for the ***temporal grouping***, channel 1 and 2 were more closely associated (positioned near to the T3/T4 region, the superior and medial temporal gyrus, see Figure 32 & 33 for International 10:20 Visualisation).

Oxygenated Haemoglobin PCA Findings

Right Hemisphere. Sampling adequacy for the overall data set was moderate to high based on Kaiser-Meyer-Olkin Measure of Sampling Adequacy (KMO=.74). Bartlett's test of sphericity indicates significant differences in variance

(X^2= 599.01, *p*<.001) which means that findings should be interpreted with caution and may benefit from normalisation if appropriate.

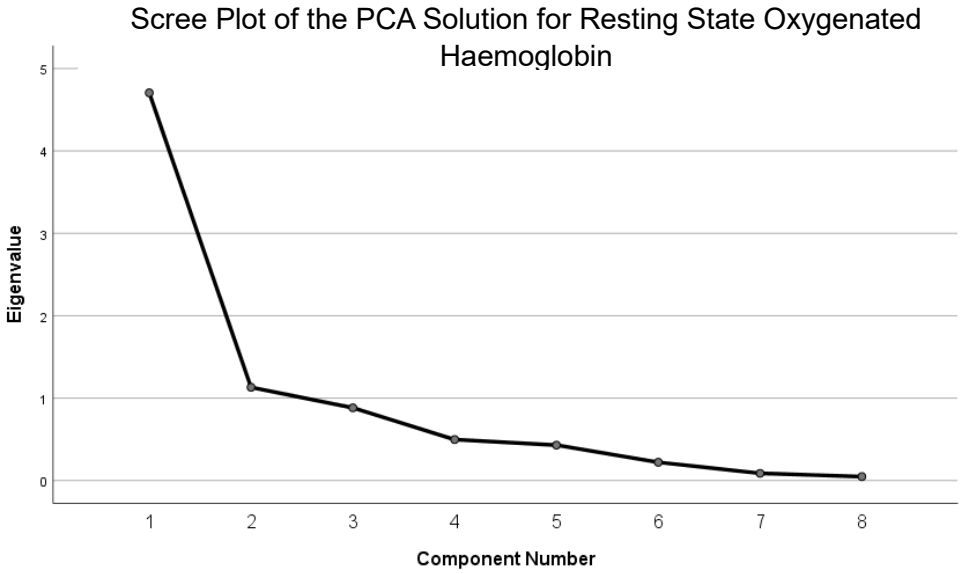


Figure 43
Scree Plot of Principle Components Analysis (PCA) Solution for Resting State Neurovascular Activity Channels
Note: Eigenvalues greater than 1.0 indicate potential individual components.

Small coefficients of less than .3 were suppressed. The PCA extracted 2 components with Eigenvalues over 1.0 (confirmed by scree plot). Varimax rotation (100 maximum iterations for convergence) and Kaiser Normalisation were applied.

The analysis was re-run with a fixed 2 component structure (both with Eigenvalues above 1.0). The rotated solution explained 72.95% of the total common variance (component 1 37.67%, component 2 35.28%) see Table 50.

Table 50

Principle Components Analysis Loadings for Neurovascular Activity Channels in the Right Hemisphere During Resting State (Oxygenated Haemoglobin)

	Component Loadings		
Optode Channel	**1**	**2**	**Communality**
RF4(OHb)	.82	.31	.76
RF5(OHb)	.78	-	.67
RT3(OHb)	.69	.34	.60
RF2(OHb)	.63	.61	.77
RF1(OHb)	.61	-	.38
RT1(OHb)	-	.96	.94
RT2(OHb)	-	.91	.87
RF3(OHb)	.65	.66	.86
Rotated Eigenvalue	3.01	2.82	
Rotated Variance (%)	37.67	35.28	
Total Variance (%)		72.95	

***Note*: Rotated Solution**: Varimax with Kaiser Normalization; RF=Right Frontal, RT=Right Temporal

Most loadings in the solution meet requirements of Pedhazur and Schemlkin (1991) other than optode RF2 and RF3, which load highly on to both components (indicating equal shared variance with both the frontal and temporal areas). The T3 optode channel appears to load more highly onto the frontal component than the temporal one. This is confirmed in the PCA plot.

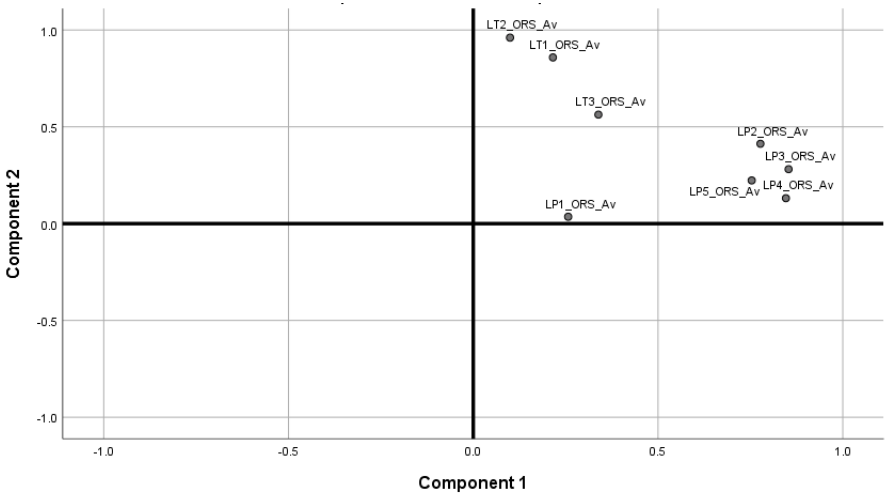
Left hemisphere. The PCA structure for the left hemisphere places ChT3 with the other two Temporal optodes (indicating that these three channels are a good fit for the left hemisphere temporal lobe area). Per the Pearson correlations, Frontal Channel one - ChF1 on the left hemisphere is not related to either component, possibly indicating that is a different area of functional connectivity.

Table 51

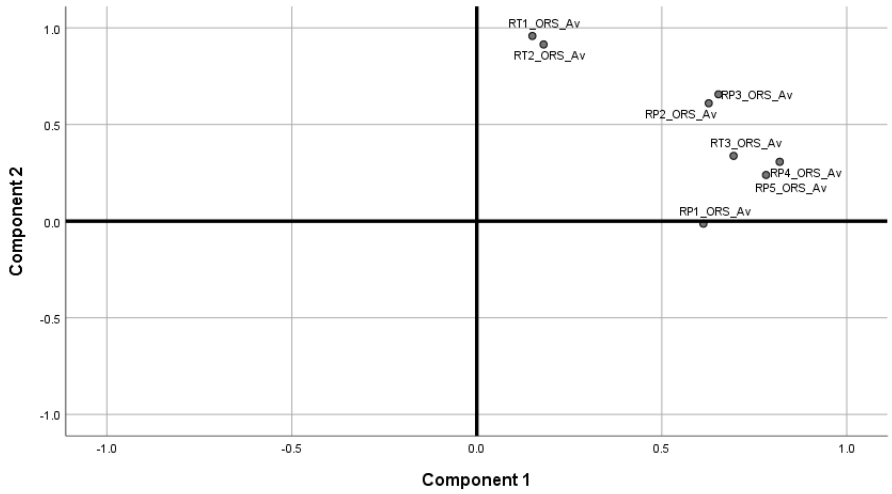
PCA Factor Loadings for Neurovascular Activity Channels in the Left Hemisphere

Left Hemisphere Channels Rotated Component Matrix		
	Component	
	1	2
LF3(OHb)	.85	
LF4(OHb)	.85	
LF2(OHb)	.78	.41
LF5(OHb)	.75	
LF1(OHb)		
LT2(OHb)		.96
LT1(OHb)		.86
LT3(OHb)	.34	.56

Note: LF= Left Frontal; LT = Left Temporal; Extraction Method: Principal Component Analysis. Rotation Method: Varimax with Kaiser Normalization.



44a Left Hemisphere 2-component solution
(oxygenated haemoglobin LT=left temporal, L*P*= left pre-frontal)



44b Right Hemisphere 2-component solution
(oxygenated haemoglobin RT=right temporal, R*P*= right pre-frontal)

Figure 44a&b
PCA Plots Rotated in Space Showing the Two-Component Structure (Left and Right Hemisphere)
Note: PCA= Principal Components Analysis; The plots indicate functionally different activity of the frontal and temporal areas for measurement channels in the left and right hemisphere.

Resting State Mean Regional Differences in Haemodynamic Activity

Mean regional haemodynamic activity in the Resting State (RS; see Table 52) shows higher values (i.e., greater overall change from baseline) in the temporal lobe than the frontal lobe, and slightly higher mean values in the left hemisphere than the right. This means that overall, at rest, greater haemodynamic activity was observed in the left temporal lobe channels (i.e.., left middle temporal gyrus appeared to be the most active region – see Figure 45). As haemodynamic activity is a measure of change the results are interpreted based on the ***magnitude*** of change rather that the direction (see Appendix Q Negative BOLD response/fNIRS Signal) for detail related to negative signal). A similar pattern was observed in both oxygenated and deoxygenated haemoglobin.

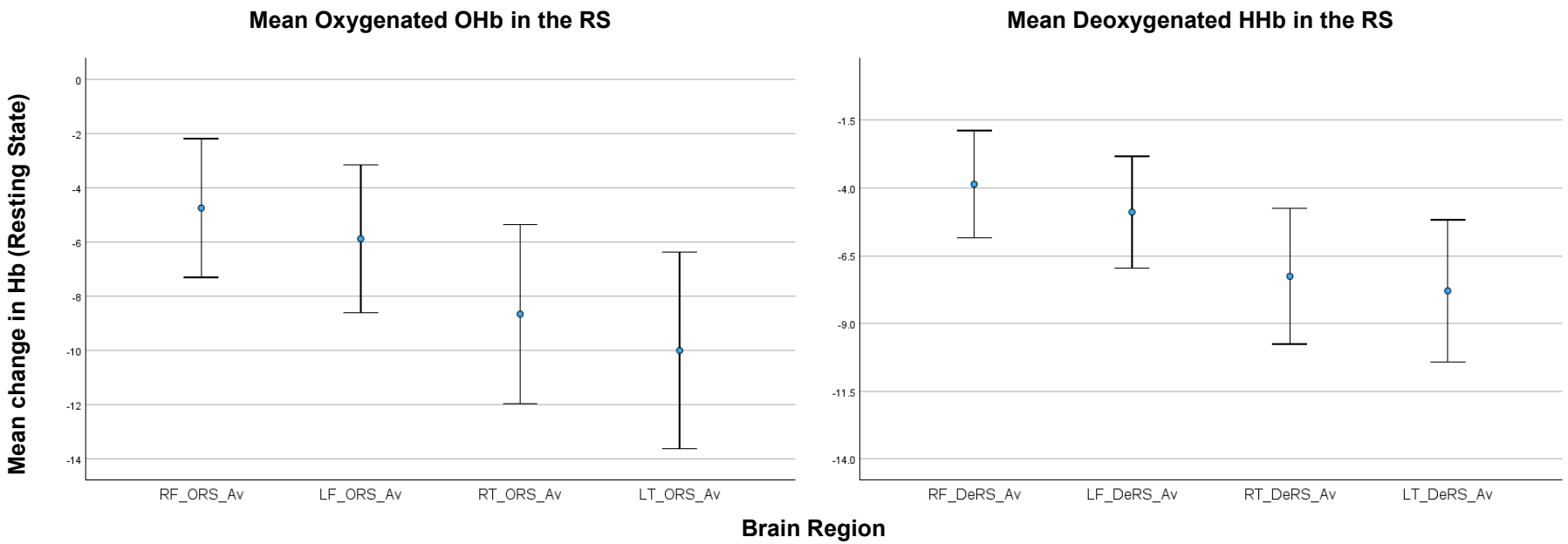


Figure 45

CI Graph of Average Changes in Oxygenated (OHb) and Deoxygenated Haemoglobin (HHb) During the Resting State by Brain Region (N88)

Note: RF_– Right Frontal; LF_ =Left Frontal; RT_ =Right Temporal; LT_ = Left Temporal

Table 52

Means and Standard Deviation (SD) for Average Change in Haemoglobin Concentration During Resting State by Brain Region (Lobe and Hemisphere).

fNIRS Resting State N=88	Frontal Lobe				Medial Temporal Lobe			
	Right (RF)		Left (LF)		Right (RT)		Left (LT)	
	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb
Minimum	-70.45	-52.91	-73.24	-48.14	-67.90	-40.88	-89.95	-50.16
Maximum	12.35	17.90	8.20	12.83	5.22	8.28	19.27	16.00
Mean	-4.79	-3.86	-6.15	-4.89	-8.68	-7.31	-10.27	-8.14
SD	12.01	9.34	13.09	9.72	15.48	11.75	17.23	12.75
Skew	-3.10	-2.41	-2.75	-2.28	-2.00	-1.34	-2.10	-1.29
Kurtosis	11.80	9.12	8.81	5.93	3.52	.55	5.40	1.61

Note: Skew SE = .26, Kurtosis SE=.51; N88 (removed case #61 due to poor signal).

Resting State Mean Regional Differences in Haemodynamic Activity - ANOVA

A one-way repeated measures ANOVA compared the mean change in haemodynamic activity over the four regions (Right Frontal=RF, Left Frontal =LF, Right Temporal =RT, Left Temporal=LT) and found a significant main effect of brain region (*F* (2.4, 211.11) =4.21, *p*=.011 (sphericity not assumed; see Figure 46 Mean Change in OHb from Resting State Baseline by Region).

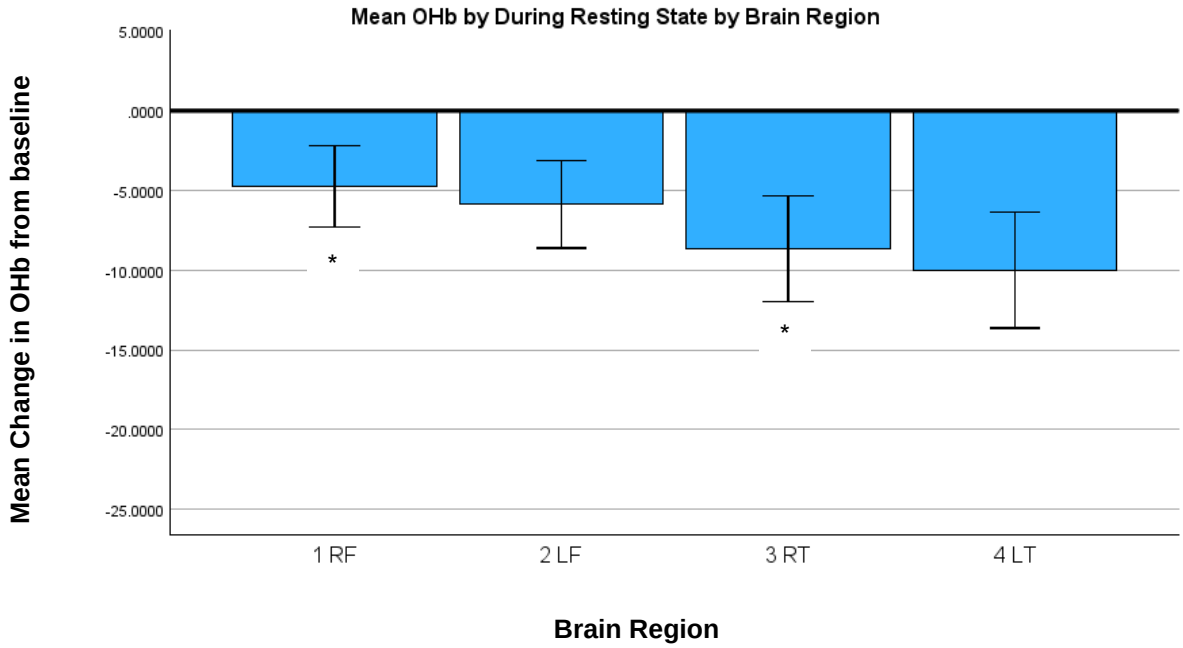


Figure 46
Mean Change in OHb from Resting State-Baseline by Brain Region
Note: OHb = Oxygenated Haemoglobin; RF_– Right Frontal; LF_ =Left Frontal; RT_ =Right Temporal; LT_ = Left Temporal

A post hoc comparison of regional differences found the right temporal region showed significantly more activity than the right frontal region (RT =-8.65, RF=-4.74; MD=-3.92, SE1.28, *p*=.017; Bonferroni adjustment for multiple comparisons). Based on previous research, this pattern of greater temporal lobe activity is more indicative of greater internal processing rather than task-based processing and successful measurement of DMN during the session. The difference between the left frontal

(LF) and left temporal (LT) was not significant (see Appendix P1 for the non-significant post hoc comparisons).

Results 4 Analyses:

Regional OHb & Continuous health Correlation	Continuous Physical health and LF region OHb Regression	Continuous Mental Health and RT-region OHb Regression	Continuous Repetitive Negative Thinking and RT-region OHb Regression
Categorical Health & Regional OHb ANOVA	Categorical Brooding Rumination and all-channel OHb		

Note: OHb = Haemodynamic Activity

Analytical Approach: Resting State Neurovascular Activity and Health

A range of methods were used to investigate haemodynamic activity and health. Firstly, a series of Pearson correlations were used to investigate the association between continuous health measures and regional haemodynamic activity (see Table 53). Secondly, regression analysis was used to find out which continuous health measures were significant predictors of regional activity. Thirdly, ANOVA were used to examine the relationship between regional haemodynamic activity by categorical health; dividing participants into binary high and low health groups (based on clinical cutoffs where available). Finally, investigation by individual channel were then conducted to determine more precisely where the significant health effects were located.

Resting State Regional Haemodynamic Activity Correlation with Continuous Physical Health.

Pearson correlation of regional haemodynamic activity and continuous physical health measures (see Table 53) indicated a significant correlation between BMI, WC and RS haemodynamic activity in the ***left frontal (LF) lobe***. The correlations were small and positive (BMI *r*=.23; WC *r*=.24). Examination of scattergrams indicate that those with higher BMI/WC tended toward greater haemodynamic activity in the left frontal region (see Figure 47 BMI and Figure 48 WC).

Table 53

Pearson Correlation Between Regional RS OHb and Continuous Physical Health Measurements

N=88		RF	LF	RT	LT
BMI (cm)	r	.13	.23*	.08	.05
	p	.216	.034	.437	.659
Mean WC (cm)	r	.12	.24*	.16	.14
	p	.250	.027	.142	.186

Note: 2-tailed significance; *=*p*<.05 ; OHb = Oxygenated Haemoglobin; RS= Resting State; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

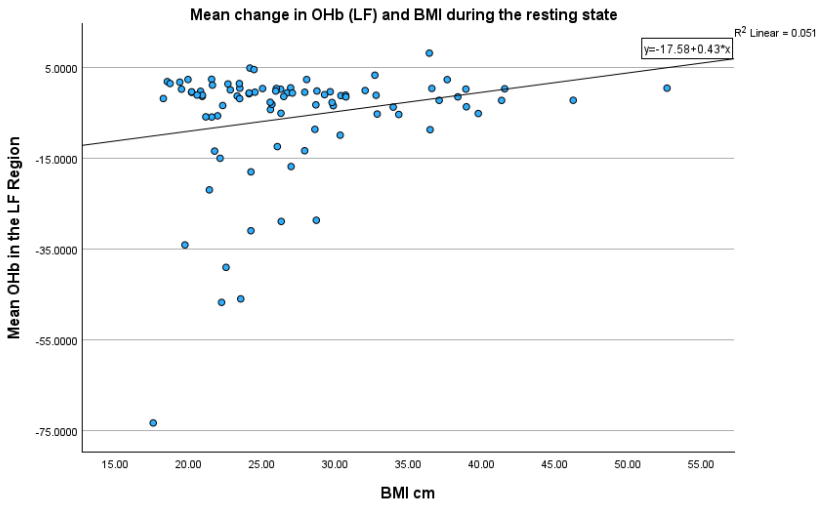


Figure 47

OHb Activity in the Left Frontal Region with Continuous BMI

Note: OHb = Oxygenated Haemoglobin; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

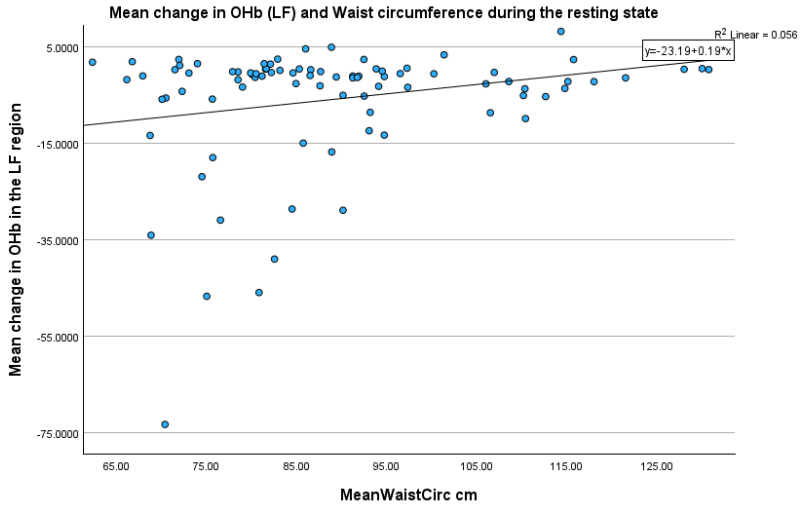


Figure 48
OHb Activity in the Left Frontal Region with Continuous WC
Note: OHb = Oxygenated Haemoglobin ;: RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

RS Regression of Physical Health on LF Haemodynamic Activity. As the left frontal lobe had shown significant regional differences based on adiposity, a backwards stepwise regression was used to examine the relative importance of WC and BMI in predicting LF OHb in the resting state (see Table 54 for predictors and model stages).

Table 54
Backward Stepwise Regression of Physical Health on Left Frontal OHb

Model Stage	Predictors	R^2	Adjusted R^2	R^2 Change	Sig. F Change
1	Mean WC cm, BMI	.06	.03	.06	.084
2	Mean WC cm	.06	.04	.00	.787

Note: OHb = Oxygenated Haemoglobin; WC= Waist Circumference; Sig= Significant

The initial model was non-significant (p=.084). In step 2 BMI was removed as it did not add significant variance. The final model with just WC was significant $F(1,86) = 5.09$, p=.027. Waist circumference had a small effect (R^2=.06) and explained 6% of the variance in OHb in the LF region (Standardised Beta=.24,

t=2.26, *p*=.027). A further regression was conducted including gender to see if it was a significant predictor of LH activity, but it did not add significantly to the model.

Resting State Regional Haemodynamic Activity Correlation with Continuous Mental Health.

Pearson correlation of regional Haemodynamic activity and continuous measures of depression and anxiety indicated a significant association between anxiety and haemodynamic activity in the right temporal lobe (*r*=-.24, *p*=.026; see Table 55). The correlation was small and negative. This indicates a trend for those who are high in anxiety to display lower overall haemodynamic activity in the right temporal lobe (compared to those with low anxiety) however this lower mean is indicative of a larger magnitude of change compared to baseline.

Table 55

Pearson Correlation Between Regional OHb and Continuous Mental Health Measurements

N=88		RF	LF	RT	LT
Depression Total	r	-.10	-.14	-.16	-.21
	p	.365	.185	.125	.054
Anxiety Total	r	-.14	-.07	-.24*	-.16
	p	.197	.528	.026	.149

Note: OHb = Oxygenated Haemoglobin; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

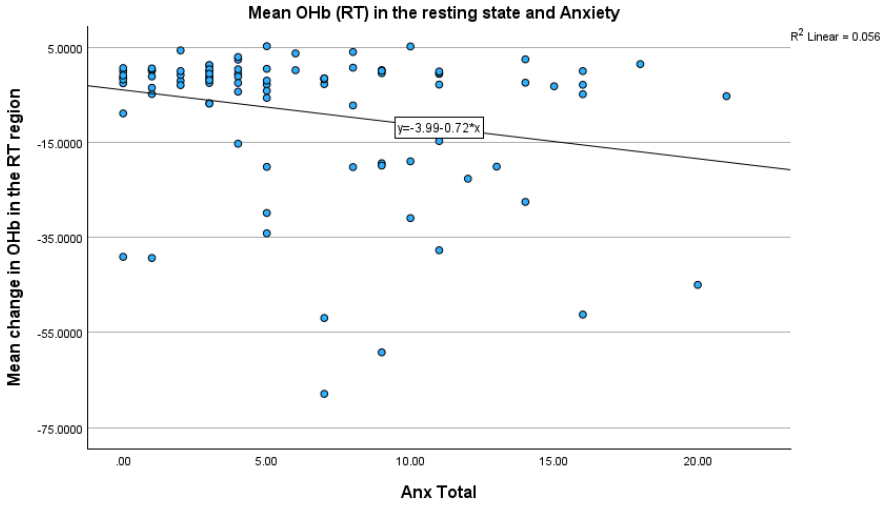


Figure 49

OHb Activity in the Right Temporal Region with Continuous Anxiety

Note: OHb = Oxygenated Haemoglobin; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

Resting State Regional Haemodynamic Activity (OHb) Correlation with Continuous Repetitive Negative Thinking.

Pearson correlation of regional haemodynamic activity (OHb) and continuous measures of rumination and worry indicated a significant association between worry and OHb in the right temporal lobe (*p*=.008; see Table 56). The correlation is larger than that of anxiety on RT haemodynamic activity, indicating that significant

association with anxiety is likely to be due to the components of repetitive negative thinking in the measure.

Table 56

Pearson Correlation Between Regional OHb and Continuous Repetitive Negative Thinking

N=88		RF	LF	RT	LT
Rumination Total	r	-.03	-.13	-.19	-.20
	p	.779	.214	.083	.056
Worry Total (PHQ Q2&3)	r	-.13	-.03	-.28**	-.18
	p	.228	.801	.008	.087

Note: OHb = Oxygenated Haemoglobin; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

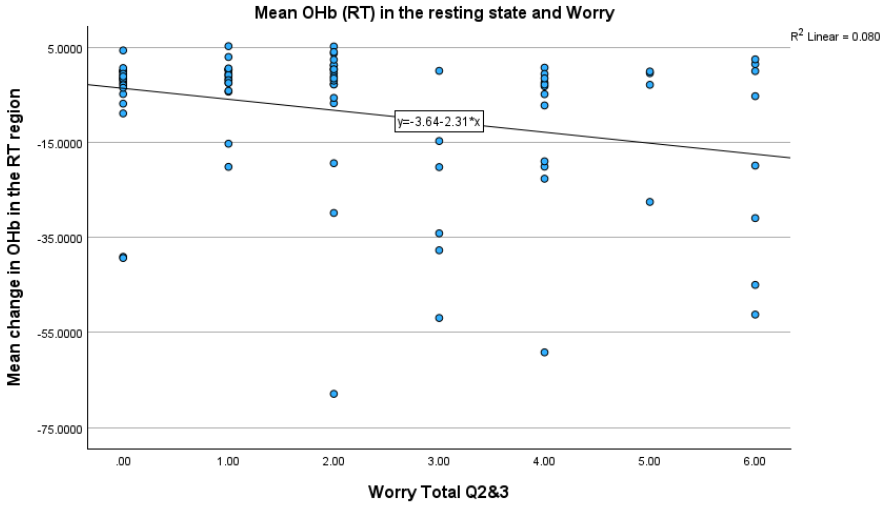


Figure 50

OHb Activity in the Right Temporal Region with Continuous Worry

Note: OHb = Oxygenated Haemoglobin; RF= Right Frontal; LF= Left Frontal; RT= Right Temporal ; LT= Left Temporal

RS Regression of Repetitive Negative Thinking on RT Haemodynamic Activity. To confirm which repetitive negative thinking continuous health measures were most important to haemodynamic activity in the RT region a backwards stepwise regression was performed (see Table 57 for predictors and R^2 change at

each stage of the model). Initially 3 variables were entered: Brooding Rumination (RUMb total score), Reflect Rumination (RUMb total score), and Worry (total score).

Table 57

Regression Model Stages Brooding Rumination (RUMb Total Score), Reflect Rumination (RUMr Total Score), and Worry (Total Score)

Model Stage	Predictors	R^2	Adjusted R^2	R^2 Change	Sig. F Change
1	Worry, RUMr, RUMb	.09	.05	.09	.055
2	Worry, RUMr	.08	.06	.00	.680
3	Worry	.08	.07	.00	.541

Note: RUMr- Reflective Rumination; RUMb=Brooding Rumination

The initial model was approaching significance (p=.055). Variables were removed that did not add significant variance (RUMr and RUMb). The final model consisted of worry and was significant: $F(1,85) = 7.48$, p=.008. The effect was small R^2 =.08 and explained 8% of the variance in haemodynamic activity (OHb change) in the RT region (worry standardised beta=-.28, t=-2.73, p=.008). A further model was created to examine the effect of gender as females are reported to have greater levels of worry and rumination (see Table 58).

Table 58

Regression Model Stages Brooding Rumination (Total Score), Reflect Rumination (Total Score), and Worry (Total Score) and Gender

Model Stage	Predictors	R^2	Adjusted R^2	R^2 Change	Sig. F Change
1	Gender, RUMb, RUMr, Worry	.13	.09	.13	.018
2	Gender, RUMb, Worry	.13	.10	.00	.719
3	Gender, Worry	.12	.10	-.01	.424

Note: RUMr- Reflective Rumination; RUMb=Brooding Rumination

The initial model was significant (p=.018). Variables were removed that did not add significant variance (RUMr and RUMb). The final model was significant: $F(1,84) = 6.04$, p=.004. The effect was medium sized R^2 =.12 and explained 12% of

the variance in OHb change in the RT region. Gender therefore explained a further 4% of the variance in haemodynamic activity in the RT (Standardised Beta=-.22, *t*=-2.08, *p*=.041).

Interim Summary: Resting State fNIRS and Continuous Health

1)Resting state brain activity in the ***LF region*** was positively associated with continuous measures of **waist circumference** (explained 6% variance). Those with higher waist circumference had a smaller magnitude of change from baseline in the left frontal region compared to those with smaller WC. This means that for those with higher waist circumference, cells in the LF region are less active than those with lower waist circumference.

2)Resting state brain activity in the ***RT region*** was negatively associated with **worry** (explained 8% variance in in OHb). Those with higher worry scores had greater magnitude of activity in the right temporal region. Further examination of the frontal and temporal channels is needed to identify which frontal brain network is being activated (see section 7.8.2 and 7.8.3 which examine functional connectivity and rumination).

Resting State Haemodynamic Activity and Categorical Health ANOVA

A series of 2x4 mixed factorial ANOVAs examined differences in haemodynamic activity in the resting state by region (LF, RF, LT, RT), and health (binary low/high groups). Findings for each health group are summarised in Table 59. Findings are explained below under the health subheadings.

Table 59

Mixed ANOVA Findings (Health Group and Brain Region in The Resting State)

IV **Health group** **Region**	**Main Effects**	**Interaction Effect**
BMI risk Region	F(1,86)=1.57, *p*=.214, Eta^2=.018 F(2, 208)=4.60, *p*=.007, Eta^2=.051	F(2, 208)=.73, *p*=.506, Eta^2=.008
Waist risk Region	F(1,86)=1.84, *p*=.179, Eta^2=.021 F(2, 208)=4.03, *p*=.013, Eta^2=.045	F(2, 208)=.32, *p*=.768, Eta^2=.004
Depression Region	F(1,86)=1.21, *p*=.275, Eta^2=.014 F(2, 208)=3.43, *p*=.026, Eta^2=.038	F(2, 208)=.11, *p*=.926, Eta^2=.001
Anxiety Region	F(1,86)=1.46, *p*=.230, Eta^2=.017 F(2, 208)=3.77, *p*=.018, Eta^2=.042	F(2, 208)=.29, *p*=.787, Eta^2=.003
Rumination Region	F(1,86)=4.47, *p*=.037, Eta^2=.049 F(2, 208)=4.39, *p*=.009, Eta^2=.049	F(2, 208)=2.28, *p*=.094, Eta^2=.026
RUMb Region	F(1,86)=9.27, *p*=.003, Eta^2=.097 F(2, 207)=4.92, *p*=.005, Eta^2=.054	F(2,207)=2.29, *p*=.093, Eta^2=.026
RUMr Region	F(1,86)=2.93, *p*=.090, Eta^2=.033 F(2,209)=4.32, *p*=.010, Eta^2=.048	F(2,209)=1.30, *p*=.276, Eta^2=.015
Worry Region	F(1,86)=.41, *p*=.524, Eta^2=.005 F(2,207)=3.94, *p*=.015, Eta^2=.044	F(2,207)=1.20, *p*=.309, Eta^2=.014

Note: Greenhouse-Geisser was used throughout (sphericity not assumed): RUMr- Reflective Rumination; RUMb=Brooding Rumination

Physical Health. There was no significant main effect of BMI risk on regional resting state OHb changes (see Table 59). Although there was a significant correlation between continuous WC and resting state OHb, there was no significant main effect of waist risk category (WCr; $F(1,86)=1.84$, $p=.179$, Eta^2=.021) and no significant interaction (F (3,208.47) =.320, $p=.811$, Eta^2= .004). This indicates no one region showed significant average differences in resting state brain activity based on whether their waist circumference was categorised as ‘normal’ compared to ‘increased. The trend (based on the mean values across each region) was for those with increased WCr to have less change from baseline i.e., less haemodynamic brain activity in the frontal regions, than those with a low/normal risk (see Figure 51 Mean Change in Resting State Baseline for Waist circumference, and Figure 52 BMI).

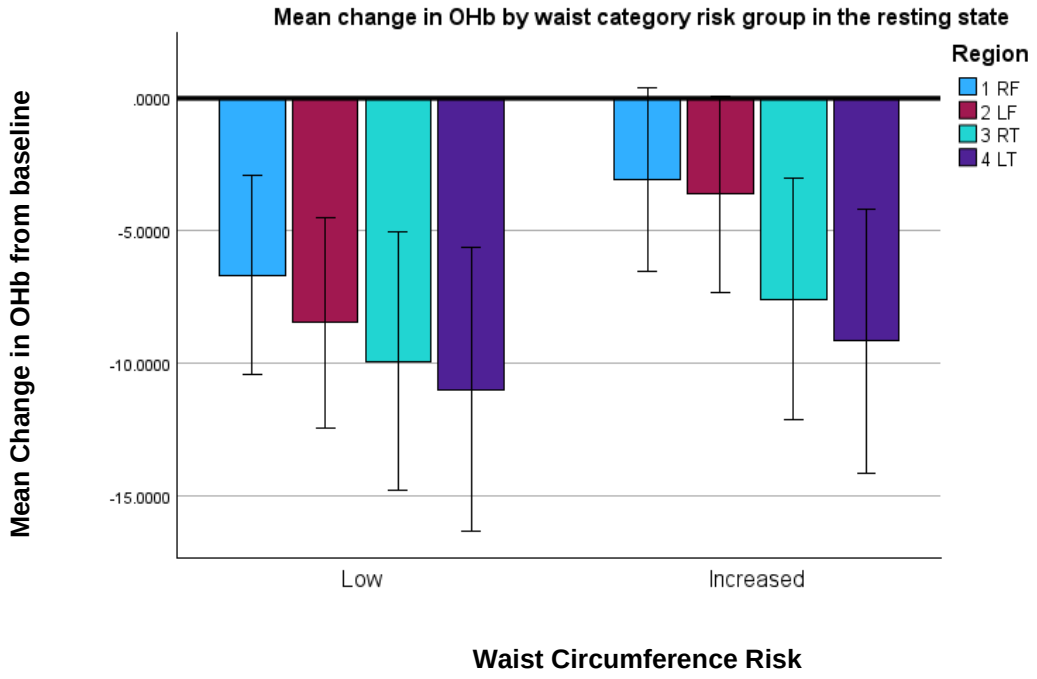


Figure 51
Mean Change in OHb from RS-baseline by Brain Region and WCr (Low/normal and Increased risk)
Note: OHb = Oxygenated Haemoglobin

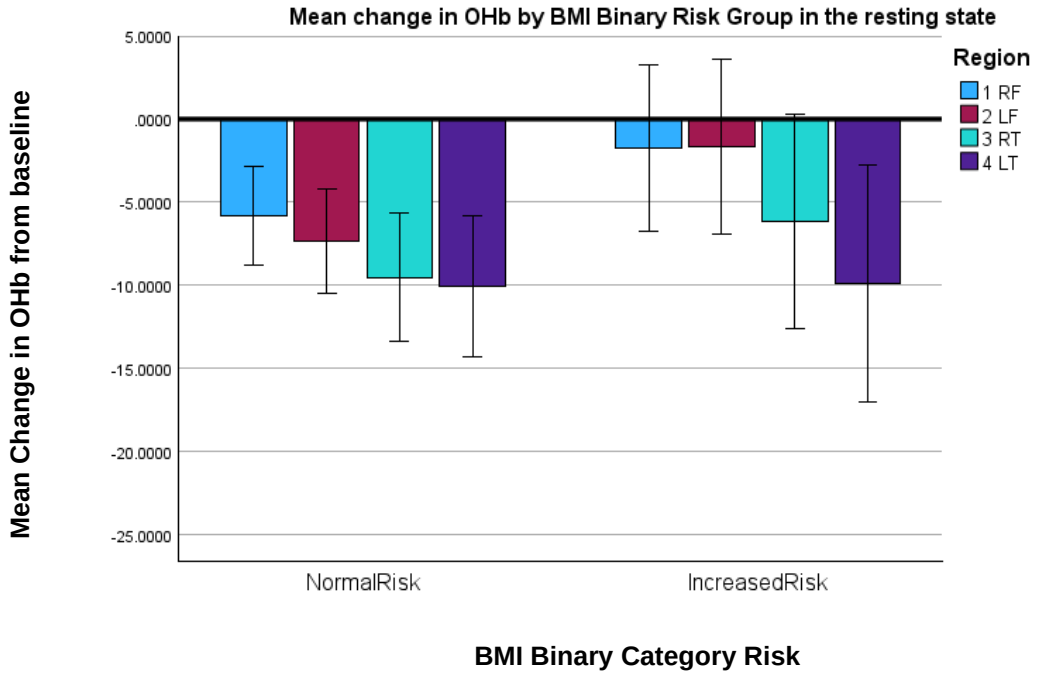


Figure 52
Mean Change in OHb from RS-Baseline by BMI Risk and Brain Region
Note: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal.

Mental Health and Repetitive Negative Thinking. There was a significant main effect of rumination group on OHb in the resting state (F (1,86) =4.47, p=.037, Eta^2=.049). Those in the high rumination group showed more activity in all brain regions compared to low ruminators (see Figure 53). Investigation of the rumination subscale scores (brooding and reflective rumination) indicate a highly significant effect of RUMb (F (1,86) =9.27, p=.003, Eta^2=.097) but no significant effect of RUMr (see Table 59) or worry (F (3,2.40) =1.20, p=.309, Eta^2=.014). These findings highlight that the activity of high RUMb group showed a different pattern of activity compared to the low/normal group, specifically that the frontal and medial temporal lobes of high brooding ruminators were more hemodynamically active in the resting state than low brooding ruminators. This finding appears in keeping with the characterisation of the brains of high ruminators' brains being more active at rest, possibly with a mixture of internal and external processing. There was no significant effect of clinical group for depression or anxiety on haemodynamic activity in the resting state, hence the results also inform discourse around qualitative differences of different types of repetitive negative thinking; brooding group clearly had more influence on haemodynamic activity than other mental health groups in the resting state.

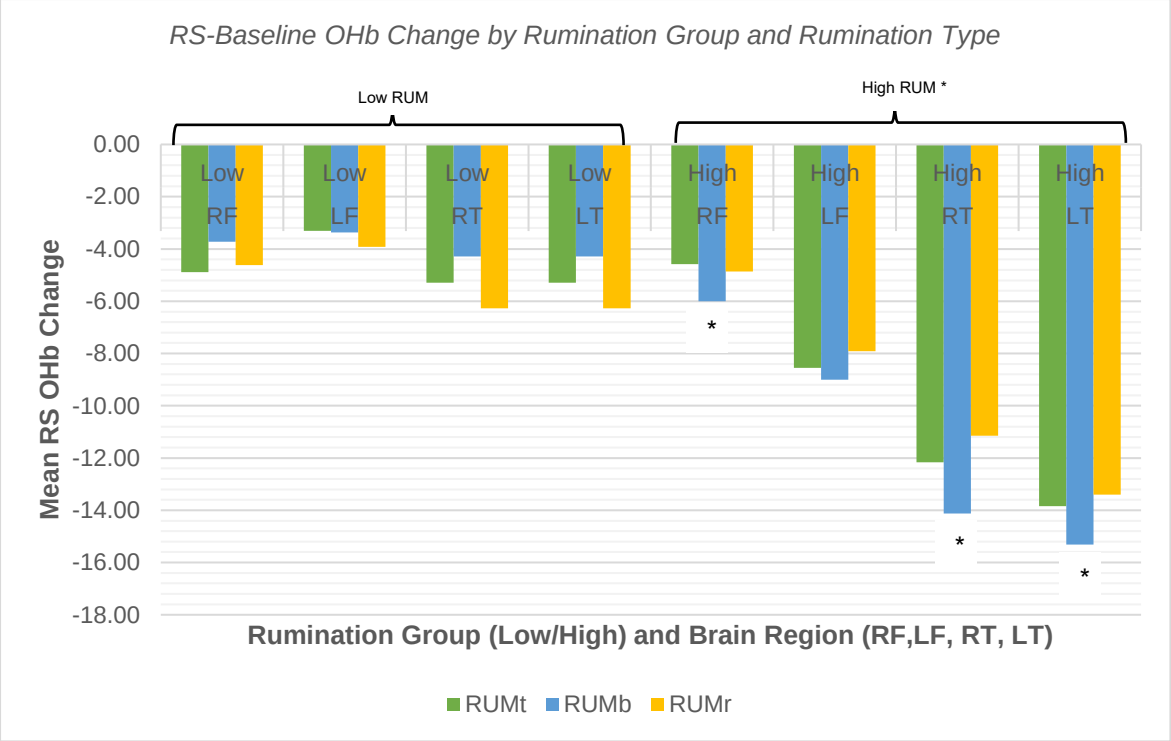


Figure 53
Mean Change in OHb from RS-Baseline for High and Low Rumination by Type (RUMt, RUMb, RUMr) and Brain Region
Note: RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal*; RUMt=Total Rumination*: RUMr- Reflective Rumination; RUMb=Brooding Rumination

Post hoc comparison of brooding main effects confirms the high RUMb group displayed greater haemodynamic activity in the resting state - more than double that of the low rumination group (High=-11.11, Low=-4.29; MD=-6.82, SE 2.24, *p*=.003).

Pairwise comparison for simple effects found no significant differences between regional haemodynamic activity for the low RUMb group (see Table 60). However, there were significant regional differences for the high rumination group. The right and left temporal regions were significantly more active than the frontal regions (LT was most active region, see Figure 53 above). The comparisons between right frontal lobe and left and right temporal lobe survived Bonferroni correction with the

difference between the RF and RT being highly significant (p<.001; see Table 60 Simple effects post hoc comparisons).

Table 60

Simple Effects Pairwise Post Hoc Comparisons of Haemodynamic Activity (OHb) by Brooding Rumination Group and Brain Region (Resting State).

	Region		Low Brooding			High Brooding			
	(a)	(b)	MD (a-b)	SE	p	MD (a-b)	SE	p	
Frontal	1 RF	2 LF	-.35	2.21	1.000	3.00	2.48	1.000	
		3 RT	.57	1.63	1.000	8.13	1.83	<.001	*
		4 LT	2.04	2.76	1.000	9.31	3.09	.021	*
	2 LF	1 RF	.35	2.21	1.000	-3.00	2.48	1.000	
		3 RT	.92	2.21	1.000	5.12	2.47	.247	
		4 LT	2.40	2.23	1.000	6.30	2.50	.082	
Temporal	3 RT	1 RF	-.57	1.63	1.000	-8.13	1.83	<.001	*
		2 LF	-.92	2.21	1.000	-5.12	2.47	.247	
		4 LT	1.48	2.19	1.000	1.18	2.45	1.000	
	4 LT	1 RF	-2.04	2.76	1.000	-9.31	3.09	.021	*
		2 LF	-2.40	2.23	1.000	-6.30	2.50	.082	
		3 RT	-1.48	2.19	1.000	-1.18	2.45	1.000	

Note: Bonferroni correction was applied to adjust for multiple comparisons. OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal.

Resting State Mean Haemodynamic Activity - All Channels

ANOVA was used to check for significant differences in mean haemoglobin (see Table 59). The left temporal region (medial temporal lobe) showed the most haemodynamic change. Channel LT1 and LT2 showed the most activity, whereas LT3 (superior and anterior to the other temporal channels) showed less than half their value indicating it was less active (see Table 59). There was a similar trend in the right temporal channels and RT3 was the least active of the three.

Brooding rumination -investigating individual brain channels. Examining the specific channels which were the source of the significant differences between high and low brooding ruminators (RUMb), post hoc pairwise comparison of

individual channels and RUMb group found significant differences in the temporal lobe channels 1 and 2 in the left and right hemispheres and left frontal channels 3 and 5 (see Table 60). Those high in RUMb therefore had significantly greater haemodynamic activity in the T3/T4 region, i.e.., the **superior** and **medial temporal gyrus** in both hemispheres. The effect was more pronounced in the right hemisphere. Compared to low, high brooding ruminators also showed more activity in the left frontal channel 5 which correspond to the **superior frontal gyrus** (I10:20 F3 region; DLPFC) and left frontal channel 3, i.e.., the medial frontal gyrus (See Figure 33 for a visual representation of the measurement channels and 10:20 regions of interest). The temporal lobe and medial frontal activity (LF3) indicate that the DMN is more active in those with high brooding rumination. The LF5 activity indicates that high RUMb also had more externally focused attention (e.g. examining the environment).

Interim Summary: Resting State fNIRS and Categorical Health

When investigating differences in mean OHb change by health group (low/high) RUMb showed a significant effect (see Table 61). While low ruminators had an even profile of low frontal and temporal activity at rest the high ruminators showed greater activity in their medial temporal lobe (right and left were significant, but the right showed a greater increase compared to the low brooders). Examination of individual channels confirmed the channels in the medial and superior temporal lobe were the main source of increased activity with some additional activity in the left medial frontal and left superior frontal areas. The medial temporal and medial frontal activity is characteristic of a more active DMN nodes in high ruminators at rest.

Table 61

Individual Channels with Significant Differences in Mean OHb for Low Compared to High RUMb

Rumination (Brooding)					**Frontal Lobe**					
Mean OHb			**Right Hemisphere Channels**					**Left Hemisphere Channels**		
Channel	**RF1**	**RF2**	**RF3**	**RF4**	**RF5**	**LF1**	**LF2**	**LF3**	**LF4**	**LF5**
Low RUMb								-4.71		-.11
High RUMb								-15.13		-5.45
MD								10.42		5.33
(SE)								(4.83)		(2.46)
Sig.								*p*=.034		*p*=.033

			Temporal Lobe			
Channel	**RT1**	**RT2**	**RT3**	**LT1**	**LT2**	**LT3**
Low RUMb	-6.32	-5.67		-7.88	-7.55	
High RUMb	-16.17	-16.90		-16.76	-18.05	
MD	12.67	14.61		12.02	12.21	
(SE)	*(4.10)*	*(4.47)*		(5.02)	(4.79)	
Sig.	*p*=.003	*p*=.002		*p*=.019	*p*=.013	

Note: Bonferroni Adjustment for multiple comparisons; OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; RUMb= Brooding Rumination

Results4 Summary

Regression analysis on mean regional activity found WC was a significant predictor of left frontal (LF) activity; those high in WC displayed less magnitude of haemodynamic activity indicating neurones were less active in those areas. Anxiety was a significant predictor of right temporal (RT) activity; those high in anxiety displaying greater magnitude of haemodynamic activity (i.e. neurones were more active in those regions).

ANOVA examining regional haemodynamic activity by high and low health groups found significant main effects of rumination and brooding rumination on regional haemodynamic activity, but no significant effects of physical and mental health groups. The main effect of region was significant in all comparisons, but interaction effects between region and health were not significant. The high brooding rumination group showed significantly greater activity in the temporal lobe region and subsequent investigation by individual channel found increased activity in the bilateral medial and superior temporal lobes (greater in the right).

Anxiety and brooding rumination therefore appear to be associated with increased brain activity in the medial temporal lobes during rest, which is commensurate with greater Default Mode Network activity.

Results 5: fNIRS During Performance Tasks

Prediction 5

a)Significant differences in haemodynamic activity in the prefrontal cortex based on obesity (low/high).

b)Significant difference in haemodynamic activity in the prefrontal cortex based on negative affect (low/high).

Descriptive Statistics: Regional Haemodynamic Activity During Performance Tasks

Mean haemodynamic activity during the attention (CT) and inhibition task (ST) showed a much smaller degree of change compared to the resting state. Standard deviations (SDs) reveal large individual differences in frontal lobe activity during the attention task (see below Table 62 Mean OHb and HHb during the performance tasks). Figure 54a&b show the regional differences in haemodynamic activity graphically. During the attention task (CT) there appeared to be a slightly higher magnitude of OHb change in the right hemisphere RF, RT) while the LF appeared least active. In contrast during the inhibition task (ST) the frontal regions appeared to have the largest magnitude of OHb change indicating that most activity was occurring in task-focused brain regions as expected (the RT appeared least active). These result help to give a benchmark to interpret the health-related differences observed in the tasks (which also represent easy and more challenging conditions).

Table 62

Mean Change from Baseline in Haemoglobin Concentration During the Performance Tasks (CT Attention and ST Inhibition)

fNIRS	CT Attention Task (N=88)								ST Inhibition Task (N=89)							
	Frontal Lobe				Medial Temporal Lobe				Frontal Lobe				Medial Temporal Lobe			
	Right		Left		Right		Left		Right		Left		Right		Left	
	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb	Oxy Hb	Deoxy Hb
Min.	-23.34	-49.86	-6.42	-8.33	-9.56	-5.62	-9.89	-2.59	-9.56	-5.62	-9.89	-2.59	-6.81	-6.49	-19.90	-19.82
Max.	15.59	6.40	24.59	7.98	7.70	3.75	7.92	5.48	7.70	3.75	7.92	5.48	5.52	9.97	6.22	4.37
Mean	-1.32	-.14	-.50	.18	-.66	.81	-.58	.93	-.66	.81	-.58	.93	-.12	.90	-.20	.37
SD	4.44	6.78	3.73	3.07	2.95	1.44	3.11	1.33	2.95	1.44	3.11	1.33	1.92	2.06	2.77	2.57
Skew	-1.30	-5.57	3.76	-.30	.43	-1.04	-.13	.11	.43	-1.04	-.13	.11	-.31	.87	-3.97	-5.53
Kurtosis	9.93	36.09	23.18	.66	1.56	3.47	1.46	1.63	1.56	3.47	1.46	1.63	2.67	6.27	29.03	43.82

Note: Skew SE = .26, Kurtosis SE=.51; OXY Hb= Oxygenated Haemoglobin; Deoxygenated Haemoglobin

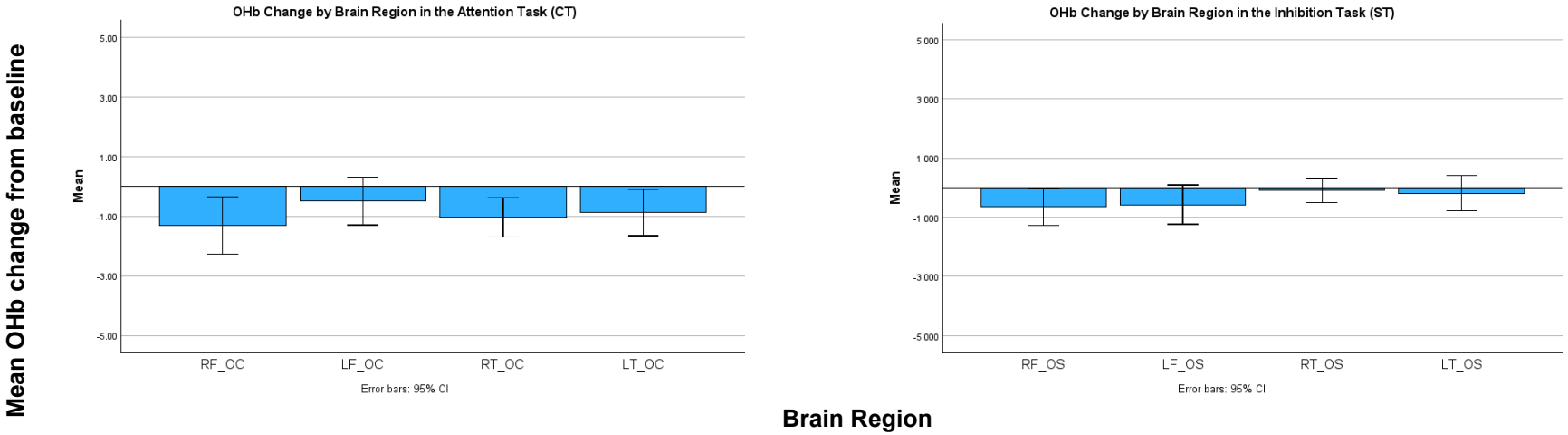


Figure 54 a&b

Mean Change in Haemoglobin Concentration from Baseline During the Performance Tasks (CT Attention and ST Inhibition)

Note: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal.

Results 5 Analysis:

Regional OHb during Performance Tasks (ANOVA)	Regional OHb and Health Category (ANOVA)		

Note: OHb = Oxygenated Haemoglobin

Analytic Approach. One-way repeated measures ANOVAs were conducted to examine the general pattern of regional changes in haemodynamic activity while participants were completing the attention and inhibition tasks. Mixed factorial ANOVAs were conducted to see if each task had significant regional differences based on health measures that had shown significant differences in previous analyses (waist circumference and rumination). These used the binary health groups.

Performance TASK Regional Haemodynamic Differences for each task.

The repeated measures ANOVA examining regional differences during cognitive tasks found no significant effect of haemodynamic activity by region for either attention/CT (F (2.33, 201.01) =1.19, p= .31, Eta^2=.014) or Inhibition/ST (F (2.73,237.70) =1.71, p=.171, Eta^2=.019; see Figure 54a&b). The null finding indicates there was not big difference between the two tasks in which areas of the brain were activated at a regional level (i.e. we would need to look at the haemodynamic differences in more detail to understand whether brain activity differed between the tasks).

Performance TASK Haemodynamic Activity Regional Differences and Health.

A series of 4x2 mixed factorial ANOVA were conducted to see if there were differences in OHb regionally (RF, LF, RT, LT) depending on ***health group*** (high/low) during the attention task. This was repeated for the inhibition task. As previously, the binary low/normal and high/ health groups were based on clinically significant health cutoffs where available. Findings for each task are detailed below.

Attention Task (CT). During the CT task there was a significant main effect of ***WCr*** group on OHb such that those with increased WCr had greater magnitude of activity than low/normal WCr (Attention/CT; $F(1,85) = 5.96$, $p = .017$). There was no significant effect of region ($F(2,198) = 1.08$ $p = .351$), and no significant interaction effect ($F(2,198) = 1.05$ $p = .359$). No other physical or mental health groups showed significant effects on haemodynamic activity during the attention task – see Appendix P2 for the non-significant comparisons.

Post hoc comparisons (see Appendix P2 and Figure 55 Mean Change in OHb by WCr) found that those with a high WCr compared to low displayed significantly greater change in haemodynamic activity in the right frontal lobe (MD=2.37, SE .93, $p = .013$) with a similar trend in the right temporal region (MD=1.29, SE=.66, $p = .053$: approaching significance; see Appendix P2 for the non-significant comparisons)

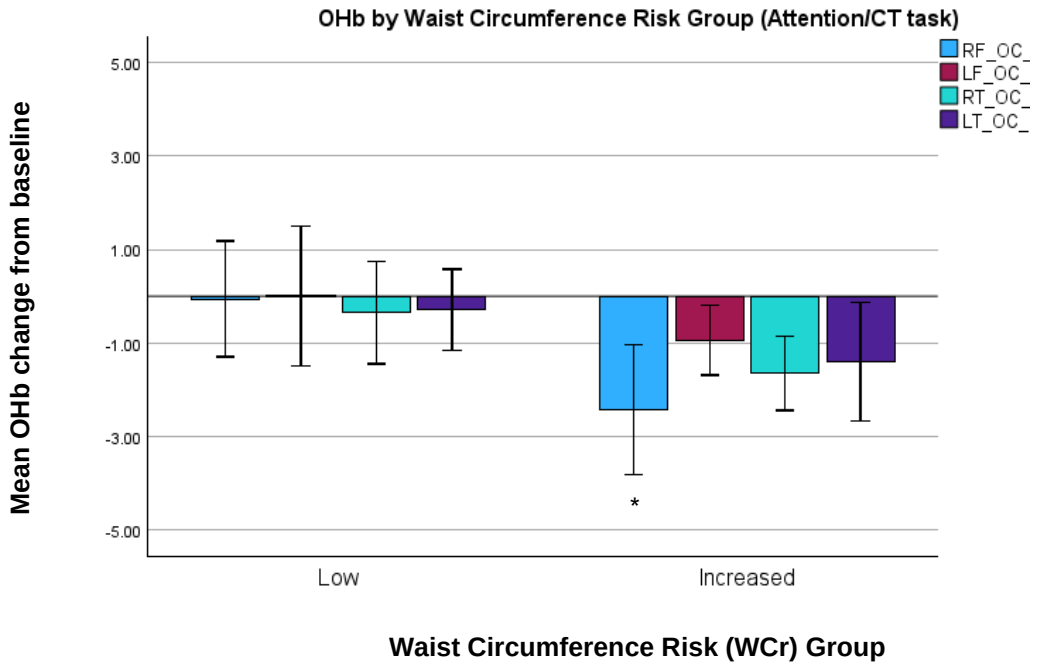


Figure 55
Mean Change in OHb by WCr Group During the Attention Task (CT).
Note: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal.

Inhibition TASK (ST). Mixed factorial ANOVAs examined haemodynamic activity during the inhibition task by health group (see Appendix P3 for the SPSS/non-significant findings). There was a significant main effect of ***RUMr*** (F (1,86) =4.06, p=.047, Eta^2=.045) such that the low RUMr group displayed a larger magnitude of activity and the high RUMr group displayed a smaller magnitude of activity, see Figure 56). There was a significant interaction between region and RUMr (F (2, 234) =2.88, p=.042, Eta^2=.032; sphericity not assumed) but no significant main effect of region F (2, 234) =1.71, p=.170, Eta^2=.020. No other physical or mental health groups showed significant effects on OHb during the attention task – see Appendix P3 for the non-significant comparisons.

Post hoc pairwise comparisons of RUMr confirmed haemodynamic activity in the LF and RT was significantly smaller in magnitude in the **high** reflection group compared to the **low** reflection group during this task (LF: MD=1.74, SE=.65, p=.008;

RT: MD=1.14, SE=.39, *p*=.005, see Appendix P3). This indicates that the high reflection is associated with less activity in the **left frontal** and **right temporal region**. Analysis by channel was needed to confirm which brain networks are likely to be affected hence this was investigated in the next section of results: functional connectivity.

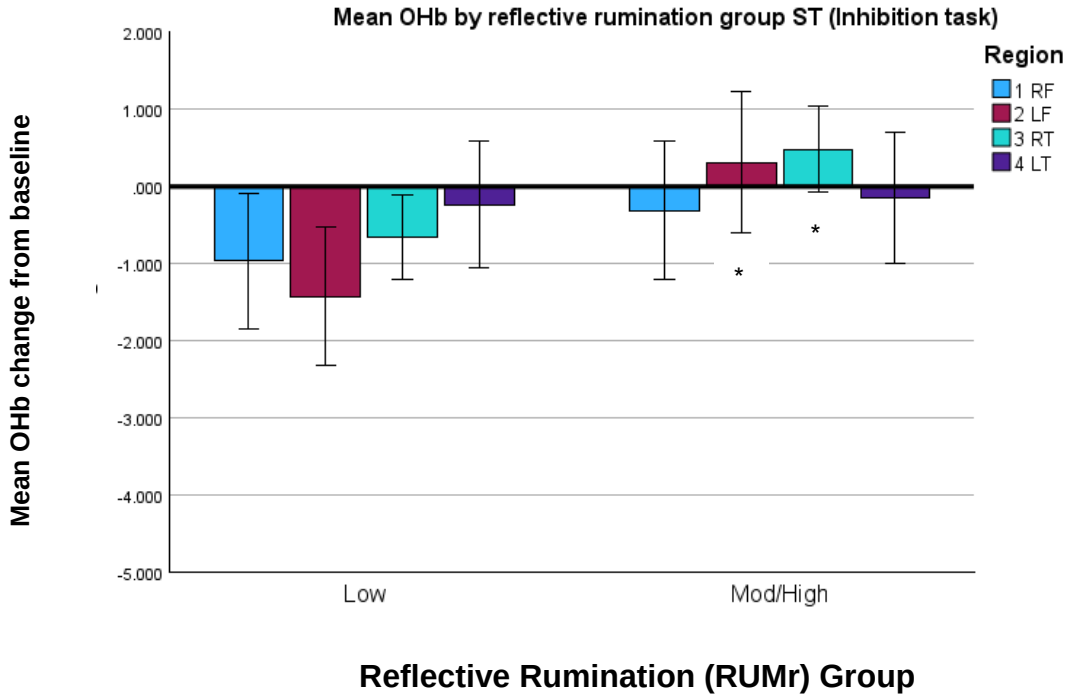


Figure 56
Mean Change in OHb by Reflective Rumination Group During the Inhibition (ST) Task.
Note: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal.

Results5 Summary

Comparing haemodynamic activity for each task, regional analysis was too general to see significant differences between which regions were active. There were however significant differences in regional haemodynamic activity by health group. In the attention task (CT) those with increased waist circumference risk (WCr) displayed more haemodynamic activity than those with low/normal WCr.

During the inhibition task (ST) those with high reflective rumination (RUMr) displayed less haemodynamic activity (left frontal and right temporal) than the low RUMr group.

Results 6 fNIRS Functional Connectivity (FC)

FC Waist Circumference Resting State CT Task & ST Task	FC Brooding Rumination Resting State CT Task & ST Task	FC Reflective Rumination CT Task & ST Task

Note: RS= Resting State FC= Functional Connectivity; CT= Continuous Performance Task; ST= Simon Task

Analytical Approach. Pearson correlations between channels were used to create functional connectivity matrices (Chao et al., 2021; Mohanty et al., 2020) to examine the effects of the physical and mental health groups (WCr and RUM) that had shown significant differences in haemodynamic activity in more detail. Functional connectivity matrix diagrams show correlation or synchronisation between channels. The aim was to examine which brain channels (and therefore likely brain networks) appeared to be working together, and whether there were differences based on health group. (See Appendix P4 for full correlation matrices). In the matrix diagrams darker green indicates haemodynamic activity (OHb) was highly similar (larger r) i.e., greater synchronisation or likelihood that those areas are working in tandem. Correlations where *p* was greater than .015 were removed from the matrices to correct for multiple comparisons.

Functional Connectivity and Waist Circumference Risk (WCr)

Resting State - WCr (see Figure 57). In the resting state, the low WCr group showed stronger correlation within regions (RF to RF and LF to LF) and more lateralised synchronisation – (moderate to strong RF to RT synchronisation and LF to LT synchronisation).

The high WCr group showed weaker and less extensive functional connectivity between RF channels. There was moderate connectivity between the LF and RF channels which was absent from the low WCr group. There was some strong functional connectivity laterally between the RF and RT (channel RF2&3 with RT 1&2) but little functional connectivity on the left (between LT and LF).

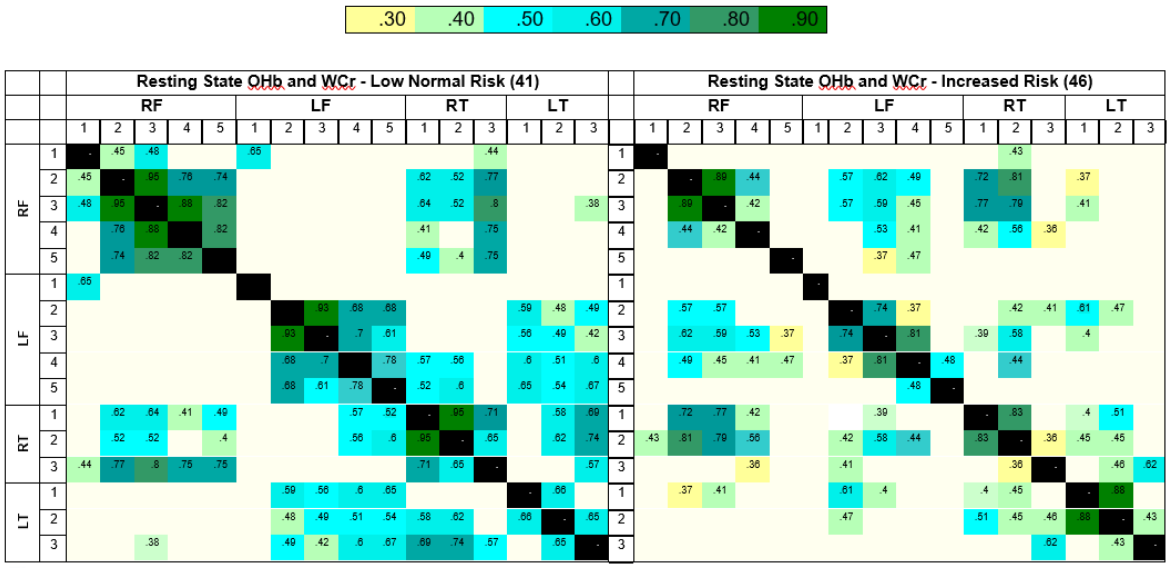


Figure 57
Functional Connectivity of Low and High WCr During the Resting State

CT Attention Task- WCr (see Figure 58). In the ***attention task*** the low WCr showed strong functional connectivity within the LF channels as (per anticipated on-task behaviour) and strong correlation with selected RF and TL channels (including left and right TL3 i.e. T3/4 medial temporal lobe) compared with the high WCr group.

Participants in the high WCr group showed less synchronised brain activity - fewer correlations and lower strength functional connectivity compared to the low WCr group. The RF region functional connectivity was limited but appeared stronger between channel 3-4. There were discreet high correlations within the left inferior PFC (LF2&3), and within the posterior medial temporal lobe (LT1&2).

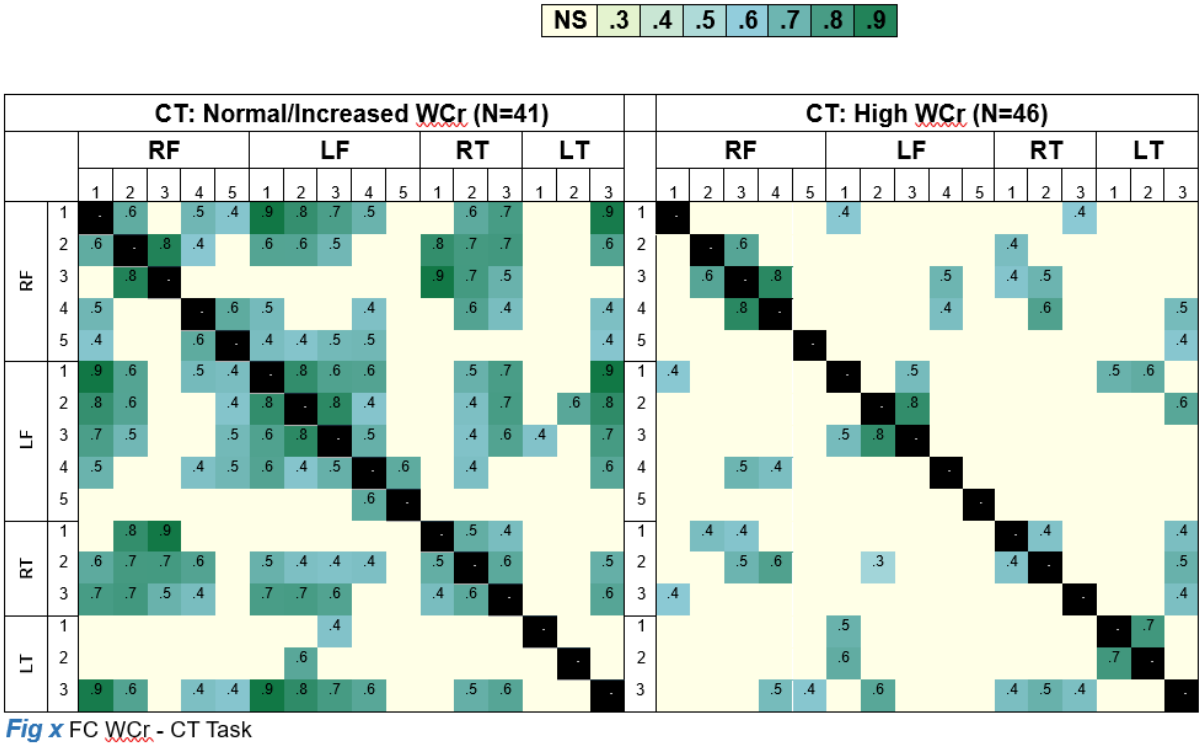


Figure 58
Functional Connectivity of Low and High WCr During the Attention Task (CT)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; CT= Continuous Performance Task

ST Inhibition Task - WCr (see Figure 59). A contrasting pattern was shown in the ***inhibition task.*** Those with high WCr showed more widespread functional connectivity compared to the low WCr group where synchronisation was more sparse and not as strong. The low WCr group synchronisation of the LF was dominant (within region LF1-3 i.e. inferior/VLPFC; between region LF4 i.e. medial PFC to RF, and LF to LT2) with some RF connectivity (within RF3; between RF5 i.e. IF4/ Superior or VLPFC). The low WCr RF did not show any cross lobe/hemisphere synchronisation with LT.

The high WCr group had strong correlations within the RF, LF, and LT (LT1&2 very strong), plus strong-moderate functional connectivity bilaterally across the FL (including RF to LF1-3) and moderate functional connectivity between lobes. The extensive moderate bilateral functional connectivity across the frontal lobes indicates these areas were working together (garnering more processing power to complete the task).

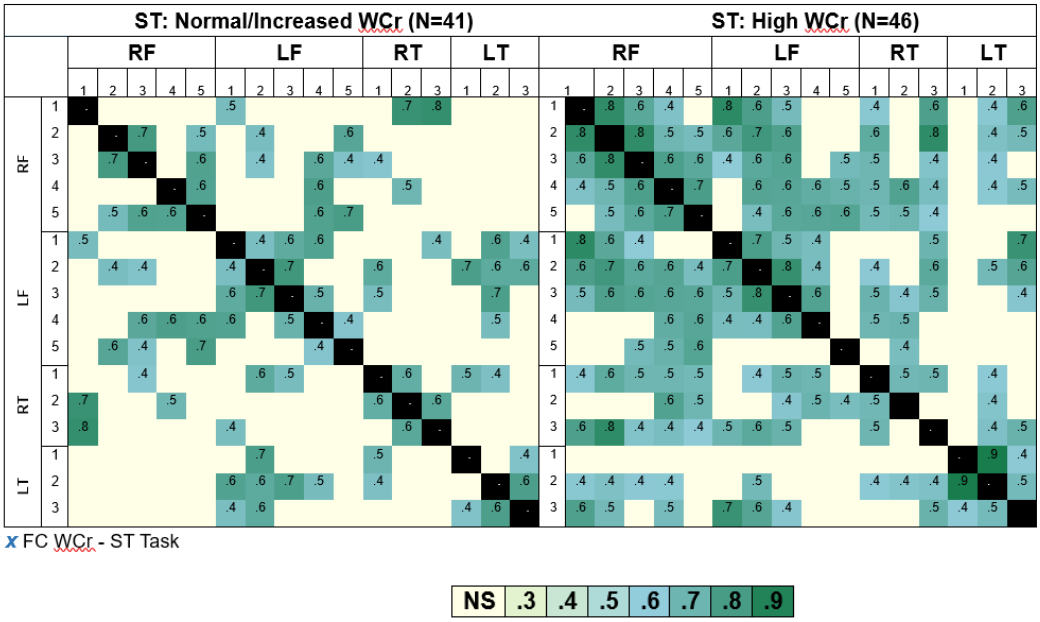


Fig x FC WCr - ST Task

Figure 59
Functional Connectivity of Low and High WCr During the Inhibition Task (ST)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

Functional Connectivity and Brooding Rumination

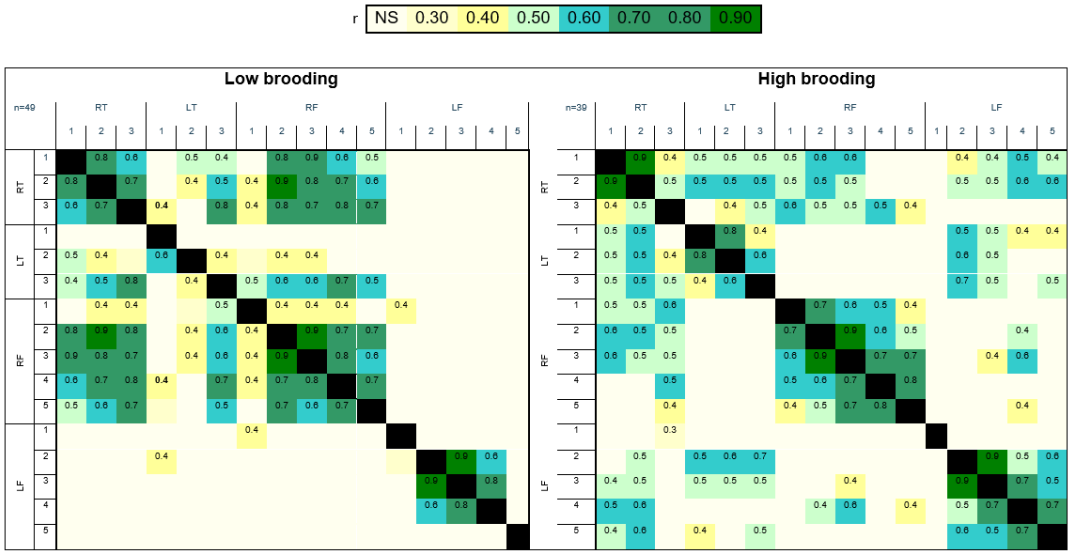


Figure 60

Brooding Rumination (Low/High) Functional Connectivity During the Resting State

Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

Resting State (see Figure 60). In the resting state the high brooding rumination (RUMb) group showed more widespread moderate functional connectivity between lobes and across hemispheres – for example the LF is correlated with both the temporal regions for high RUMb, whereas in the low RUMb group the functional connectivity in the LF is largely independent of the other regions. This pattern could reflect that those with high RUMb have a reduced ability to down-regulate the influence of the left frontal lobe on temporal regions during rest. The low RUMb group showed stronger synchronisation within the right hemisphere (between RT and RF) whereas this is weaker in the high RUMb group.

Performance Task Brooding Rumination. In summary, both the attention (CT) and inhibition task (ST), participants in the high RUMb group showed markedly less functional connectivity activity compared to the low RUMb group. Activity was much more localised with little synchronisation within and between lobes. ***Note***: During the performance tasks there were no significant difference in errors between the RUMb groups, but as it has been significant in most other comparisons it was decided to examine patterns of functional connectivity for both RUMb and RUMr.

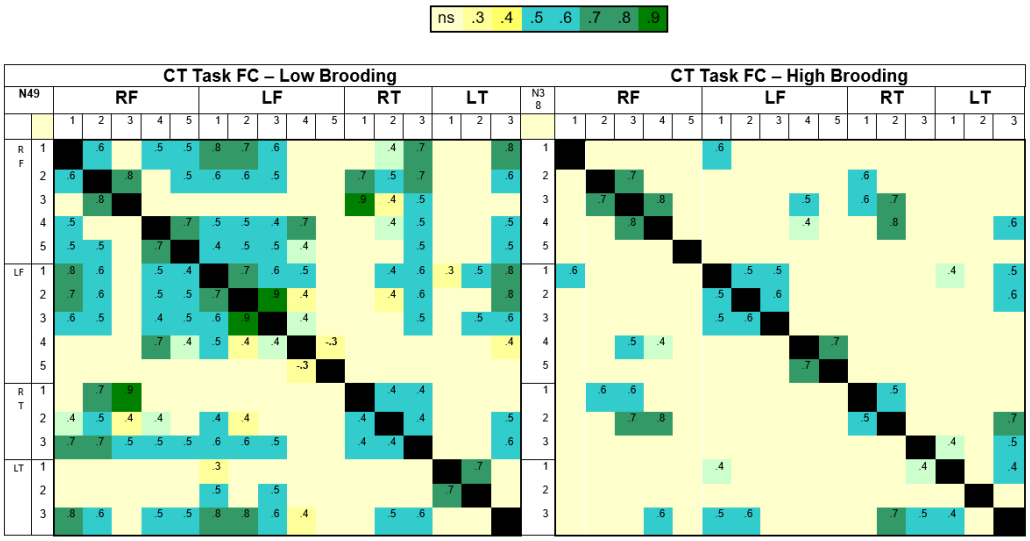


Figure 61
Functional Connectivity of Low and High RUMb During the Attention Task (CT)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

CT Attention Task -RUMb (see Figure 61). In the ***attention task***, high brooders showed weaker (moderate) correlation in the LF channels. Selected RF channels appeared to have more functional connectivity although medial PFC (LF4 &5) were also strong – this is different to the pattern for most groups where normally the inferior VLPFC (LF1-3) is more strongly synchronised. ***Inhibition*** Low brooders showed stronger synchronisation LF region and more widespread frontal connectivity in general (within channels LF1-3, and between this area and RF1-2, 4, 5). There

was also selective moderate synchronisation between the frontal lobe and the third temporal lobe channels (RT3, LT3 i.e., the IT3/4 medial temporal lobe) which was largely not evident in high brooders.

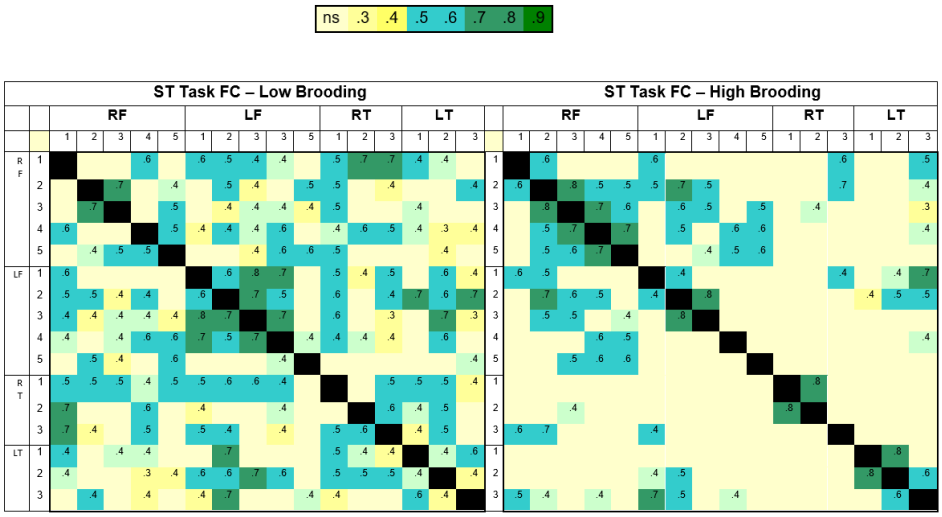


Figure 62
Functional Connectivity of Low and High RUMb During the Inhibition Task (ST)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

ST Inhibition Task- RUMb (see Figure 62). In the ***Inhibition task*** the high brooders had multiple strong connections in the RF and there were discrete strong connections within the other regions (LF2&3, RT 1&2, LT 1&2) . Low brooders again had strongest connectivity within several LF channels, and strong functional connectivity between RF2&3 (inferior/VLPFC) combined with more diffuse moderate activity across the frontal lobe and selected temporal channels.

Functional Connectivity and Reflective Rumination

Performance Task Reflective Rumination (RUMr)

The RUMr groups showed significant differences in regional OHb concentrations during the performance tasks. These findings are reflected in the considerable difference in regional activity observed in the inhibition task functional connectivity matrix., participants in the high RUMr group showed markedly less synchronised activity in both tasks, like the pattern shown in the RUMb performance task matrices.

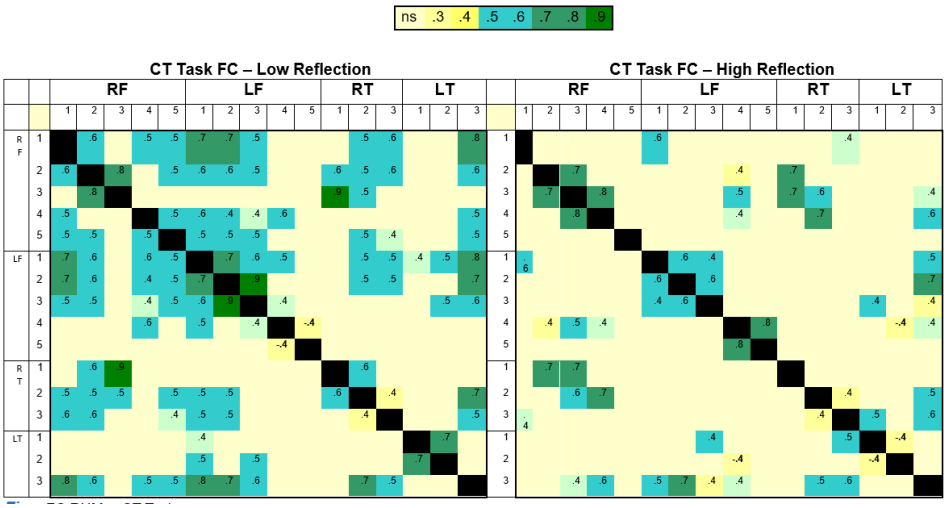


Figure 63
Functional Connectivity of Low and High RUMr During the Attention Task (CT)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

CT Attention Task – RUMr (see Figure 63). In the ***attention task*** the low RUMr group functional connectivity activity was focused within the LF channels (LF1-3; Inferior/VLPFC) with moderate correlations between the LF and RF. There was relatively strong functional connectivity within LT1&2 (posterior medial temporal lobe). The high RUMr group had highest connectivity in the RF but frontal channels appeared less synchronised with one another compared to the low RUMr group. This indicates the frontal areas were working independently rather than in unison.

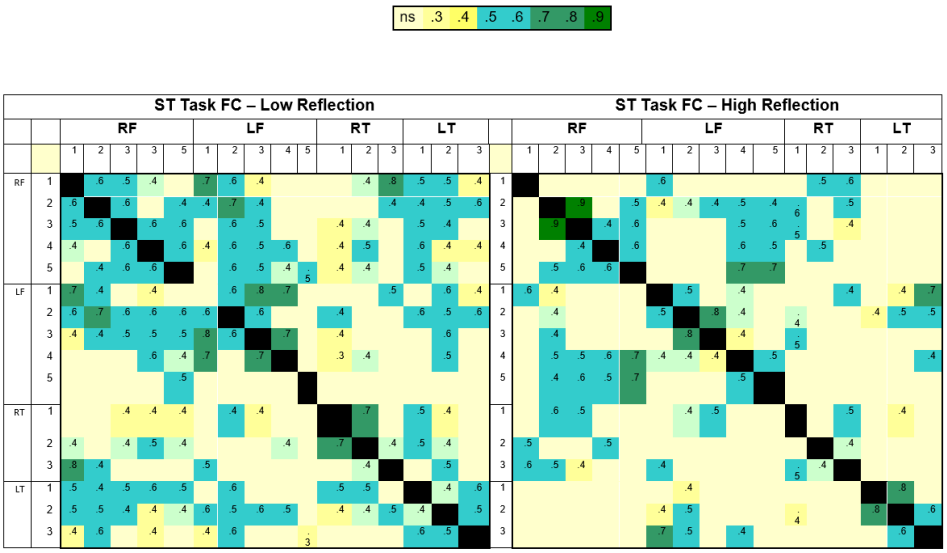


Figure 64
Functional Connectivity of Low and High RUMr During the Inhibition Task (ST)
Notes: OHb = Oxygenated Haemoglobin; RF=Right Frontal; LF= Left Frontal; RT= Right Temporal; LT= Left Temporal; WC*r*= Waist Circumference Risk; ST= Simon Task; CT= Continuous Performance Task

ST Inhibition – RUMr (see Figure 64) In the ***inhibition task*** participants in the low rumination group showed widespread moderately correlated activity across the frontal regions (left and right) and between the frontal regions and the LT. The RT had lower functional connectivity with other regions compared to the LT, but high correlation within the RT region (channel RT1&2).

The high RUMr group showed strongest functional connectivity in the RF but this was localised to the inferior and medial PFC channels (RF2&3)- not widespread. There was selective moderate synchronisation between the RF and the LF channels LF4-5 (i.e. medial and superior PFC) and high functional connectivity betweenRF5 and LF 4-5. There was little functional connectivity between and across the temporal lobes, although within the LT, channel 1&2 functional connectivity was strong (posterior medial temporal).

Summary Functional Connectivity and Health

Table 63 summarises the trends of functional connectivity identified from the matrices for waist circumference and rumination.

Table 63

Summary of the Main Differences in Functional Connectivity (FC) for Participants High in Waist Circumference Risk (WCr) and Rumination (RUMb/RUMr)

Functional Connectivity	High WCr Findings (compared to low)	High Rumination Findings (compared to low)
Resting State	High WCr weaker and less extensive FC	High RUMb. LF has moderate FC with RT and LT (absent from low group). Weaker lateral FC on the right.
Attention (CT)	High WCr weaker and less extensive FC. High LF-LT on selected channels.	Weaker and less extensive FC (very little). Low group have bilateral FC in frontal lobe.
Inhibition (ST)	High WCr: More extensive FC. Extensive moderate bilateral FC in FL. High LT FC (absent from Low group).	Weaker and less extensive FC. Higher FC within the RF (low group have higher FC in the LF plus widespread moderate FC).

Summary of Study 2 Results (1-6)

Results1 Summary Mental and Physical Health

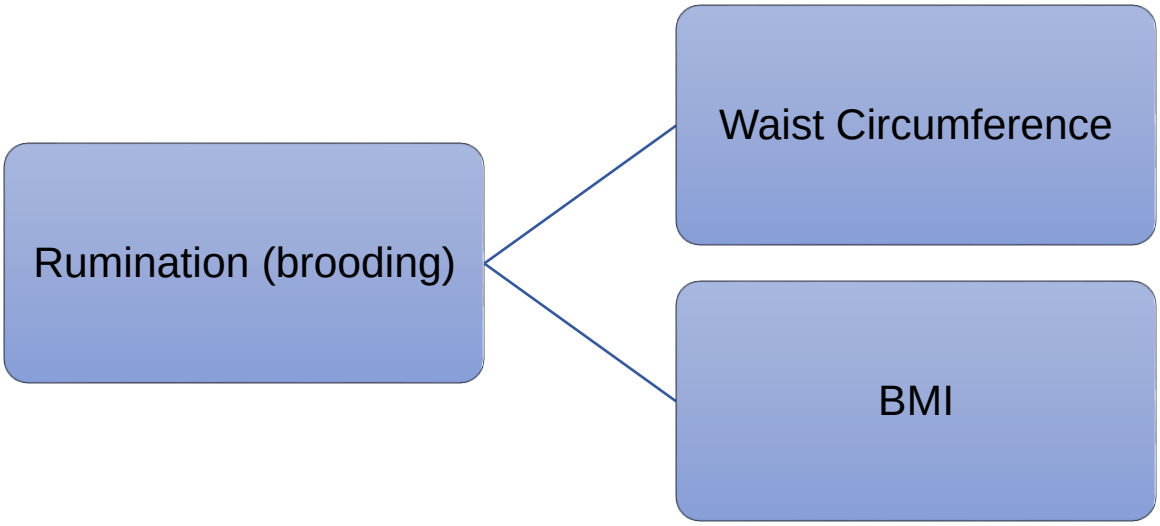


Figure 65
Associations between Mental Health and Adiposity (Continuous Measures)

Physical and Mental Health Correlation. Waist circumference (WC) and BMI were positively correlated with brooding rumination (RUMb, r=.22, p=.043; r=.22, p=.041).

Physical and Mental Health Regression. Brooding Rumination (RUMb) explained 5% variance in BMI and 5% in WC (Depression was not significant). An additional 4% variance in WC was due to gender. WC explained 5% variance in RUMb (BMI not significant).

Results2 Summary Cognitive Performance and Health

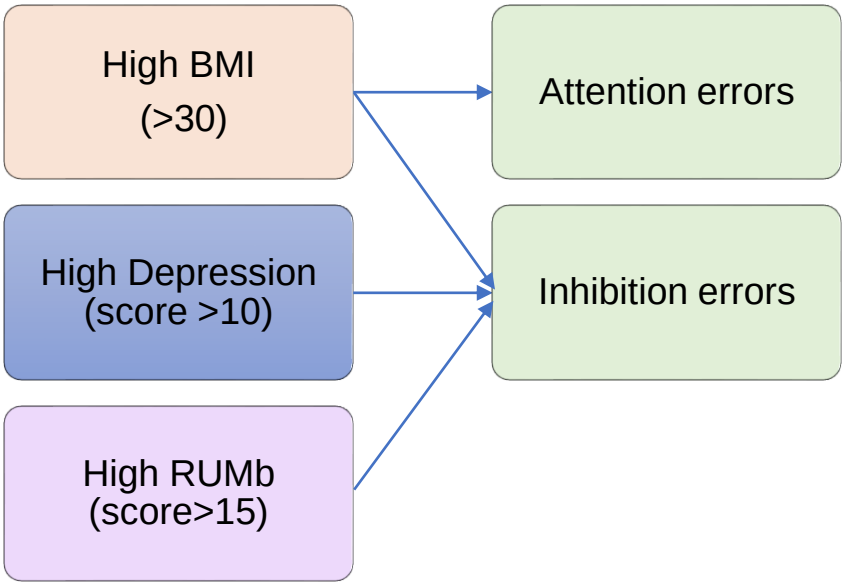


Figure 66
Findings Executive Function Performance and Health (high/low categorical)
Note: RUMb= Brooding Rumination

Paired T-Test (Task Difficulty). The ST task was more cognitively challenging than CT (longer reaction time and more errors, p<.001, see Figure 66 Findings Executive Function Performance and Health (high/low categories).

Independent T-Test (Health). High BMI significantly more attention (CT) errors (p=.037) and inhibition (ST) errors (p=.006); High depression significantly more inhibition (ST) errors (p=.011); High rumination significantly more inhibition (ST) errors (brooding p=.006; see Figure 66 Findings Executive Function Performance and Health (high/low categorical).

Results3 Summary Executive Function in Daily Living, Health, and Cognitive Performance

Correlation. Health and executive function in daily living. ***Adiposity*** was associated with the Behaviour Regulation Index (BMI=.30; WC=.32), strongest correlations were with self-monitoring, followed by emotional control and inhibition

but there was no significant association with shifting. ***Mental health/ repetitive negative thinking*** had moderate associations with both the Behaviour Regulation Index (*r*=.51 to .65; highest with brooding rumination) and the metacognitive Index (*r*=.36 to.59; highest with depression).

The BRIEF scales (self-reported executive function problems) with the highest correlations to health are shown in Figure 67 Highest Correlations Between Self-Reported Executive Function Problems and Health).

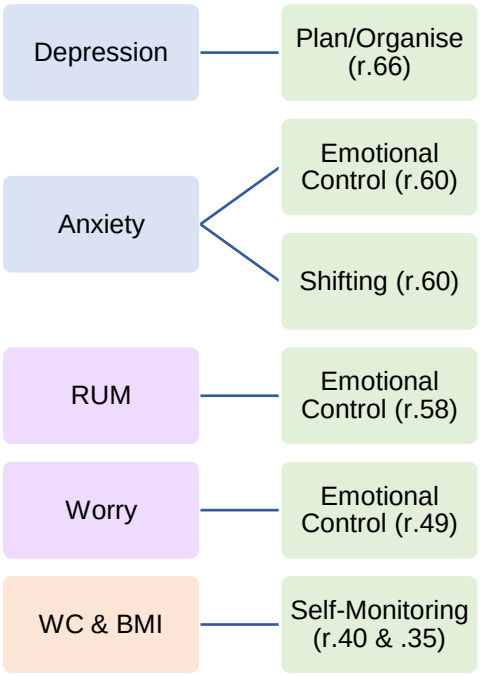


Figure 67
Highest Correlations Between Self-Reported Executive Function Problems and Health (continuous)
Note: WC= Waist Circumference

When examining the BRIEF scales that are most relevant to this investigation (inhibition and emotional control) health conditions most highly correlated with inhibition were depression and anxiety (*r*=.44), and the health conditions most highly correlated with emotional control were anxiety, depression and rumination (brooding).

Regression. The first model (Worry, WC and Anxiety) explained 31% of variance in self-reported inhibition but anxiety was the only significant predictor of

inhibition problems (Beta .75). The second model (worry, WC, RUMb and anxiety) explained 51% variance in Emotional Control problems and RUMb (Beta .28) and anxiety (Beta .52) were significant predictors.

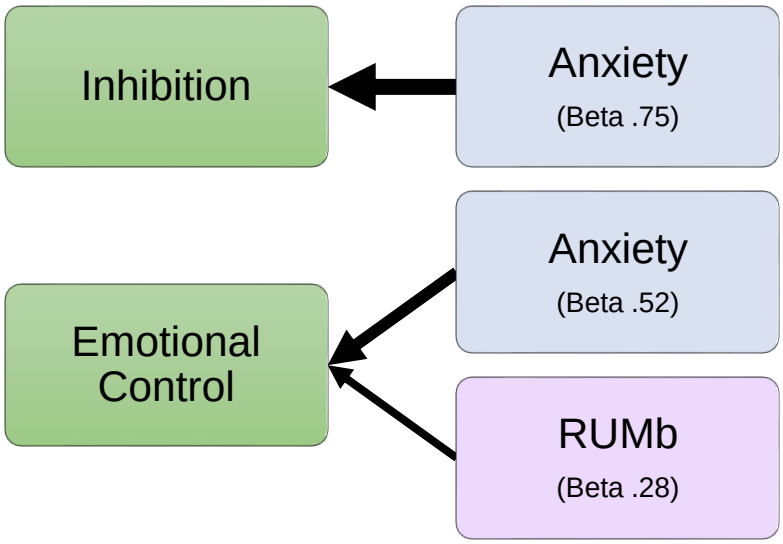


Figure 68
Executive Function in Daily Living Regression with Health

Mental health and repetitive negative thinking measures had moderate to strong correlations with the behaviour regulation index (*r*=.51 to.65) and the Metacognitive Index (*r*=36 to.59), whereas measures of adiposity had moderate correlations with the Behaviour Regulation Index (*r*=.30 to .32).

Investigation of which BRIEF scales were associated with mental and physical health (adiposity) found; Self-monitoring problems had the strongest correlation. For mental health emotional control had the strongest correlations (apart from depression where the plan/organise scale was higher). Emotional control appears to be relevant to both mental and physical health; in regression, a model consisting of worry, WC, RUMb and anxiety explained 51% of the variance in emotional control problem T-scores, but only anxiety and RUMb were significant predictors.

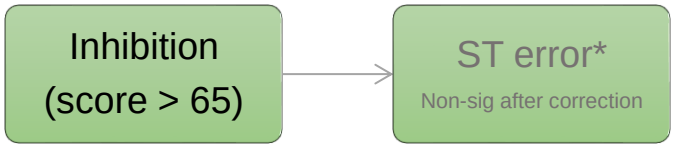


Figure 69
Findings for Self-Reported Inhibition and Cognitive Task Performance Errors

Executive Function in Daily living and Cognitive Performance

Those with high self-reported inhibition problems had higher mean percentage error in the ST task (*p*=.037) however this did not survive correction for multiple comparisons (alpha = .016). None of the other subscales had a significant effect on attention or inhibition error, including the scales that were identified as relevant to health outcomes (emotional control, self-monitoring and plan/ organise). There was no effect of BRIEF scales on reaction time.

Results4 Summary Resting State Neurovascular Activity.

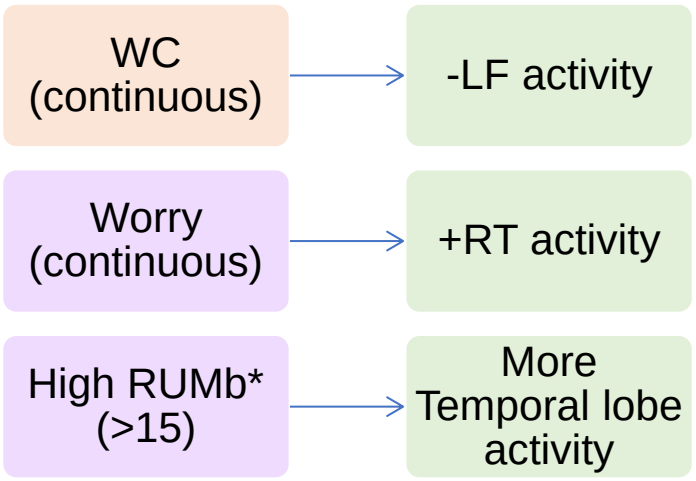


Figure 70
Neurovascular Activity and Health in the Resting State Summary (Regression and ANOVA)
Note: *=ANOVA results based on change in regional haemodynamic activity from baseline for those with high brooding rumination compared to low. += Positive association; -= negative association; RUMb= Brooding Rumination; WC= Waist Circumference; LF= Left Frontal, RT= Right Temporal

Regression. Higher Continuous WC was related to lower left frontal brain activity and explained 6% variance; Higher Continuous worry was related to greater right temporal lobe activity, and worry explained 8% variance.

ANOVA. High Binary RUMb: Greater activity in the bilateral temporal lobes (T3/T4) compared to those in the Low RUMb group.

Results5 Summary Cognitive Task Neurovascular Activity.

ANOVA. ***Attention (CT) task:*** On average the right hemisphere was more active (RT and RF) and the LF region was least active. **High WCr** individuals displayed greater activity in the RF (*p*=.013) compared to Low WCr; this could indicate they found the task more challenging***. Inhibition (ST) task:*** On average the frontal channels were more active than the temporal channels during this task. **High RUMr had** less activity LF(*p*=.008), and RT (*p*=.005) compared to low RUMr. This could indicate those with high RUMr found the task easier or that they were using a different area of the brain to deal with the task.

Results6 Summary Functional Connectivity.

For ***waist circumference risk (WCr)*** the high group (compared to low) had weaker and less extensive functional connectivity in the resting state and during the attention task, but extensive functional connectivity during the inhibition task (including strong functional connectivity within the RF, and left hemisphere).

For ***rumination,*** during the resting state the high group (compared to low) had more functional connectivity within the bilateral temporal lobes (indicating a high degree of internally focused processing), but their right hemisphere connectivity was weaker. During both the cognitive tasks the high RUM groups both showed weaker and less extensive functional connectivity. This lack of functional connectivity could

indicate lack of focus on the task, or that other brain regions were being used to complete the task. In the ST task the high rumination group showed higher functional connectivity within the RF whereas the low group recruited the LF.

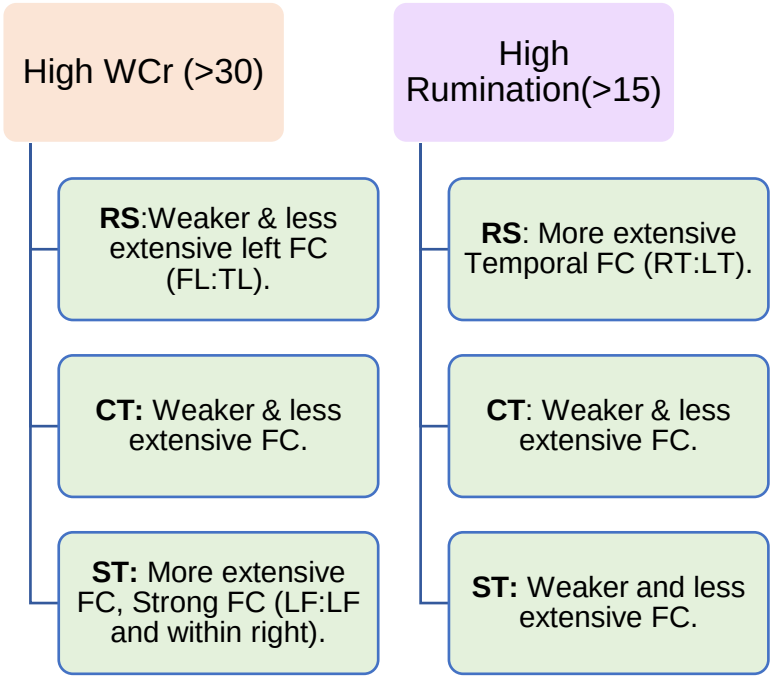


Figure 71
Summary of Functional Connectivity Findings for Those High in Waist Circumference Risk and Rumination (compared to low)
Notes: FC= Functional Connectivity; RS= Resting State; CT= Continuous Performance Task (Attention); ST= Simon Task (inhibition); WC*r*= Waist Circumference Risk

8.0 Study 2 Discussion

Study 2 aimed to investigate the relationship between adiposity and negative affect by investigating the nature of their effect on inhibitory function. Findings were triangulated from multiple methods (cognitive experiment, neural function and self-report) to see if there was any evidence of a shared causal mechanism for the effects of adiposity and negative affect on inhibition. The wider aim of the study was to feed into the discussion about the efficacy of the CAMMPI model to explain the interaction between adiposity and negative affect (examples of comorbid mental and physical health conditions). During the analysis, an exploratory approach was taken to examine the relationships between different types of measurement variables, including both continuous and categorical scores, to identify which have more merit to help explain the observed effects: general predictive associations or risk-related health categories.

Physical and Mental Health

This study examined the relationship between physical and mental health by investigating the link between negative affective conditions (depression and anxiety) and adiposity which are both associated with cardio-metabolic problems (heart problems, diabetes, blood-pressure, earlier mortality) and neurocognitive problems. In line with previous work, the current study found a significant association between mental health (negative affective conditions) and adiposity, however the mental health association was explained by repetitive negative thinking (rumination and worry) rather than wider symptoms of depression or anxiety. Repetitive negative thinking therefore seems to provide an important link between mental and physical health via neurocognition, however it is not yet clear if this is a causal relationship or

whether repetitive negative thinking is a marker of individuals who are potentially more vulnerable to cardio-metabolic health problems. The mental and physical health measures both related to negative effects on cognitive task errors, self-reported executive function problems and neurovascular differences, but the nature of the effects were not the same (indicating different mechanisms of effect). The mental and physical health effects can be summarised as follows:

Physical Health and Cognition. Those high in adiposity displayed more errors in both the attention and inhibition tasks. Neurologically, these participants had weaker functional connectivity during the resting state and attention task but greater connectivity during the inhibition task compared to those with lower adiposity. Adiposity was also associated with self-reported problems in self-monitoring.

Mental Health and Cognition. In most cases the effects of depression and anxiety were outweighed by the effects of repetitive negative thinking (brooding rumination and worry). Those high in rumination displayed more errors in the inhibition task but not the attention task. Neurologically, they had strong functional connectivity in the resting state but weak functional connectivity during the cognitive tasks. Rumination was associated with greater self-reported problems in emotional control. The significant cognitive findings are explained in more detail below.

Cognitive Performance Errors

The high BMI group (BMI>30) displayed more errors in both attention and inhibition tasks, whereas the high rumination and depression groups (RSS scores>15 and PHQ-8 scores >10) displayed more errors in the inhibition task. This indicates that adiposity affected neurocognitive mechanisms of both attention and inhibition. These tasks specifically examined the constructs using visuospatial tasks.

In previous research, most inhibition tasks have used tasks like the traditional Stroop, which rely on verbal ability which could be a confounding factor in our interpretation of the nature of neurocognitive effects of health. The motor speech areas of the brain include the inferior frontal gyrus and superior temporal lobe (Butler et al., 2020) hence this would potentially interfere with interpretation of activity in these brain areas. The task was also more inclusive to participants who have learning difficulties in reading speed/ word-finding (which is frequently undiagnosed) and would have been placed at a disadvantage in such tasks (Wagner et al., 2020).

Self-Reported Executive Function Problems

Self-reported executive function problems were measured to gain insight into the realistic daily living effects reported by participants with and without adiposity, mental health and repetitive negative thinking. The nine scales within the BRIEF tap two main executive function constructs: the Metacognitive index (relevant to working memory and planning) and the Behaviour Regulation Index (relevant to inhibition, shifting and emotional control). Adiposity was most strongly associated with the Behaviour Regulation Index, specifically self-monitoring problems. Mental health and repetitive negative thinking had moderate to strong association with the Behaviour Regulation Index and moderate relationship with the Metacognitive Index. When all the health conditions were considered, self-reported inhibition problems were only predicted by high anxiety. Emotional control (another facet of inhibition) was predicted by high anxiety and high brooding rumination.

Daily living problems were also examined in relation to cognitive performance. Inhibition problems were the only BRIEF scale associated with cognitive performance (attention and inhibition errors) but the relationship with inhibition errors was weak and did not survive multiple correction. Self-reported inhibition and

inhibition task errors could be measuring different but related constructs. One issue could be the way the BRIEF has separate scales for inhibition and emotional control whereas a cognitive task just looks at the general efficacy of behavioural responses. Both areas are indicated to have very similar neural substrates (Diamond, 2013).

Neurovascular Activity

fNIRS was used to explore whether physical and mental health measures were associated with different patterns of neurovascular activity – a proxy measure for how active different areas of the brain are in comparison to others. Brain activity was examined in areas associated with inhibitory function (frontal lobe) and internal thinking (medial frontal and temporal lobe) during rest and task and found significant differences in relation to health.

Resting State. During the resting state (RS) the participants with high adiposity had a smaller magnitude of activity in the Left Frontal (LF) lobe (6% of LF variance was explained by waist circumference) and functional connectivity was weak. Participants with high worry had a larger magnitude of activity in the right temporal (RT) lobe (8% of RT variance was explained by anxiety). When examining participant high/low groups there was a main effect of brooding rumination; greater brooding was associated with more resting state brain activity in the temporal lobes and stronger, more extensive functional connectivity.

During Task Completion. There were further health-mediated differences in brain activity when participants were completing the cognitive tasks. During the attention task the high WCr (waist circumference risk) group displayed more activity in the right frontal lobe than the low WCr group. Functional connectivity found that

the high WCr group had very weak active connectivity during the attention task, but higher connectivity during the inhibition task.

For high ruminators, during the inhibition task they displayed less activity in the LF(left frontal), and RT (right temporal) regions compared to low ruminators. Functional connectivity was similarly weak compared to low ruminators during both tasks. This could indicate that high ruminators are using different areas of the brain to process the task. Mental health problems are sometimes associated with thinner grey matter in certain regions (Kandilarova et al., 2019) which could account for reduced activity (and by implication cognitive deficits) being due to fewer cells in these areas (therefore oxygen requirements are lower). This later explanation seems less likely however, as during the resting state the high ruminators had more haemodynamic activity compared to low ruminators (indicating the physiological capability for greater haemodynamic activity is still there).

Adiposity and Mental Health

When investigating the significant links between adiposity and mental health, RUMb was associated with continuous measures of adiposity. Prediction 1a), that larger body measurements are related to greater negative affect and greater ruminative cognition, was generally supported. Brooding rumination score was a significant and stronger predictor of waist circumference and BMI than depression score. Mental health (brooding rumination) and physical health (WC) explained a similar amount of variance in one another (around 5%).

This partially meets predictions of an association between adiposity and mental health although it indicates that excess body fat is more relevant to repetitive negative thinking, (i.e., the cognitive symptoms of depression/anxiety) than other components of depression and anxiety, such as negative feelings. This interpretation was strengthened as multiple regression found RUMb explained 5% variance in WC and 5% in BMI, but depression did not add significant variance.

One explanation for the link between depression and increased body mass or adiposity is comfort eating. Not all emotional eaters become obese (Vasileiou & Abbott, 2023) therefore a propensity for depression or repetitive negative thinking could help identify subgroups who are vulnerable to future health problems. A large prospective study (Konttinen et al., 2019) examined the link between depression, BMI / WC and emotional eating (Three-Factor-Eating Questionnaire) in over n = 3700 people at baseline and 7-year follow-up. Importantly, the anthropometry measurements were conducted by trained nurses. They found that self-reported emotional eating predicted increased BMI and WC in those who slept fewer hours (<7 per night). This suggests a role for lack of sleep within the process (lack of sleep could be a symptom of stress/ rumination or a causal factor). Emotional eating mediated the effects of depression on BMI and WC in young and middle adults. Interestingly exercise was not a significant moderator of the effects.

Work by Gordon et al. (2012) suggests that binge eating behaviour serves as an emotional escape from aversive feelings perpetuated by ruminative thinking, (per Escape Theory, Baumeister, 1991 and Cascade Theory. Selby et al., 2009), i.e. that rumination and body dissatisfaction interact to produce binge eating behaviour. Escape theory and the Emotional Cascade Model propose that people can use extreme behaviours to escape from cycles of extreme repetitive negative thinking.

Baumeister used Escape Theory to explain that suicide can arise from a chain of events that begin with internalised feelings of inadequacy that make introspection painful and leads them to cut-off from their emotions and limit their thinking (a form of attentional restriction). This effortful process uses up cognitive processing resources which can result in disinhibited behaviour (Baumeister, 1990). Selby (Selby et al., 2008) proposed a model of emotional cascades whereby negative affect and aversive rumination/ catastrophising (a form of intense worry) reinforce each other in a cycle, leading to thought suppression and behaviour dysregulation (self-injury/ binge-eating) as a coping mechanism. These behaviours bring relief, but only for a short period, this means they can become habit-forming.

Rumination has also been found to affect sleep (Hairston et al., 2022; Zawadzki et al., 2013) which it appears is an additional vulnerability factor in the depression-adiposity association. A further study (Kornacka et al., 2021) examined rumination, mood and emotional eating in obese and normal weight controls by self-report (n=88) and Electronic Momentary Assessment (EMA; n=26) allowing a more objective assessment of eating behaviour and rumination. The EMA study (although small) found that EMA rumination predicted EMA emotional eating in both obese and normal weight participants. The authors emphasise the importance of unconscious behaviour in relation to eating and ruminative thinking, which means it can be problematic to rely on self-report. This should be considered in future rumination studies.

Cognitive Performance and Health

Prediction 2a) that worse performance will be seen on the more cognitively demanding inhibition (Simon Task; ST) than the simpler attention task (Continuous Performance Task; CT) was supported; the inhibition task required significantly more time to respond and had significantly more errors.

Prediction 2b), that significantly worse cognitive performance would be observed for those with greater negative affect and larger body size was supported although health effects were selective. Those with high BMI (>30) had more errors on the attention task but not the inhibition task (compared to those with normal/increased BMI <30). Those with high depression and brooding rumination had more errors on the inhibition task (compared to those with lower levels of negative affect). Overall, this indicates that excess weight was significantly associated with lower accuracy in both attention and inhibition, while mental health (negative affect/ repetitive negative cognitions) affected inhibition performance (not attention).

There was no significant effect of health on mean reaction time, however there was a significant Simon Effect. The normal/increased BMI risk group showed more discrimination between incongruent and congruent trials (a significantly greater difference between reaction times indicating that the high BMI risk group had slower reaction times than the normal/increased group (not significant). Despite this, the Simon Effect was less apparent because congruent performance was also slower in the high BMI group. The effect on congruent performance indicates that these BMI mediated effects on reaction time were not only due to differences in inhibition. Details of the findings are discussed in more detail below.

Attention and Inhibition Errors - BMI

Those in the moderate to severe BMI risk group (BMI >30) made more errors than the low to normal risk group in the attention task (Continuous Performance Task; CT) and the more challenging inhibition task (Simon task; ST) however, it should be noted that the number of errors was small. Notably, in the ST those in the high BMI group were more prone to errors on the congruent trials compared to the low BMI group. In the ST participants are expected to make more errors on the incongruent trials because they require participants to withhold their natural tendency to respond laterally to stimuli, i.e., if you see the stimuli in the left visual field, your natural reaction is to respond to it using your left hand. Overall, as expected, there were more errors on incongruent trials. The high BMI group also had more errors on congruent trials. The inhibition findings in this study are similar to cognitive experiments by Sellaro and Colzato (2017) who found that an interaction between a Gratton effect (a known adaptation effect whereby participants make errors based on the congruence of previous trials) and the high BMI group (low/high) showed more congruent errors. This indicates the high BMI group may have had more difficulty with suppressing the outcome from previous trials which (per Sellaro and Colzato) this may translate to a bias toward automatic (reward-driven) responses rather than effortful goal-related ones. Sellaro and Colzato (2017) also utilized a ‘go-no go’ trial task and like the current study, they did not find significant differences in ‘Go trial’ reaction based on BMI.

An experimental study of obesity and inhibition (Iceta et al., 2020; n=90) found that obese females who also reported disinhibited eating made significantly more omission and commission errors. EEG scans found that obese participants regardless of disinhibited eating had reduced attention signalling compared to

normal weight participants. This suggests a relationship between adiposity and reduced attentional control, but there is not a clear-cut relationship between obesity and disinhibited eating behaviour– rather it is a subgroup which are affected. The link with attention control might suggest more difficulty with monitoring food intake which could affect eating behaviour and the extent that participants were aware of the issue. This explanation is reinforced by the current findings of an association between high adiposity and more self-reported difficulties with self-monitoring.

Inhibition Errors: Depression and Brooding

In the current study there was also a significant effect of depression and RUMb on inhibition errors – those in the clinical (high) RUMb group showed more errors on the inhibition task (ST) but not the attention task (CT). It should be stated that when the Simon Effect was investigated, this measure of inhibition did not show a significant difference between groups. The effects of depression and rumination on spatial response inhibition should therefore be treated with caution, thus the nature of the inhibitory effects bear further investigation.

Introzzi et al., (2016) examined the effects of rumination on perceptual (visual search), cognitive (learned cue recognition task) and behavioural inhibition (Stop Signal Task). Their study found that behavioural and perceptual inhibition were poor predictors of rumination but showed a more robust effect of cognitive inhibition (learned cue recognition). The authors indicate this meant that rumination did not so much effect attention capture (perceptual inhibition control) or established behavioural habits (motor inhibition) but it exerted effects through the internal representations of thought processes. They found significant effects in relation to reflective and brooding rumination, however the effects were more pronounced in

relation to brooding rumination. The cognitive task was a variation of a Steinberg task and the impact of rumination on this task tends to indicate that rumination is indirectly affecting inhibition through an inability to suppress (or forget) previously learned information. This is commensurate with the findings of Study 2 that high adiposity was related to more cognitive errors due to difficulty 'forgetting' or suppressing previous trials/responses. Interozzi et al.,'s finding also calls into question whether executive inhibition (and executive function in general) is actually the cognitive construct that is affected, or whether reward learning or adaptation is more relevant to the issue.

When error type was examined in the current study, those with high levels of depression symptoms were more prone to errors on incongruent trials than the non-clinical group, whereas those with high RUMb were prone to errors on both congruent and incongruent trials compared to those with low RUMb. This means that high ruminators displayed a similar pattern of cognitive effects to those with high BMI, where performance (errors) were affected by both inhibition (incongruent trials) and another cognitive effect (on congruent trials). Based on previous literature this is likely to be a response adaptation effect as outlined by Sellaro and Colzato (2017). Neurocognitive investigation of cognitive adaptation effects indicates they are exerted though episodic (experiential) memory, rather than executive function (Mayr et al., 2003). Specifically, the rostral Anterior Cingulate Cortex (ACC) is implicated in adaptation effects that are asserted to occur during selective attention and causes stimulus-specific priming in repetitive tasks, allowing them to respond more quickly. Importantly this priming effect is proposed to result from memory rather than conflict-monitoring. Further investigations of cognitive adaptation effects in relation to health should therefore consider the action of the rostral ACC.

Returning to the other mental health group of the current study, in contrast to the self-reported executive function findings, there was no significant effect of worry or anxiety on cognitive errors. These findings are in line with a cognitive study (n=80; Ng et al., 2012) which found depression was associated with more Simon Task errors, but anxiety was not. The authors suggest this could be due to different brain areas affected by the conditions: anxiety was suggested to affect the ventral cortico-limbic structures (e.g. the inferior temporal, and orbitofrontal cortex- adjacent to the areas we measured) which affects control of focused attention, whereas depression affects dorsal cortico-limbic structures including the superior frontal lobe (DLPFC) which affect external attention and spatial processing. If it was the case that anxiety has more of an effect on focused attention than depression, we may have expected to have seen an effect of anxiety on the CT attention task performance, but this was not the case.

Majeed et al. (2023) found that the effect of anxiety on cognitive performance was more nuanced. In their meta-analysis, those with anxiety had lower accuracy on inhibition tasks, but greater accuracy on working memory task (updating). This can be explained in reference to the Attention Control Theory (ACT; Eysenck et al., 2007; McNally, 2019) i.e., that under stress those with anxiety can recruit additional resources to accurately respond to tasks, particularly when these tasks are relatively simple and do not directly contain stressful stimuli. In some tasks those with anxiety have outperformed controls. Notably, in the meta-analysis (Majeed et al., 2023), the severity of anxiety disorder was not significantly related to cognitive outcomes which could explain the observation in this study that subcomponents of the condition (i.e., repetitive negative thinking) are more relevant to inhibitory function (or adaptation

response) than the whole condition itself. In future it would therefore be recommended to measure the extent of repetitive negative thinking as a priority over general anxiety or depression symptoms.

fNIRS Resting State (RS) Differences in Brain Activity

Prediction 4a) that the temporal lobe areas will show a greater increase in haemodynamic activity than the frontal cortex during the resting state was supported. The region with the greatest magnitude of change from baseline was the left temporal region (indicating that this was the most active area). There was also a significant difference in the right hemisphere with the right temporal region being significantly more active than the right frontal region. This is in keeping with greater internal processing during the resting state.

Prediction 4b) that ***larger body-sizes*** will be related to significant differences in the pattern of haemodynamic activity during the resting state in the frontal and temporal areas was partly supported. Those with higher waist circumference risk (WCr) had less activity in the left frontal lobe reflecting a different pattern of brain activity compared to participants with normal WCr. As the frontal lobe is implicated in task-based processing, this could indicate that high WCr participants were giving less attention to their external environment during the task.

Prediction 4c) that ***greater negative*** affect will be related to significant differences in the pattern of haemodynamic activity during the resting state was also partially supported. In continuous health measures those with high worry had more haemodynamic activity i.e., change in right temporal activity compared to baseline (small moderate correlation). During high-low group-level analysis the high brooding rumination group also displayed significantly more activity in the medial and superior temporal lobes (bilaterally but more on the right) compared to RUMb. Worry and rumination therefore appear to be related to increased temporal lobe activity in the resting state. As the temporal lobe is implicated in internal processing this could

reflect greater levels of self-focused brain activity compared to participants with low levels of worry and rumination. The findings are discussed in more detail below.

Regional Differences in Resting State Brain Activity

Initial analyses sought to confirm whether the different brain regions examined (frontal and temporal lobe) were reliably measuring different neurovascular indices. It was predicted that the medial temporal lobe, (as a potential part of the Default Mode Network; DMN) would show greater haemodynamic activity in the resting state than the frontal cortex, as this area deals with internal and self-focused thinking. This prediction was supported; participants' medial temporal lobe region (a node of the DMN) was more active during resting state than the frontal lobe region. The left temporal lobe (medial temporal gyrus/Brodmann's area 21) appeared to be more active, and the right temporal lobe was significantly more active than the right frontal lobe. These findings were broadly in keeping with previous studies of the frontal/temporal BOLD signal in the **resting state.**

Mesquita et al. (2010) conducted a whole brain fNIRS functional connectivity analysis in the resting state with 11 healthy adult males. They examined various seed regions (sampled brain areas) and found for each seed there was a reliable contralateral brain activation i.e. a smaller activation (correlation strength of around .2) was seen on the opposite side of the brain. In examining inter-regional functional connectivity, the frontal and temporal regions on the same side were highly correlated (.8) but contralateral correlations with other regions were not.

It has been reliably established that during rest DMN regions become more active (N=10; Fox et al., 2005) and areas in the DMN (which includes the medial prefrontal cortex and medial temporal lobes) have stronger functional connectivity. A healthy brain response therefore would be expected to show an anticorrelation

(phasic negative correlation) between these DMN regions and that of the task-related regions such as the Pre-Frontal Cortex. This was the case in the current study for the overall sample during the resting state.

Resting State and Health

Continuous measures of WC, BMI, anxiety and worry, and categorical measures of RUMb were significantly associated with haemodynamic activity (OHb) activity during the resting state. There were negative correlations between regional haemodynamic activity and physical health and positive correlations between mental health and OHb: Those with higher BMI/waist measures showed less haemodynamic activity in the left frontal region however those with higher anxiety and worry tended toward greater haemodynamic activity in the right temporal region. In short, in the resting state, greater body mass was related to less LF activity, whereas higher negative affective conditions were related to greater RT activity.

Rumination, particularly RUMb, was the only health measure that showed a significant between-group difference in resting state activity. The effects of adiposity, worry and anxiety on neurovascular activity were no longer significant when analysis used high versus low group comparisons, but the effect of RUMb became more pronounced; participants in the high brooding group (who reported moderate or high brooding) displayed more than double the haemodynamic activity compared to the low brooding group. This is further indication that repetitive negative thinking (or its underlying cause) has an important influence on neurocognitive function.

Regional comparison of high and low brooders found that high brooders had significantly more haemodynamic activity in the medial temporal lobe which is consistent with high brooders being more engaged in internal processing/ self-

related thinking. High brooders also had a more active left frontal lobe during the resting state. We would expect the frontal lobe to show little activity during the resting state so this finding could mean that participants who were high in rumination were also less effective at down-regulating the task-relevant areas of their brain than low ruminators (Bartova et al., 2015).

Resting State Functional Connectivity. Functional Connectivity (FC) analysis was used to investigate the relationship between WCr and frontal/temporal lobe activity in more detail as it allows a compact visual comparison of all channels and the regions, they are most synchronised with.

Rumination. High RUMb participants display significantly more haemodynamic activity in the medial temporal gyrus (particularly the left) and had a more diffused profile of functional connection than those with low brooding levels. High ruminators had more cross-regional connections (left medial and superior frontal gyri showed synchronised activity with their more active temporal regions) which was not the case for low ruminators. Low ruminators recruited fewer regions in the resting state indicating less diffuse activity, but connections were synchronised more strongly. Functional connectivity patterns therefore supported the assertion that brooding ruminators are less affective in downregulating the left frontal brain regions. The effects of this could include that ruminators are less able to mentally 'switch off' during quiet time. This could be a factor that influences inhibition task performance. As the resting state would not directly affect task outcomes unless it intrudes into task completion so this effect may not be due to extra cognitive load (as hypothesised within the model) as much as fatigue effects. An alternative

explanation could be that the stark reduction in frontal and temporal functional connectivity during task completion could be in keeping with participants using a different area of the brain to process the task. There was a further difference during the Simon task (inhibition), that high ruminators had stronger localised activity within the right frontal lobe, whereas the low ruminators had stronger activity within the left frontal lobe (and more connectivity in general). Left frontal lobe activity is associated with more efficient processing in the obesity studies examined.

Waist Circumference. Functional connectivity was also examined in relation to waist circumference and waist circumference risk (WC/ WCr; the adiposity measure with the most significant health associations). During the resting state those with high WCr had weaker and less extensive functional connections than the low WCr group. A similar pattern was observed during the attention task. A contrast was seen during the inhibition task however, as the high waist circumference group had extensive functional connectivity including strong connections between the right frontal lobe and the left hemisphere.

In-line with these findings, Rosch et al. (2020) found that obese compared to normal weight participants showed less activity in the frontal lobe (left VLPFC/ IFG and right DLPFC) and this was related to evidence of inhibitory control problems. Analysis of their fNIRS data in relation to questionnaire findings found that self-reported impulsivity and emotional dysregulation were also significantly related to frontal lobe activity (left VLPFC activity and bilateral DLPFC activity) – however no cognitive performance measures of inhibition were reported. In more recent work Stinson et al., (2022; n=29) found that transcranial magnetic stimulation (tCDS) to

the frontal lobe (DLPFC) improved performance on a food-related inhibition task and reduced snacking and hunger in a randomised control trial with 31-day follow-up. This implicates the superior frontal lobe (DLPFC) as an important area in the link between inhibition and eating behaviour, however tCDS for non-food inhibition tasks indicates that the inferior frontal gyrus (VLPFC) is the most relevant frontal area for non-food related inhibition (N=67; Schroeder et al., 2022). This means that less effective inhibition task performance should not be assumed to mean less self-control overeating behaviour as different areas are involved.

fNIRS Task-based differences in Brain Activity

Task-based fNIRS scans were used to explore whether participant health groups also showed differences in brain activity during the simpler attention task versus the slightly more demanding inhibition task. This was a novel exploration as it examined the general activity during the task, rather than brain activity that was time-linked to the individual tasks. As RUM and WCr groups had shown significant effects on regional haemodynamic activity they were explored in more detail using functional connectivity analysis.

Waist Circumference Risk and Brain Activity During Task Performance.

Prediction 5a) that there would be significant differences in haemodynamic activity in the prefrontal cortex based on obesity (low/high) were supported. Those with high WCr had more activity in the RF region, possibly indicating they found the task more difficult (having to expend more effort to maintain their attentional focus) than those with low WCr.

Prediction 5b) that there would be significant difference in haemodynamic activity in the prefrontal cortex based on negative affect (low/high) was also supported. Those with high reflective rumination (RUMr) had less activity in the LF and RT regions compared to low RUMr. This could indicate that those high in reflective rumination found the task easier or it could indicate that they were using a different area of the brain to deal with the task compared to those with low rumination. Findings are discussed in more detail below.

An fNIRS experiment by Rosch et al., 2020 (n=40) found that right inferior frontal lobe (VLPFC) showed more activity in a more challenging Go/NoGo task than a passive viewing task. This illustrates the link between task demands and neural activity and supports the interpretation that greater activity *during a task* is a sign of greater effort or engagement. Rosch examined the difference in brain activity between obese and normal weight participants during a passive viewing task and an inhibition task. The obese participants had less activity in the left superior frontal lobe (DLPFC) compared to those with normal weight. The applicability of Rosch's study findings should be treated with caution as it used food-related stimuli and the task conditions were slightly different to the current study but still, in regional analysis the current study found similar to Rosch that those with high waist circumference risk had less activity in the left frontal lobe during the resting state. During the attention task in the current study participants with high waist circumference displayed greater right frontal activity than those with low waist circumference. The functional connectivity analysis in the current study provides an additional perspective on neurological activity; this agrees with Rosch's findings, showing weaker and less extensive connectivity in the frontal and temporal lobe during the attention task.

The participants in Rosch et al., (2020) also self-reported impulsivity (Behavioural Inhibition System/Behavioural Activation System Questionnaire) and emotional dysregulation (Difficulties in Emotion Regulation Questionnaire). Impulsivity (which has some relationship to inhibitory control (Bari & Robbins, 2013) was related to stronger prefrontal cortex responses while emotional dysregulation (which is relevant to awareness and control of emotional responses; Hallion et al., 2018) was related to weaker prefrontal cortex responses during the task. The findings in relation to impulsivity could help explain why participants in the high waist circumference group show more functional connectivity during the inhibition task; they were putting in more effort to complete the task (inhibiting response). Rosch's findings in respect of emotional control are also of interest because in the current study, participants with higher repetitive negative thinking also displayed reduced frontal lobe activity during the cognitive tasks. As the frontal lobes were less active this tends to indicate that these individuals were using a different area of their brain to complete the task, however Rosch only examined the frontal lobe.

In a review of fMRI findings relating specifically to the Simon task of inhibition (ST; Cespon et al., 2020) the most common brain area reported as being activated during the task was the inferior parietal lobe. This is nearer to the back (posterior) of the brain, so it was not possible to examine this region in the current study, however the DLPFC is usually activated as part of the same network (the frontoparietal network). The next most common areas activated during the Simon Task were the medial frontal gyrus, superior (DLPFC) and inferior frontal gyri (VLPFC). The authors found there was an even split between studies reporting left and right hemisphere

activation in these areas, so this means any interpretations made of hemispheric differences are tentative.

Cespon et al. (2020) suggests that the DLPFC is not essential to ST performance but that it might be involved in working memory processes that encode the stimulus-response rules, and as such assist with preparing a response in advance (leading to the sequential effects observed). Correct preparation helps the participant to anticipate what will happen next, facilitating a fast and accurate response (rather than just a fast guess). This explanation would fit with the interpretation of the increased congruent task errors (related to WCr) in the current study. Under this explanation, the increased congruent task errors could be explained by ineffective sequential preparation effects – i.e., having to proceed with a fast guess based on the previous trial rather than work through the longer process of detecting the stimuli, remembering which was the correct button and signalling the correct response.

In all, the review by Cespon et al., (2020) identified four different patterns of brain activity associated with the ST: ***two frontal*** routes (middle cingulate cortex and medial frontal cortex; left middle frontal gyrus and left pre-central gyrus), ***parietal*** (right superior and inferior parietal lobes) and ***occipital-temporal*** regions (middle and inferior temporal gyri and middle occipital lobe). They also concluded that the route involving the cingulate cortex (specifically the ACC) was involved in sequential effects i.e. learning the pattern of effect and using this to predict upcoming stimuli. As the distribution of trials was random if a participant was reliant on this processing route it may be anticipated to lead to more errors. The current study was not able to measure activity in the cingulate cortex in this pathway but greater middle frontal

gyrus activity in high waist circumference and high ruminators would support this theory.

Functional Connectivity During Task Performance

No specific predictions were made about functional connectivity beyond those noted in Result 4 and 5. The investigation was exploratory to see which functional areas were working together and identify if any areas were less well connected to shed light on the nature of any inhibition deficits in the high compared to low health groups. The pattern of functional connectivity was different depending on the task (resting state, attention task, inhibition task).

Waist Circumference Risk. Synchronisation in the fNIRS channels during the tasks was compared the low/high groups for WCr and RUM using Functional Connectivity matrices. For WCr those in the high WCr group showed markedly less synchronised activity between channels during the ***attention task***. This could signal lower engagement or more of an 'auto pilot' response for the high WCr group as this was a very repetitive task i.e. the findings could indicate less active attentional focus. The attention task findings contrast with the ***Inhibition task*** where the high WCr group showed much higher levels of functional connectivity compared to the low-risk group; there was strong connectivity within the RF, the LF and the LT channels. Certainly, the frontal lobe areas appeared to be more active in the high WCr group than the low WCr, as they displayed relatively strong functional connectivity between frontal hemisphere channels RF and LF1-3 (IFG). This could indicate that the high WCr participants were expending more effort and recruiting more brain areas to complete the task than the low WCr group.

A small fNIRS study of patients with sport-related concussion related brain damage (n=9; Kontos et al., 2014) predicted functional impairment would result in more bilateral activation (increased PFC and DLPFC activation) or more diffuse activity during a cognitive task battery due to altered cognitive resource allocation. In a symbol matching task, the presentation phase showed activation of the superior frontal lobe (DL PFC) and the recall phase showed activation of the left frontal cortex – the concussed group had reduced activation in the left frontal cortex and more incorrect responses in the task. This is in-keeping with the findings from the WCr functional connectivity whereby the simpler attention task had very little functional connectivity, but the more challenging inhibition task had more activity including bilateral recruitment of both frontal lobes.

Rumination. Health related findings for the inhibition task found a significant effect of rumination on brain activity– the high RUMr group showed greater activity in the left frontal (LF) and right temporal (RT) region compared to the low RUMr group. ***Medial temporal*** activity is less commonly associated with ST performance but has been reported, primarily in the right hemisphere. Cespon et al. (2020) suggest that this is consistent with recruitment of the dorsal ACC (Anterior Cingulate Cortex) to detect conflict. ACC involvement is further related to Medial Frontal Negativity revealed by EEG which is a type of conflict adaptation where the action is locked to the previous response. Garavan et al. (2002) found that increased ***medial frontal*** activation (anterior cingulate cortex; ACC) was associated with more inhibition task errors – participants recruited this area when the inhibition tasks were hard, and they were working at fast speeds. They suggest this represented an 'urgent' response pathway, used instead of the DLPFC, and found it was more likely to be used by

participants who self-reported high cognitive failures. Cespon et al. (2020) also linked the use of the ACC pathway to more complex or demanding task conditions. The high RUM participants recruitment of the Medial Temporal regions therefore fits the characterisation that they had additional cognitive demands on them compared to the low RUM participants, or that the normal DLPFC pathways were unavailable or actively being bypassed.

A series of fNIRS studies by Rosenbaum et al (n=84; Rosenbaum et al., 2017; Rosenbaum et al., 2018) found that during stressful tasks (and control tasks), high ruminators had reduced activity in the right inferior frontal gyrus (IFG/ inferior frontal lobe). Due to the known function of these networks the authors concluded this reduced activity was likely to reflect general deficits in inhibition and attention of high ruminators compared to controls. A more recent investigation (Rosenbaum et al., 2021) found that MDD (Major Depressive Disorder) participants (n=22) had reduced haemodynamic activity in their cognitive control network (left DLPFC and inferior frontal lobe) compared to controls (n=23). The fNIRS measurements were taken during a range of conditions (pre, post and stress tasks); reduced haemodynamic activity during the stressful arithmetic task was related to group level differences in post stress rumination (r^2=.62). It should be noted that fNIRS scans were conducted with a variety of task conditions, but only selected tasks found significant effects. This illustrates the ambiguity in findings when investigating this area.

A lesion study (n=12; Swick et al., 2008) also highlighted the importance of the left inferior frontal gyrus (VLPFC) in suppressing prepotent responses. Participants with left inferior frontal gyrus lesions displayed more commission errors on more difficult inhibition tasks – seemingly they were responding more impulsively.

The participants were noted to only recruit both the right and left inferior frontal gyrus when the task was more challenging (requiring some working memory), therefore when the task was easy and they were likely responding on ‘auto pilot’, they did not use their right inferior frontal gyrus. Conclusions from lesion studies are somewhat limited as the lesion can affect multiple areas, however the findings seem to be in accordance with other research suggesting that although the right inferior frontal gyrus is generally important for inhibition, the left inferior frontal gyrus can provide extra support on more demanding tasks (Cespon et al., 2020). Strong recruitment of the bilateral inferior frontal gyrus therefore suggests the task is difficult or the individual is finding the task more difficult.

Rumination. For the RUMr and RUMb groups during both tasks, low ruminators showed stronger synchronisation within the left frontal lobe channels (including LF1-3 i.e. IFG/VLPFC) with smaller and fewer connections across the frontal lobe to the right hemisphere. There is some discussion over the lateralisation of brain activity during inhibition tasks (whether one or both hemispheres are important) the consensus has supported the importance of the right frontal lobe (right Inferior frontal gyrus) in combination with the parietal lobe to the extent that most inhibition studies do not even examine the left hemisphere. In a well-controlled tCDS study the left hemisphere was also not found to be important to inhibition task performance in the Stop Signal Task (n=67; Schroeder et al., 2022). In the current study, high ruminators tended to show the strongest functional connectivity in the RF but had substantially fewer synchronised channels. High and low ruminators therefore were recruiting slightly different brain regions in the task. High ruminators appeared to be depending more on their right frontal lobe and fewer brain channels were working together in unison across the lobes.

The observation of more functional connectivity in the inhibition compared to attention task fits with predictions and extant research. As the attention task recruited fewer strong functional connections than the inhibition task and based on previous research relating to fNIRS activity to cognitive effort (Fishburn et al., 2014; n=16), it could also be presumed that the high brooders were less engaged in the attention task or less able to focus on it, whereas the inhibition task paradigm prompted greater engagement. Further, low brooders only showed left temporal connection in the simpler attention task, whereas high brooders showed the reverse pattern with strong connections between LT1 & 2 in the more challenging task.

One explanation for the pattern of functional connectivity observed by high ruminators could have been task difficulty. There were very few errors made in either task which suggests most participants did not find the tasks very difficult. In a critical review of response inhibition fMRI findings, Criaud and Boulinguez (2013) suggest that much of the neural activity attributed to inhibition is actually focused on attentional and working memory resources. The review examined simple versus complex inhibition tasks, with and without high attentional and working memory requirements, and identified that activity in the right superior and inferior frontal regions (right DLPFC and right IFG/ VLPFC) are found in more complex tasks with and without working memory overheads. In the current study, one task focused on attention (CT) and another, slightly more demanding task, focused on response inhibition (ST). In both tasks the high RUM participants had weak and less extensive functional connectivity, however they also showed more connectivity in the RF region than the left. The high RUM participants in this study therefore appear to be relying

more heavily on working memory and attention resources to complete the tasks than the low rumination participants.

Garavan et al. (2002) suggested (in contrast to Criaud and Boulinguez, 2013) that normative inhibition function was more reliant on the right DLPFC, and the left DLPFC was recruited when participants were adjusting their behaviour in response to an error, but the study did not attempt to differentiate between inhibition/working memory/attentional constructs. Somewhat contradictorily, Goldberg et al. (1994) emphasised that the right frontal lobe has an important role in processing and adapting to cognitively novel situations whereas the left frontal lobe is more adept at dealing with well-learned tasks. It could therefore alternatively be suggested that high RUM participants were not able to habituate to a task as well as low RUM participants or had more difficulty remembering the task instructions (which button to press and when). This fits with the findings of Criaud and Boulinguez (2013) and may account for greater reliance on rDLPFC.

Support for the Theoretical Model and Further Investigation.

The findings support the proposed model that high brooding rumination is associated with inhibition and sustained attention errors, increased resting state functional connectivity, and decreased functional connectivity during attention and inhibition tasks compared to low ruminators. Repetitive negative thinking therefore provides a promising link to explain the reciprocal effects between adiposity and affective mental health conditions. One explanation is that extra cognitive load arises due to a failure to downregulate negatively valanced internal processing which puts strain on executive functions (Nolen-Hoeksema et al., 2008), including inhibitory control and sustained attention. This may result in different pathways in the brain

being recruited to deal with more complex tasks, such as the anterior cingulate cortex leading to fast but less accurate responses (as they are based on participant predictions based on previous trials). Use of this neural pathway is found when participants are depressed, anxious and have increased adiposity. With high adiposity an additional effect occurs that leads participants to have greater attention problems and could be linked to reduced activity in the left frontal lobe (or diversion of processing resources away from the left frontal lobe).

In the current study, resting and task based haemodynamic activity in the temporal lobe and frontal lobe was affected more by self-reported brooding than clinical levels of depression or anxiety. It illustrates the important impact of RUMb on brain function in the frontal cortex, indicating brooding is more relevant to self-reported executive function difficulties than other negative affective symptoms. It could be tempting to characterise this as an emotional effect – that anxious and depressive symptoms cause negative feelings resulting in biased thinking and more-negative self-assessments. However, the fact that executive function difficulties are more strongly related to rumination than depression emphasises the role of negative thought-processes in exacerbating cognitive difficulties.

Although correlations between continuous measures of body size and haemodynamic activity in the left frontal lobe were significant, the binary BMI and WCr groups (based on clinical anthropometric risk categories) did not show significant differences in haemodynamic activity in these regions. This could indicate that the risk-based groupings (derived from negative physical/ cardiometabolic ill-health rates) were not sensitive enough or do not map on to the observed neural (mental health) processing effects. Obesity (BMI >=30; high risk of co-morbidities) was chosen as the clinical cut-point (WHO, 2000) used in the binary BMI risk

variable and the Waist Circumference cut-point was >=94cm for males and >=80cm for females (increased risk of co-morbidities). Different cut-points for excess weight in relation to mental health effects may therefore warrant investigation. Although the binary groupings maximised the size of the groups and used clinical cutoffs that were associated with increased health co-morbidities the 'normal-increased' BMI group was considerably larger than the increased risk group. A purposive sampling approach in future work may also be beneficial to check whether these findings hold true with more balanced group sizes.

Self-reported Executive Function, Health, and Cognition

Prediction 3a), that self-reported executive function in daily living was more strongly associated with negative affect than adiposity was generally supported. Self-reported executive function problems were significantly correlated with adiposity, mental health, and repetitive negative thinking. BMI and WC were moderately associated with subscales in the Behaviour Regulation Index. Self-monitoring and emotional control both had moderate-sized, highly significant associations with adiposity. There were small positive correlations with self-reported Inhibition, but these did not survive adjustment for multiple correction. Mental health scores correlated with both indexes of executive function problems in general these were moderate to strong and highly significant. Brooding rumination and depression had stronger correlations with the Behaviour Regulation Index than the Metacognitive Index (MI).

Reflective rumination scores did not correlate significantly with any of the BRIEF scales. This was interesting as RUMr should signal more adaptive repetitive

thinking (Nolen-Hoeksema et al., 2008) but if this was the case we might have expected to see negative correlations with some MI subscales. There was a small association with planning and organising, but this was a positive correlation and did not survive multiple correction. Overall findings indicate that greater RUMr was not beneficial to executive function, and it has less association with executive function than other types of mental health, repetitive negative thinking, and adiposity measures.

BRIEF and Cognitive Performance. When cognitive performance was compared based on self-reported executive function (low/high group), the group with high self-reported inhibition problems had more errors on the inhibition task, but this did not survive multiple correction. There was no effect of self-reported executive function on reaction time, and there was no significant effect of self-reported inhibition on the Simon Effect.

The weak association between inhibition performance and self -reported inhibition (contrary to prediction 3b) tends to indicate that the Simon Task and the self-reported executive function were not measuring the same constructs (Toplak et al., 2013). Garavan et al. (2002) examined the intricacies of the neurocognitive inhibition response in those who reported high or low (n=30) on another self-report scale – the Cognitive Failures Questionnaire (Broadbent et al., 1982). Like the current study they found no significant differences in reaction time between participants with and without self-reported cognitive problems. In construction of the BRIEF, Roth et al. (2005) took considerable effort to map their questions to the different domains highlighted in neurocognitive research and the BRIEF has concurrent/criterion validity in comparison with several other rating scales for

executive function problems, such as ADHD (Gioia & Isquith, 2011), however as a measure of cognitive problems we would expect to see a stronger association between the BRIEF scale and cognitive performance than mental health. This was not the case, in fact, in regression analysis the only significant predictor of self-reported inhibition was anxiety, and the only significant predictors of emotional control were anxiety and brooding rumination. Previous research concurs that BRIEF scores have a moderate to strong association with mental health outcomes, for example a study of executive function in adolescents (Gillespie & Rao, 2022) found that those with depression had the most executive function difficulties but there was no significant association between self-reported executive function and performance measures, or performance measures and depression. A study of executive function in veterans concluded that the BRIEF measured emotional distress rather than cognitive ability (Shwartz et al., 2020) however the study did not include detailed cognitive performance testing as a comparative. The inhibition and emotional control problems highlighted in the BRIEF scale therefore seem to be more relevant to mental health and repetitive negative cognition than cognitive deficits per se.

The lack of robust association between inhibition on the BRIEF and performance measures in the current study could have been due to ceiling effects on the task (being too easy). A further explanation could be low power due to the small size of the group who reported levels of executive function problems above the clinical cutoff. Alternatively, Rabin et al (2006; n=59) found that in a group of elderly people with neurologically verified mild cognitive impairment, the BRIEF was able to detect cognitive impairment but their scores on a neurocognitive battery were not clinically meaningful. They concluded that the BRIEF had greater sensitivity than

neurocognitive testing to identify mild cognitive impairment. This may also be the case in the current study – people with conditions such as depression and anxiety may therefore be experiencing more cognitive difficulties than most neurocognitive tests identify. A discussion paper (Toplak et al., 2013) suggests that neurocognitive performance tasks should be regarded as measures of processing efficiency in highly abstracted contexts. The influence of context on cognitive performance task should not be underestimated. Cognitive tasks are likely to elicit a high degree of motivation to perform well and the ACT attention theory (Eysenck et al., 2007) emphasises that extra cognitive resources can be recruited (in a time-limited fashion) to deal with such tasks. In daily living tasks people are likely to be coping with more competing thoughts and demands so it is very difficult to give singular, attention to every given task.

Measuring both cognitive performance and executive function in daily living is useful but neither method provides an ideal solution to understanding the nature of cognitive deficits related to health. Use of daily living measures that provide tasks that more closely match everyday problems could provide a better balance of observable performance and ecological validity. Anecdotally, after the cognitive performance tasks there were some participants who reported being extremely drained while others had no such issues. Further, participants who completed the tasks more quickly tended to report having a lot of computer game experience. Future investigations of computer-based cognitive performance may wish to record overall task completion times, how participants feel after the tasks and their level of game play experience to explore these as confounding factors in this style of cognitive testing. A further measure to consider is intra-participant variability which can help identify uneven performance profiles where the participant could have

average performance in the normal range but be using less-efficient cognitive processes (Marciano & Yeshurun, 2017; Phillips et al., 2013).

Strength and Limitations of Study 2

The study is novel in its attempt to reconcile mental and physical health effects and keeping an open mind to multiple avenues of association and causation. The study examines the links between several variables in a single sample to aid comparability between mental and physical health without running separate testing sessions. The multiple measures of the physical, mental health and cognition constructs allowed examination of both correlational associations between variables and between group differences for a more robust picture of the extant effects. As the investigation style was novel and as the extant research on the links between health and neurocognition are highly variable, each variable was examined thoroughly as a continuous and categorical measure. Binary categories were used to reduce the number of comparisons required but also to see if effects occurred at purported clinical cutoffs. Pairing continuous and categorical methods also helped identify type 2 errors that could have arisen if only the clinical cutoffs were used, (for example the association between BMI and left frontal lobe activity).

The neurological investigation was extensive, examining brain function in the resting state and during two cognitive tasks for the full sample. While the study numbers were small for an average ‘health’ study, the number of participants was large for an experimental neurological study. The study used a sample of young adults to avoid conflation of health-related executive function effects with normal aging while also utilising a combined university and community sample with less restrictively screening as a more representative group than many other studies e.g.

late adolescent/single gender/no co-morbidities. Demographic variables (including education, ethnicity, handedness and measures of general cognitive ability) were recorded to ensure that the sample was balanced and aid future investigators in understanding the type of participants who took part. The lack of demographic data in many weight-related studies has been cited as a problem for comparing data.

Neurovascular activity was examined in the resting state and during easy and more challenging task performance to get a broad view neurovascular differences between health groups. In addition, the analyses looked at regional differences, channel differences and functional connectivity for significant health groups. The functional connectivity matrices indicate a stark difference between the brain connectivity for high and low 'health' groups. The body of literature supporting fNIRS is still being built as evidenced by the wide range of findings on regions of interest and these exploratory observations add this literature to help understand haemodynamic differences related to waist circumference and rumination during the resting state and two levels of cognitive tasks (attention and inhibition). By examining resting state and task together this study also helps to draw attention to the different effects of health in each of these contexts which are rarely compared. There were also large benefits to the use of fNIRS for participant comfort, affordability and testing speed compared to a technique like fMRI. It is notable that since this thesis began there is a much wider range of fNIRS and health research to reference which illustrates the growing popularity of the technique.

Limitations

The executive function task was a less well-known inhibition task to investigate inhibition spatially, reduce the influence of verbal effects on the findings and add to the literature base. The disadvantage of the task was that the number of errors overall were very small, hence there were probable celling effects (more challenging tasks may have given more opportunity to see health-related differences between groups). This was also the case in an investigation by Ng (Ng et al., 2012). The multi-source interference task (MSIT) may provide a good option for a more challenging task requiring suppression of multiple sources of task interference, including Stroop, Erikson, and Simon tasks per Harrivel et al. (2013). The downside is that more task facets lead to more neurological complexities to disentangle.

The time-bound nature of the BOLD signal and its effect on post error brain activity (serial presentation effects) was not examined in this investigation and that may reveal more detailed information about health-related differences in participant performance. The block design of this task had limitations as the BOLD signal is sensitive to repeated presentations of the same or similar stimuli (Hall et al., 2016) therefore the cognitive tasks may have benefited from shorter task blocks and more frequent rest periods (allowing the signal to return to baseline). An event related design was rejected due to the need to give participants enough repetitions of the task to see an effect (Pilgrim et al., 2001; Plichta et al., 2007). Efforts were made to give participants enough task repetition to see robust effects, but short rest periods were a trade-off to make the overall length of the task suitable for participant engagement and comfort.

The evidence base for fNIRS scans is still in development, as are the processing procedures. Unlike fMRI, the study was only able to examine limited regions of interest in the brain and there are other important areas such as the

temporal-parietal junction the cingulate cortex and limbic regions that could help to shed light on the nuances of inhibitory control and health. The current study did however enable a detailed focus on the frontal and temporal networks under different rest and task conditions.

Negative affect and excess adiposity are both symptomatic of complex conditions with a range of interacting causal factors. There are other variables that are likely to be important in the interaction between physical and mental health including sleep patterns, nutrition and exercise that should also be investigated. The study was designed to be inclusive and did not exclude participants on the basis of neurodivergence or learning difficulties, but it may have been useful to collect data on this as the interaction between obesity and cognition is directly relevant to some conditions such as ADHD/ADD and there is still no conclusive explanation for this effect (Cortese, 2019; Cortese & Tessari, 2017).

As Study 1 illustrated the importance of time in seeing the effects of adiposity and negative affect on one another, a key limitation of Study 2 is its cross-sectional nature. Although the regression analysis can give some indication of predictive causation, follow-up studies would be essential to re-enforce the findings. It is also suggested that a hierarchical regression would generally have been the more appropriate in place of the forward stepwise regression as it was used to answer a theoretically motivated question i.e. to check the relative variance contributed to the mental and physical health outcomes in Chapter 7. Although the hierarchical regression and forward stepwise regression operate in a very similar way, a forward stepwise regression has the option to add previously excluded variables back into a model, whereas hierarchical regression has greater experimenter control. The drawbacks of forward stepwise regression (Smith, 2018) are less relevant to the

current investigation as the analysis used a small tightly controlled, theoretically motivated predictor entry, rather than being run to exclude nuisance variables, or develop a wholistic explanatory model for a condition. In general, the method used takes good account of issues identified by Petrocelli (2019), including consideration of the causal priority of variables. Mean centring the predictors as in Study 1, per Aiken and West, (1991; noted in Petrocelli, 2019) could have been considered.

A further strength of the study was consideration of the differences between continuous BMI and WC scores and their risk scores. Categories of BMI and WC were devised to reflect increased *health risk at different levels of adiposity*. This was initially for life insurance purposes. Most health applications use these categories to judge and communicate the likely risk of metabolic health complications to individual patients, but they are not a direct measure of health. Based on the extant literature, higher BMI appears to be related to select disadvantages in inhibitory control, however there are no guidelines on the cut-point where an increased mental health risk from adiposity would begin. WC is used as an indicator of visceral fat (in the belly area) which is also predictive of metabolic illness but appears to explain different variance than BMI. WC may also highlight fat-related metabolic risks in normal weight individuals. This is advantageous as the main drawback of BMI as a measure is a failure to identify individuals with excess body fat (false negatives) and in the context of this study, could highlight additional individuals who are at risk of mental health issues. As more detailed adiposity scanning methods are expensive and equipment can be difficult to obtain, bio-bank studies may offer an opportunity to investigate existing data. The role of circulating lipids has been hypothesised as a mode of action of adiposity on neurocognition. Some studies have found associations between free lipids in the blood and depression, (von Zimmermann et

al., 2020), however their relationship with body fat distribution is not straightforward, for example the relationship between central adiposity and blood lipids was not associated in a large biobank data study. Despite the lack of association between fat distribution and circulating body fat, there remains an association between body fat and increased inflammation biomarkers such as the interleukins. A very recent study has found that high blood pressure can cause T-cells (immune cells) in the outer brain (dura) to produce interlukin-17 activating macrophages which cause cognitive impairment (Santisteban et al., 2024). Blood serum levels of interlukin-17 had small effects on bodily cells, but it was local effects in the brain and cerebrospinal fluid that led to the damage and the researchers were able to halt the effects. This illustrates the importance of local bodily effects and the fact that global measurements are not always sufficient to understand mechanisms of effect. Future studies may consider other physiological measures of fats within the body.

Future Investigations

It would be beneficial to examine the role of repetitive negative cognition and physiological measures of adiposity. Additionally whole head fNIRS would be an advantage to incorporate the parietal lobe, or alternatively fMRI would allow us to examine the subcortical regions and brain pathways during tasks with more precise detail. Longitudinal studies may show different associations as the type of brain atrophy proposed to be associated with inflammation and excess fat would only be apparent over time. fNIRS investigations are amenable to follow-up studies as they have no health impact, take less time to set up, can be portable and do not cause the same level of discomfort or claustrophobic feeling as other BOLD signal measures such as fMRI. Stawarezyk et al.,(2011) used a combination of

neurocognitive scanning and SART task probe questions and found that greater activations of the Medial Pre-Frontal Cortex (MPFC) were related to reports of conscious external distractions and internal thoughts like mind wandering experiences unrelated to the task. Future executive function tasks may therefore want to include probes during the task to check participant reports of what they are thinking about in real time. This could help give more certainty to the assertions about what greater or lesser neural activations of different brain regions mean in practice. Future studies of adiposity could also consider body scans or blood lipid analysis to be give more certainty about which type of adiposity is related to negative affective issues. With growing research related to the Vagus nerve and in consideration of whether to include it in the model, it is suggested it would be important to examine heart rate variability and blood pressure patterns to see if health groups do in fact show variation in vagal nerve tone. These measures could also be examined in relation to attention and inhibition errors and the role of adaptation effects.

Study 2 focused on inhibition and sustained attention due to links with weight and negative affect-related effects, but there are other aspects of executive function such as cognitive flexibility/shifting and indeed working memory that are worthy of investigation. Episodic memory has also been implicated in depression and BMI-related cognitive deficits (Airaksinen et al., 2005) and this could be relevant to the adaptation effects that appear to underly some inhibition task errors observed in this study (Mayr et al., 2003). Better conclusions about the relationship between executive function in daily living and experimental cognitive performance could also help to further our understanding of the interaction between mental health and adiposity.

Conclusion

To answer the research question about mental and physical health, there seems to be good evidence that WC and negative affect are predictive of one-another, and both affect cognition. However, Study 2 findings provide evidence that the conditions are not showing the same type of neurocognitive effects. This means that the type of cognitive issues experienced by those who have high adiposity (more sustained attention errors, inhibition errors and adaptation effects) are different from those experienced by people with mental health and repetitive negative thinking symptoms (more inhibition errors and adaptation effects). Both effects are important, but the mechanism of effect is subtly different. This is reinforced by the different functional activity profiles during the task and during rest. Despite this difference repetitive negative thinking has been highlighted by this study as an important linking factor between mental and physical health. Rumination or worry scores and eventually patterns of fNIRS activation may help to identify individuals who are at greater risk of mental health problems and cardio-metabolic health problems as well as cognitive difficulties in neurocognitive testing and daily life. The comprehensive neurocognitive findings take an important step towards a holistic understanding of these comorbid conditions and their shared impact on health and cognition.

9.0 Overall Thesis Discussion

The current thesis aimed to investigate ways to integrate mental and physical health research and find promising avenues for future larger scale research. Study 1 showed that BMI and negative affect are predictive of one another over time. Study 2 provided detail about the neurocognitive nature of the association. Study 2 makes an important contribution in finding that repetitive negative thinking is a better indicator of interacting health and cognitive problems than either adiposity or negative affect scores alone. Apart from increased affective mental health symptoms and increased waist circumference, high brooding rumination scores were associated with more inhibition task errors (and adaptation errors), reduced neurovascular activity in the frontal and temporal lobe during task completion, plus increased neurovascular activity during the resting state that could impair restful recouperation.

A further contribution of the thesis is a theoretical model to help visualise and explain the interaction between cognitive and emotional neurocognitive processes that influence health and subsequent coping behaviour. The study findings support the model by confirming the importance of negative cognitions in the interaction between mental and physical health. Further investigations should examine whether negative cognitions are the cause of ill-health through chronically maintaining the stress response, or whether they are a byproduct of that stress response due to the brain using different pathways to process tasks in people who experience greater repetitive negative thinking.

Investigations within the thesis have further provided increased understanding of methodological factors in the investigation of mental and physical health, such as the diverse relationships between categorical and continuous measurement variables of the same construct. The diverse multi-disciplinary approach has helped

to identify that i)continuous measures of waist circumference (a proxy for visceral body fat), ii)worry and anxiety, and iii)categorical measures of rumination all explain variance in attention and inhibition processing. Additionally, in examining executive function in daily living, the study identified self-reported problems that are associated with different health measures which inform our understanding of the cognitive effects of different conditions. It is hoped that the 'joined-up' thinking within this investigation will help the formulation of future research priorities in cardiometabolic health, contributing to a more holistic understanding of health which can help innovate research and more effective treatments.

Reflections on the Research Goals

Lack of integration of biomedical understanding with psychology and psychiatric research was identified as a significant barrier to the translation of research into clinical practice (Schumann et al., 2014). The current reliance on behavioural observation and patient report are not sufficient to understand the complexity of mental health problems. Researching the effect of health conditions on the brain was identified as a priority, as well as finding ways to translate biomedical research (often based on small case studies at a cellular or individual level) and population level epidemiological research in a way that can be used to support evidence-based clinical practice that works for society and practitioners. To help address this lack of integrated understanding, the current programme of study used multi-disciplinary and multi-method research to get a fuller understanding of the relationship between two prominent mental and physical health issues: adiposity and negative affect via their neurocognitive substrates. Further, a model of mental and physical health was proposed that takes account of current neurocognitive

understanding and explains the link between emotional, cognitive, and behavioural pathways to worse and better health outcomes for both conditions.

Reflections on Study 1

Study 1 gives a detailed insight into the longitudinal effects of both loneliness and BMI in early adolescence (age 10-14), looking at bi-directional effects, and curvilinear effects that are often not considered. Strengths include the large population sample, rigorous data collection methods (rather than biased self-reported anthropometry) and longitudinal data. The data did not meet requirements for a full structural equation model, (which would have superior handling of error variance and better predictive qualities). However, a robust cross-lagged hierarchical regression analysis strategy was employed, including careful consideration of variance explained at previous time points, control variables and bootstrapping to verify the reliability of the findings. The study was successfully published (see Appendix D), illustrating its original contribution to the field.

Effect strength between loneliness and BMI was small (in the region of 1%). This may be expected with complex multi-causal issues such as obesity and loneliness over time, but there are variables that explain more variance, and could be more promising to follow-up. Additionally, although lonely adolescent girls increased in BMI over time, their mean loneliness was moderate, rather than high. Participants therefore did not seem to be very lonely in comparison to other studies (Yang et al., 2020). Alternative explanations for the finding could be a lack of sensitivity of the loneliness measure, or greater complexity of the socio-emotional effects at play. Income sufficiency was not related to either variable. Similar results

were found for another large Canadian cohort study (Johnson-Down et al., 1997). This may reflect a lack of sensitivity of the measure, although the purported link between household income and BMI has been questioned (Kim & von dem Knesebeck, 2018).

While this study utilised secondary data, it allowed us to make full use of a rich, high quality data set that has taken considerable time and resources to collect. A great deal has been learned about the handling of vast longitudinal data sets, including translation, the impact of missing data on sample sizes and transforming data, which has not been consistently collected or coded for across multiple time points. I completed valuable training in structural equation modelling, and selection alternative methods when data did not meet criteria for testing (advanced hierarchical regression; bootstrapping). Finally, the study afforded invaluable experience in the realities of collaboration with international research teams, and journal article writing and submission.

Reflections on Study 2

Study 2 sought to test key ideas in the model. It used a young adult sample (aged 25 to 40) in an ambitious joined-up study that utilised multi-disciplinary research methods to investigate potential links between negative affect and adiposity. The study found that repetitive negative thinking was an important factor in the link between adiposity, mental health and cognitive deficits including inhibition task errors. In discussion of the results in-relation to existing literature several avenues suggest that the action of the ACC (Anterior Cingulate Cortex) could be implicated in behavioural cognitive performance effects. The link with self-reported executive function problems was less clear and it could be that different

investigations that combine cognitive performance tasks with more externally valid contexts are needed to properly understand how health effects cognition.

The study had several advantages as it not only examined activity of the physical substrates (i.e. the tissues) that underlie cognition (via haemodynamic activity) but also examined self-reported problems in daily life, which have higher ecological validity. The study findings also indicate that negative affect explained more variance (anxiety explained 31% variance in inhibition and anxiety and rumination explained 51% variance in self-reported emotional control) and was as much a factor in self-reported daily living problems in behaviour regulation as adiposity. This indicates that mental health should be a given much higher priority in understanding individual's health and ways to maintain or restore wellbeing.

Methodological Insights

To help understand why significant outcomes vary for studies measuring adiposity, mental health and cognition, Study 2 examined different measures of each construct. There is controversy in the literature over the numerous measures of health and cognition (see Chapter 2) and the heterogeneity of study methods is often cited as a limitation of study findings. The relationship between several different measures of each construct were investigated (adiposity, mental health, repetitive negative thinking, and inhibition – see Chapter 2, Table 2 for an overview of the measures). This was done to find out which measures were showed the most robust effects and would be worthy of further study (Forsman et al., 2015). Loneliness was not measured in Study 2 as the effects were small and non-significant cross-

sectionally. Previous studies identified that rumination and depression were likely to explain more variance in overweight/obesity than loneliness, however it is acknowledged that including loneliness in Study 2 could have helped to confirm this assertion. Although the continuous and categorical measures of WC and BMI were highly correlated, they did not always show the same associations with other variables. For example, there was no effect of WC group on inhibition errors but there was an effect of BMI group, whereas the neurological effects were observed in relation to WCr (waist circumference risk) rather than BMI. Comparing different measurement types can give clues about the nature of the association between different variables. The effects related to waist circumference (more than BMI) could be attributed to the different physiological effects of adiposity depending on location/distribution in the body (Fox et al., 2007; Kurth et al., 2013), the cut points imposed by standard risk-related measures, or it could be a sign that the association between neurocognition and adiposity is mediated by another related variable. A similar effect was observed with the mental health measures and constructs – a significant difference in right temporal lobe resting state neurological activity was explained by worry (and to a lesser extent anxiety) whereas health-related effects on the inhibition task errors were predicted by brooding rumination (and to a lesser extent depression). Executive Function in daily living had different findings again; self-reported inhibition was predicted by anxiety and emotional control was predicted by continuous anxiety and rumination, yet categorical anxiety did not predict task performance errors. With so many possible ways to represent the independent and dependent variables it is easy to see why many studies end up with conflicting findings. Brooding rumination was the measure with the most consistently significant findings across health and cognition analyses, so it is a good candidate for further

study. When studies measure rumination, if they use the full ‘total rumination’ scores (which include reflective rumination) they may experience type 2 errors as questions that tap reflective rumination seem to have a weaker association with adiposity, mental health, inhibition errors and self-reported EF problems. Rumination measures or subscales that focus on negative valence rumination are therefore recommended for future health research. Further work to establish cut points on adiposity and scales that are most appropriate to mental health and cognitive health risk could also be an important area for future investigation.

Evolution of the CAMMPI Theoretical Model

What Study 1 Added to the Model

Findings from Study 1 of the reciprocal nature of the association between mental and physical health (negative affect and adiposity) and the longitudinal nature of the effects influence the cyclical nature of the model. Theories of the physical health effects of loneliness including hypervigilance and the role of chronic health effects such as persistent low-grade inflammation pointed toward a repetitive action that was maintaining the stress response in a subgroup of individuals. As stress is highly subjective, adaptive / maladaptive coping strategies are a clear way that behaviour could further influence health positively or negatively e.g. toward persistent negative thinking/ overeating or away from this mindset.

In Study 1 as the variance explained by loneliness was low (and the explanations for the mechanism of effect for loneliness on health was similar to other negative affective conditions) it was apparent that the investigation needed to consider negative affect more widely. This led to the inclusion of repetitive negative

cognitive as it could provide an explanation for the maintenance of the stress response over the long term in some individuals.

Methodologically, Study 1 raised several questions, including why the health effects develop in some individuals and not others, plus why the effects are so inconsistent from study to study. Apart from the extremely diverse measures of health, the idea arose that some individuals were being affected by transient deficits that occur despite a person appearing to be neuro/physiologically ‘normal’. One explanation that fit was cognitive load – in a limited resource system we can only give attention to so many stimuli, so perhaps some individuals were being overloaded with too much emotionally laden information that interfered with their ability to cope and execute adaptive coping strategies. Neurocognitive evidence was therefore sought to see if there was any evidence that cognitive overload could be the underlying mechanism of effect between reciprocal adiposity and mental health.

Prior to the conception of Study 2 the various explanations for the effect of negative affect on adiposity were used to create a proposed model to understand and integrate mental and physical health effects. Existing cognitive health models were investigated (see Chapter 3). The thoughts, feelings and emotions around ill-health were captured well in the five dimensions of the Common-Sense Model (CSM) of illness representation, (cause, consequences, cure/control, identity, and timeline; (Hagger & Orbell, 2003; Leventhal et al., 2016). The CSM serves as a type of filter for how people appraise threat relating to feeling ill which in turn affect their coping strategies and health outcomes. The dimensions consider both the concrete and abstract facets of health conditions and within the model, separate pathways are shown for emotional and cognitive appraisal processes which reflect different (but

linked) neural pathways (functional networks in the brain). These emotional and cognitive appraisals are akin to ‘hot’ and ‘cold’ cognitive processes seen in the literature on self-regulation (Hofmann et al., 2012), and fast and slow thinking processes outlined by Kahneman (2011) and they are central in the integrated model of physical and mental health in this thesis.

In evolutionary terms, hot and cold/fast and slow thinking processes equip us with both the ability to deal quickly with immediate threats (e.g., predation or a social threat or pain) through fast emotional responses and make thoughtful, planned responses to other more systematic challenges (e.g., how to access a hard-to-reach food source, or negotiate with a tribe member). The type of thought process we access influences the way we react – the coping strategies we use (Schumann et al., 2014), and the CSM holds that the way we deal with ill-health follow the same process. Perceptions of ‘hot’ threats tend to produce a flight, fight, freeze style response such as emotional outbursts (externalising behaviour) or avoidance, whereas ‘cold’ challenges can be dealt with through goal relevant problem-solving. Supporting this conceptualisation there is evidence of neurological differences in the way we process hot and cold problems and reward, and motivational issues (highly relevant to mental and physical health) are grouped within the hot or emotion-laden category (Salehinejad et al., 2021).

The proposed CAMMPI model took ideas from the CSM about coping with ill-health combined with current understanding of neurocognitive processes and applied them to the *experience* of well-being or ill-health to aid an integrated understanding of physical and mental health. This integrated (transdiagnostic) explanation for the way that health outcomes can get better or worse depend on three key areas: *physical attributes* (levels of nutrients, hormones, neurotransmitters, cell/tissue

health etc.), *emotional feelings*, and *thoughts* (influenced by the current context and past experiences). In simplistic terms, our experience of well-being or ill-health is a combination of these areas, which influence how we behave. The same three areas are relevant for both physical and mental health conditions, for example someone may have broken a bone in their leg (causing physical tissue damage), but their thoughts and emotions will also play a part in their outlook, pain perceptions and physical healing process. Physical and mental pain have been shown to affect the same area of the brain, the cingulate cortex, therefore overlapping with an important area for cognitive control (Shackman et al., 2011). As with a physical health problem someone with a mental health condition is experiencing pain and the source is also a combination of emotions and thoughts plus underlying physical attributes (nutrients, hormones, neurotransmitter levels or cell/tissue health) – but the physical substrates are not as easy to see and the source of mental or psychological pain is more difficult to point to.

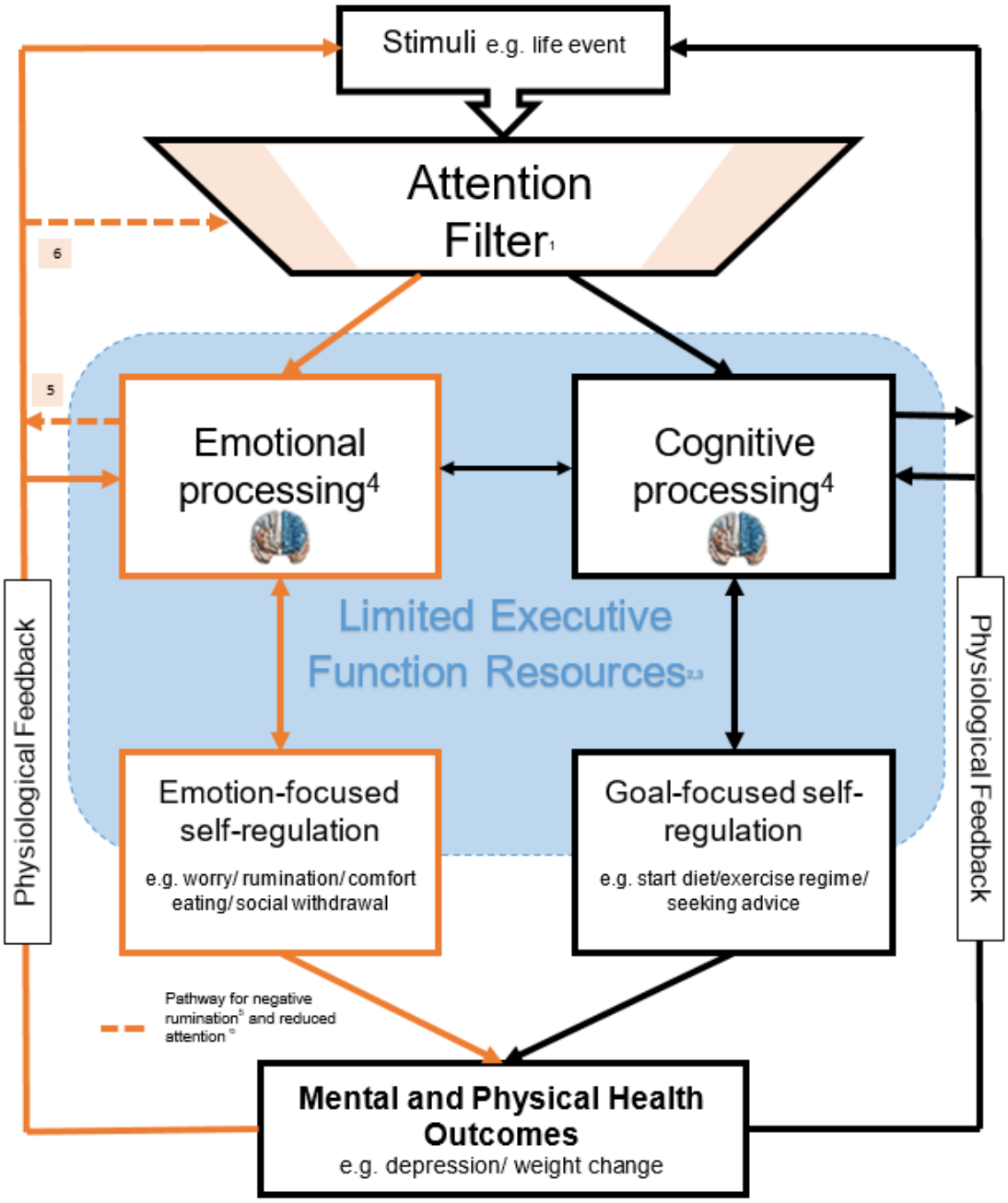


Figure 72
CAMMPI Model Version 1

What Study 2 Added to the Model

A core feature of the CAMMPI model is the separate neurocognitive processes that occur when a person is reacting based on emotion compared to when they are acting based on calm thoughtful decision-making. Study 2 compared

neurocognitive activity and performance with participants with and without affective symptoms. The study was not able to investigate the effect on the deeper subcortical emotion-focused areas of the brain but being high in RUMb appeared to have more influence on executive function than having high affective disorder scores such as depression or anxiety. RUMb also predict more variability in adiposity than clinically significant depression/ anxiety scores. The study confirmed that cognition is relevant to the link between mental and physical health, but negatively valanced thought processes show the strongest link between the conditions.

One of the key findings of Study 2 is that Brooding rumination (RUMb) is part of an important pathway in mediating between physical and mental health, and the same pathway is also important to executive function issues in performance and everyday living. Rumination, and to a lesser extent waist circumference, appear to effect cognitive performance, brain activity and functional connectivity in areas of the frontal and temporal cortex. RUMb is therefore worthy of further investigation as a process to explain or show an important process that links physical and mental health. Based on the findings of Study 1 and 2 and related research, the CAMMPI model is discussed and updated below (see Figure 73 Cognitive-Affective Model of Mental and Physical Health Interaction (CAMMPI): Version 2).

During Study 2 the frontal cortex (superior and inferior frontal lobe) was examined due to its ubiquitous association with executive function. Areas of the default mode network (medial frontal cortex and medial temporal lobe) were examined due to their links with internal processing and the deeper emotional centres in the brain (limbic loop – see Figure 5). The study examined whether mental

and physical health conditions (depression, anxiety, and excess adiposity) shared similar deficits in executive function by looking at task performance and neurological activity in these areas. The theoretical model led to predictions that those who were high in adiposity and depression would display more cognitive errors, and this would be paired with less brain activity in the frontal cortex and /or greater activity in the internal/emotional processing areas of the brain. This would indicate that cognitive resources were being diverted away from the usual areas involved in executive function.

It appears that repetitive negative thinking is a byproduct of being in this hypervigilant mindset and due to i) attention being automatically focused on negative or potentially threatening stimuli and ii) use of different neural pathways that are primed to quickly process and remember threatful stimuli. This diverts processing away from the slow, thoughtful, higher cortical evaluation of the frontoparietal network and away from recognising less urgent bodily signals (e.g. hunger, satiety, tiredness). This faster emotional processing network (see Figure 72) effectively filters out positive/neutral experiences and reduces attention to subtle bodily signals like interoception that help us to notice how we are feeling e.g. feeling full, feeling uncomfortable from sitting in an awkward position. It is suggested that over time failure to attend to these signals affect our health. In relation to obesity, the assertion that failure to attend to subtle bodily signals affects health is supported by genetic research indicating genes that sense when we are full are strongly implicated in childhood obesity (Carnell & Wardle, 2009; Llewellyn & Fildes, 2017) but the action of the gene has not been expressly linked to emotional response pathways.

Although the neurological findings of Study 2 are exploratory, the picture emerging is that repetitive negative thinking (associated with greater negatively

valanced emotional processing) is linked to more inhibition task errors. The type of errors (on both congruent and incongruent trials) and the pattern of brain activity (medial frontal and temporal areas) appear consistent with participants defaulting to 'fast and dirty' processing pathways (via medial areas like the anterior cingulate cortex) to guess what stimuli will come next (based on previous learning), rather than sacrifice processing time to wait for their brain to signal which action to take. This itself could be regarded as an impulsive action – precisely in keeping with the nature of inhibition difficulties.

Results of Study 2 indicate that the propensity for repetitive negative thinking is a more important link between mental and physical health than depression or anxiety (at least Study 2 indicates this is true in the case of obesity, inhibition. and self-reported executive function). It remains to be established whether repetitive negative thinking exerts extra cognitive load on executive function to cause inhibition errors, or whether repetitive negative thinking is symptomatic of the state of chronic arousal/repeated activation of emotional processing networks (including the anterior cingulate cortex) and failure to suppress previously learned information. As repetitive negative thinking is associated with hypervigilance and the stress response, one hypothesis could be that it has a physiological root in the autonomic stress response (ANS). The Vagus nerve is an important but often neglected part of the ANS and has a role in our perception of interoception and a means to bypass interoception when the body is in a stressful state (Prescott & Liberles, 2022) so rather than only focusing on the effect of stress-hormones, it may be beneficial for future studies to examine the action of the nerves within the ANS and their influence on neurocognition and health.

Marker of Vulnerable Individuals. Study 2 found that at rest, repetitive negative thinking (continuous worry and categorical RUMb) is associated with greater right temporal activity, and greater functional connectivity between the frontal and temporal lobes. In contrast, during task completion frequent repetitive negative thinking is associated with less activity in the left frontal and right temporal lobe, weaker functional connectivity, less synchronised activity, and greater reliance on the right frontal lobe. If this is validated in future studies, this pattern of activity could be used as a biomarker of repetitive negative thinking and being in a state of increased emotional processing which could identify individuals at increased risk of future mental health and cardio-metabolic problems. These individuals may benefit from interventions to calm the SNS (such as those used in Somatic Therapy/ breathing techniques, and EMDR).

Using Reflections on Study 2 to Improve the CAMMPI model: a Missing Piece in Explaining the Response to Threat

In most theories of hypervigilance to stress, the role of stress hormones and neurotransmitters which ramp up the stress response are emphasised (hypothalamus and adrenal glands), as well as the link to adrenal fatigue and chronic low-grade inflammation. However, the role of specific nerves in the stress response are less well discussed. The Vagus nerve is a key part of the ANS – more specifically the parasympathetic nervous system which signals the body to calm down after a stressful experience. It innervates the organs including the heart, lungs and digestive system and brain influencing heart rate, breathing rate, blood vessel dilation and hormone secretion, to bring the body back into a state of ‘rest and digest’ (Capilupi et al., 2020). However, there is growing recognition that this ‘return

to normal' effect is not a certainty and can easily become disrupted (Payne et al., 2015). This lack of return to baseline could be as damaging to health as repeated activations of the stress response by the sympathetic nervous system, and both states affect cognitive performance and brain function (Forte et al., 2019; Thayer & Lane, 2009). This means issues with the proper function of the Vagus nerve could be an alternative explanation for repetitive negative thinking and the cognitive-health effects observed in Study 2.

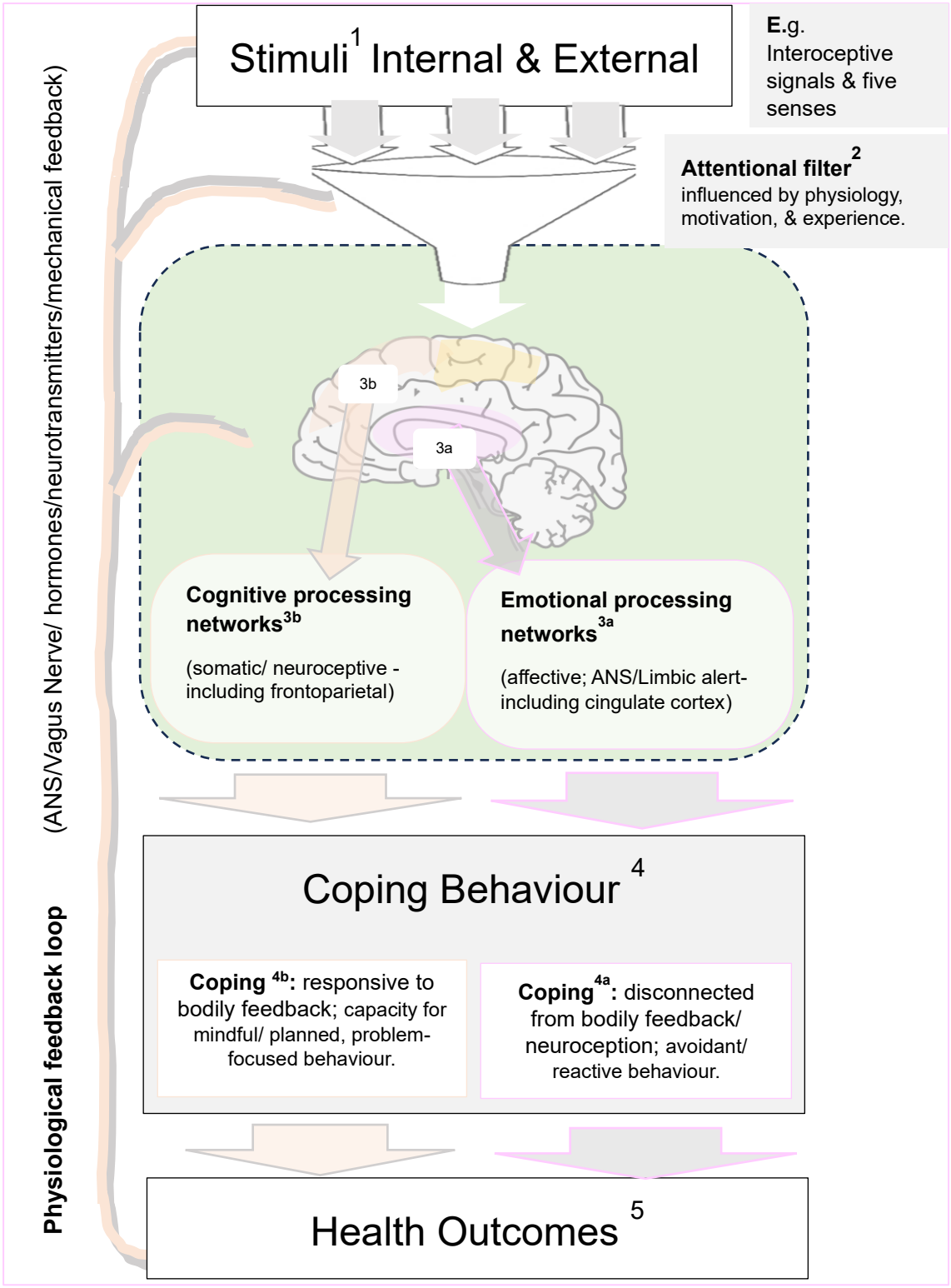


Figure 73
Cognitive-Affective Model of Mental and Physical Health Interaction (CAMMPI): Version 2
***Note*:** [1-5]=Mental and physical health outcomes suffer due to:

- Attention filter is narrowed by emotional processing systems due to hypervigilance /feelings of threat increasing negative thinking bias and activating emotional processing systems.
- Negative thinking bias leads to repetitive negative thinking which further increased perceived threat.

- Emotional processing systems remain activated through physiological feedback: chronically elevated SNS, depressed PNS and/ reduced vagal tone (somatic, nervous, and neuroendocrine alert systems) leading to chronic low-grade inflammation.
- Unconscious emotion-based coping behaviour to feel better rather than slow thoughtful cognitive processing to problem-solve/ address underlying needs.

Suggested Preventative Interventions to Support High Risk Individuals

A study by Rosenbaum et al., (2021) combined fNIRS with Electronic Momentary Assessment (EMA) and highlighted that recurrent rumination was specifically related to socially-mediated stress. This indicates that interventions that deal with negative social cognitions (such as those developed for loneliness), coupled with measures to reduce internal thoughts should be useful interventions to explore. As depression and excess adiposity often begin in childhood, studies using these techniques would be a benefit to introduce to children along with strategies to affirm individual worth and self-esteem. In addition, the genetic propensity for negative affective conditions and adiposity should not be ignored, so it is extremely important that measures are taken to reduce blame and stigma around these conditions.

Given the current findings high ruminators appear to be an at-risk group for both mental health and adiposity-related conditions (such as type 2 diabetes, heart problems, hypertension etc.). Interventions that reduce internal processing were identified to help affected individuals address executive function problems in daily living and reduce their risk of future adiposity and mental health conditions that result long-term. As repetitive negative thinking is regarded as a symptom rather than a condition there are no direct therapeutic pathways for this, but perhaps this should change. Consideration should be given to trialling techniques that could provide early intervention for negative ruminative thinkers. Looking at conditions where repetitive

negative thinking is an important feature (depression, anxiety, PTSD and possibly OCD) the National Institute of Clinical Excellence recommend therapies such as Cognitive Behavioural Therapy and trauma-focused CBT and Eye Movement Desensitisation and Reprocessing (EMDR). CBT is a talking therapy that involves changing the way a person thinks about issues. Both therapies appear to influence the same brain areas (the limbic system) to reduce the negative valence of stressful or traumatic events (Santarnecchi et al., 2019). EMDR has several advantages over CBT as it takes less time to implement and does not rely on the individual being able to verbalise and work through the nature of their problem or do tasks outside of therapy sessions which are known factors that reduce CBT compliance/increase drop out.

A further option to help address repeated negative thinking is mindfulness training. Mindfulness and attention control related techniques have shown promise in improving Default Mode Network function, reducing negative cognitions and emotional eating (Dunn et al., 2006; Egan et al., 2021; Fergus & Wheless, 2018; Laicher et al., 2023). A drawback of mindfulness like some CBT techniques is the time and willpower it takes to learn and become effective in using the strategies in daily life. These factors reduce adherence and ultimately effectiveness of the therapy. As the thesis findings show, inhibition errors and their neural substrates are important in the link between adiposity and mental health therefore interventions that require willpower would place the recipients at an immediate disadvantage.

EMDR is a different type of therapy which uses bilateral body and eye movements to reset the Vagus nerve back to a state of ‘rest and digest’ whilst calling stressful experiences to mind. Research has also been conducted examining the utility of wearable devices that stimulate the Vagus nerve directly as a treatment for

mental health conditions (Bremner et al., 2020). These techniques are faster to implement, do not require as much skill /learning, and rely less on good verbal articulation, so they may be more promising. EMDR still has the drawback of requiring a trained therapist to implement (entailing expense and time) which is not likely to be implemented as a measure to prevent possible ill-health. The neurological and nervous effects of bilateral body and eye movements as exercises could therefore be investigated.

Incorporating the Vagus Nerve in the CAMMPI Model

The Vagus nerve communicates between the amygdala and prefrontal cortex. According to the polyvagal theory of safety (Porges, 2009, 2022) Vagus nerve stimulation can help to move individuals out of an emotional 'fight or flight' mental state into a calm state, so normal executive function processing can resume (Woody et al., 2014). The effect of the Vagus nerve can be incorporated into the theoretical model as it pairs with stress hormones as an essential part of the physiological pathway that switches an individual back from a fast negative emotional, hypervigilant state and mindset (linked in this study to more inhibition errors) to a calm state with slow thorough cognitive processing (see Figure 73).

The evolutionary focus on safety taken by Polyvagal theory is interesting as it forms an underlying reason for the existence of the separate (but interconnected) 'emotional' and cognitive processing structures outlined in the CAMMPI model. Emotional or affective pathways in the brain and body allow fast reactive responses in the face of perceived danger, whereas we also have more thorough 'neuroceptive' pathways that allow greater cognitive flexibility when the person feels safe enough to

exercise it. Payne et al. (2015), cites work by Gellham (1967) on ergotrophic (sympathetic) and trophotrophic (parasympathetic) systems that respectively mediate alerting systems such as the fight / flight and rest feeding and recuperation systems. This is somewhat simplistic as the parasympathetic nervous system is also involved in extreme stress responses (freeze/immobility, dissociation/shut down). Payne et al., (2015) terms the somatic, CNS and neuroendocrinal systems involved in the high alert system as the 'Core Response Network' (The ANS, Reticular Arousal System, Limbic system, and Emotional Motor system) and that these are primarily affective systems. Vagal nerve tone offers a means to help calm and reset the CNS back to baseline. The frontoparietal network is engaged during calm processing whereby individuals are more able to tune in to their interoceptive signals (such as breathing/ heart rate). Further the authors note that that stimulation of the ventral branch of the Vagus nerve can help to calm the sympathetic nervous system through social engagement including eye contact and verbal interaction. This is interesting due to the observed link between repetitive negative thinking and loneliness. Those who are lonely may experience fewer opportunities for this calming social engagement than they would prefer so their SNS remains elevated contributing to negative health effects over time.

The theory of neurovisceral integration (Thayer & Lane, 2009) provides further support for the separate cognitive and emotional processes outlined in the current proposed model and implicates the action of the Vagus nerve on neurocognition in creating negative thinking biases. The research indicates the Vagus nerve is a likely candidate for controlling the 'filtering' process explained in the CAMMPI model, whereby attention to external and internal stimuli are restricted. The extent of the time spent in a negative emotional mindset will be influenced by a person's genetic

propensities for hypervigilance and/ or trauma experiences, and therefore, the extent of their cognitive filtering process and subsequent learned behaviours including coping strategies.

Implications of the Research

Adiposity and affective mental health conditions are extremely prevalent, especially since the COVID-19 pandemic. Current treatments do not seem to address the underlying conditions effectively; most negative affective conditions are under detected and not successfully treated (Forsman et al., 2015) and most weight loss is not sustained over time (Kheniser et al., 2021). Taking creative approaches, such as looking at potential mind-body interactions could afford a way to break away from the limited scope of traditional methods.

The CAMMPI model offers a parsimonious account for the shared variance between the conditions; a bias toward negative emotional brain processing pathways (such as the anterior cingulate cortex), rather than slower cognitive processing (via the inferior and superior frontal lobes). Use of these pathways appears to maintain fast responses based on previous learning but results in more inhibition error. This style of thinking likely extends to daily living and the tendency to resort to simple well learned emotion-based coping strategies rather than being able to generate or enact problem-focused solutions. These coping strategies result in less adaptive health behaviour. Paired with an associated tendency for repetitive negative thinking, the emotional neurological pathways maintain the stress response and contribute to worse health over time. Further investigation is needed to verify this explanation.

Suggestions are made here for preventative interventions that could be explored to help to address repetitive negative thinking before mental and physical

health problems develop. See Appendix R, Areas for Future Study: Vagal Nerve Tone and Heart Rate Variability for further discussion of a likely association between the observed health and cognition findings in this study and function of the Vagus nerve. Brooding rumination and Heart Rate Variability (controlled by the Vagus nerve) could be promising variables to examine in future work related to negative affect, obesity and cardiometabolic health-risks and the neurocognitive effects highlighted in this thesis.

More knowledge of executive function difficulties and the impact of attention and inhibition in certain health conditions could have considerable implications for treatment and health regulation strategies. For example, increasing understanding of the real-life impact of cognitive issues associated with mental health conditions, influencing the argument for increased societal health interventions rather than reliance on individual accountability (e.g., in food-related regulation). The cognitive findings could also improve our understanding of the impact of marketing strategies (Folkvord & Hermans, 2020) on at-risk groups. Research suggests that improving awareness of the multiple causes of obesity promotes greater acceptance of societal prevention policies (Beeken & Wardle, 2013). This is highly important as obesity is an overlooked area of bullying and bias (Puhl & Heuer, 2010) that worsen the condition through increasing stress and impairing self-esteem. The current thesis illustrates that mental health affects physical health, but it is difficult to move away from socio-cultural values that give visible physical health outcomes a priority. Comparison of the effects of physical health and mental health (like this thesis) and investigation into the optimal cutoffs where adiposity, negative affect and rumination affect cognition may help to quantify and increase understanding of the relative importance of mental and physical symptoms. Quantitative data on integrated health

effects (linking mental and physical outcomes) may provide better arguments to address disparities in resources and care, as well as highlighting individuals with increased support requirements that would have otherwise been missed.

Integrating thinking on mental and physical health gives new perspectives for research. The current symptomatic diagnoses patterns for mental health conditions would benefit from revision to help map them more closely to cognitive and physical health outcomes rather than hierarchical topologies of mental health alone. Predictive modelling of health symptoms could be beneficial in identifying clusters of mental and physical symptom patterns which has previous been too complex to achieve. Integrated approaches such as transdiagnostic models could be expanded to support more holistic health research methods. Health research is more likely to be translated into practice when it involves people who can action the findings, so unified models of the interaction between mental and physical symptoms could help to explain the overlap to healthcare professionals. More work needs to be done in making the argument that mental health *is* physical health and that the medical model is not the only valid approach to treatment/care.

Conclusion

This thesis addresses the poor understanding of the links between mental and physical health variables. Due to complexity and out-dated or biased thinking public and practitioners fail to see mental health conditions as somatic health problems. To avoid misguiding future research and funding away from impactful study areas more studies should experiment with joined up cross-disciplinary thinking. Cross disciplinary investigations using robust methods holds promise for furthering our understanding of health and providing innovative whole-body solutions for conditions

like depression where little progress has been made toward treatment in decades. The ROAMER research project (Forsman et al., 2015), outlined in Chapter 1, perfectly illustrated the power of cross-sector collaboration in moving toward better healthcare. Transdiagnostic theory is being used to help improve the dialogue between different disciplines, but continued work needs to be done to push boundaries and move research out of single discipline silos.

The large neurocognitive study reported in this thesis examined the effect of adiposity and negative affect on cognitive performance and found evidence that rumination and waist circumference are useful predictors of cognitive errors, self-reported executive function problems, and brain function differences in resting state and task. Future work based on the CAMMPI model should include multi-disciplinary investigations of the neurocognitive decision-making pathways of those with high rumination and adiposity during daily living tasks to see if individuals do default to fast heuristic cognitive processes and whether this is influenced by ANS nerve function. The potential for high rumination as a biomarker of future cardiometabolic health risks should also be investigated longitudinally.

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APPENDICES

Appendix A Ethics & Consent

A1 Ethics Approval Study 1

Date 11 April, 2017

Pamela Qualter/Ruth Hurley/Alice Eccles
School of Psychology
University of Central Lancashire

Dear Pamela, Ruth and Alice

Re: PSYSOC Ethics Committee Application
Unique Reference Number: PSYSOC 348

The PSYSOC ethics committee has granted approval of your proposal application; Do lonely young people eat more and move less? Cognitive mechanisms and protective factors in loneliness and obesity. *(RHurley).* Childhood Loneliness: Health outcomes and Social Cognition Intervention.*(AEccles).* Approval is granted up to the end of project date.

It is your responsibility to ensure that

- the project is carried out in line with the information provided in the forms you have submitted
- you regularly re-consider the ethical issues that may be raised in generating and analysing your data
- any proposed amendments/changes to the project are raised with, and approved, by Committee
- you notify roffice@uclan.ac.uk if the end date changes or the project does not start
- serious adverse events that occur from the project are reported to Committee
- a closure report is submitted to complete the ethics governance procedures (Existing paperwork can be used for this purposes e.g. funder's end of grant report; abstract for student award or NRES final report. If none of these are available use e-Ethics Closure Report Proforma).

Yours sincerely

Emma Bray
Deputy Vice Chair
PSYSOC Ethics Committee

* for research degree students this will be the final lapse date

A2 Ethical Approval Study 2

30 July 2018

Janice Abbott/ Ruth Hurley
School of Psychology
University of Central Lancashire

Dear Janice / Ruth

Re: PSYSOC Ethics Committee Application
Unique Reference Number: PSYSOC 348 Study 2

The PSYSOC ethics committee has granted approval of your proposal application 'Investigating the Influence of Body Size on Attention'. Approval is granted up to the end of project date.

It is your responsibility to ensure that

- the project is carried out in line with the information provided in the forms you have submitted
- you regularly re-consider the ethical issues that may be raised in generating and analysing your data
- any proposed amendments/changes to the project are raised with, and approved, by Committee
- you notify EthicsInfo@uclan.ac.uk if the end date changes or the project does not start
- serious adverse events that occur from the project are reported to Committee
- a closure report is submitted to complete the ethics governance procedures (Existing paperwork can be used for this purposes e.g. funder's end of grant report; abstract for student award or NRES final report. If none of these are available use e-Ethics Closure Report Proforma).

Yours sincerely

Emma Threadgold
Deputy Vice Chair
PSYSOC Ethics Committee

* for research degree students this will be the final lapse date

NB - Ethical approval is contingent on any health and safety checklists having been completed and necessary approvals gained as a result.

A2 Study 2 Consent form

Pg 1 of 2

Investigating the Influence of Body Size on Attention: CONSENT FORM

Ruth Hurley
PhD Researcher and Graduate Teaching Assistant
rhurley2@uclan.ac.uk

Please read the following statements; initial the boxes and sign the form to indicate your agreement.

		↓Your initials↓
1.	I confirm that I have read and understand the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	☐
2.	I understand that my participation is voluntary and that I am free to withdraw at any time up until I leave the room where my last measures have been taken.	☐
3.	I agree to take part in the above study.	☐

Name of Participant:	**Signature:**	**Date:**
.....	.....	.....
Name of Researcher:	**Signature:**	**Date:**
.....	.....	.....

PTO →

Investigating the Influence of Body Size on Attention: CONSENT FORM

If you would be willing for us to **contact you in future** please leave your name, and **either** an email address or postal address below, then tick your contact choice/s:

NAME:

EMAIL:

or

ADDRESS:

Contact choices:

☐ I would like to receive an overview of the findings of the study

☐ I would like to be contacted if you need participants for follow up/future studies

Appendix B Debrief

School of Psychology
Darwin Building
University of Central Lancashire
Preston PRI 2HE

Investigating the Influence of Body Size on Attention

Thank you for taking the time to complete this study, your participation is greatly appreciated.

Previous research has indicated that there may be a link between weight status (such as grades of BMI and waist circumference) and our ability to perform certain cognitive tasks such as focusing attention and inhibiting distracting information. There is some evidence that this may affect the activity of certain brain areas.

In the study, the cap that we placed on your head measured areas of increased oxygen in various parts of the brain. Specifically, we were looking at regions that are involved in attention and inhibition. Brain area that are more active than others need a greater supply of oxygen, and we are examining whether there is more activity when you perform a task that requires attention and decision making skills compared to one that doesn't.

You filled out a questionnaire designed to measure your reported levels of attention and other thinking skills in everyday life. We also asked you to complete measure of your mood and emotion (including feelings of depression, anxiety, worry and whether you experience ruminative thoughts). This is because some researchers have suggested that these types of thought processes could also influence our attention.

In this research, we hope to understand more about which areas of the brain are more active during attention and inhibition tasks, and whether this varies for different people.

You will never be identified in any presentation of the findings of this study, and it will not be possible to link the results back to you. All data collected will be stored in a locked filing cabinet, and all electronic data will be held on a password protected computer. It is intended that the

Appendix C Third Variables

A series of third variables (not exhaustive) was identified within the literature. The potential relationship of these third variables with the main variables in the study (obesity, negative affect, and response inhibition) are considered in the table below. The relationship between these variables is examined bi-directionally, hence variables are considered as an IV and as a DV. These are potential theoretical relationships, not based on structural equation modelling (see Chapter 2)

Table C1 Potential Third Variables and the Nature of their Association with the Main Study Variables:

Potential third variables	Nature of associations **(**with the main study variables**)**	Obesity & Negative Affect **Bi-directional Relationships**		Negative Affect and Inhibition Bi-directional Relationships		Obesity and Inhibition Bi-directional Relationships	
		Effect of obesity on negative affect	Effect of negative affect on obesity	Effect of negative affect on inhibition	Effect of inhibition on negative affect	Effect of obesity on inhibition	Effect of inhibition on obesity
Age Potential Mediator/Covariate (developmental effects in childhood and age-related decline in middle to late adulthood).	**Obesity**. Tendency to increase BMI with age (NICE, 2014). **Negative Affect**. Bidirectional increased risk of physical and mental health problems (e.g. depression and obesity) with age and aging in middle and older age groups (Gao et al., 2023). **Inhibition**. Fully developed at 25, and declines with age (most markedly after around 60 years (Epp et al., 2012).	Should be consistent relationship, but there could be a more pronounced effect in adults with older age.	Should be consistent relationship, but there could be a more pronounced effect in adults with older age.	Should be consistent relationship, but there could be a more pronounced effect in adults with older age.	Should be consistent relationship but there could be a more pronounced effect in adults with older age.	Should be consistent relationship, but there could be a more pronounced effect in adults with older age.	Should be consistent relationship but there could be a more pronounced effect in adults with older age.
Sex at Birth	**Obesity**. Slightly more females are obese than males	Some evidence that females are more likely to	Small sex differences in effects (females	No known sex effects. otential link if females are	No convincing sex effects.	No known effects of sex.	No known effects of sex.

Potential moderator/ confounding variable.	(29%;27%); complex interactions with sex and a variety of social and physiological factors e.g. marriage, menopause (Cooper et al., 2021). **Negative Affect**. Females have higher rates of negative affect than males (potentially due to social adversity). [NOTE: **Inhibition**. No convincing sex differences. Stroop task shows better performance for females due to superior verbal skills in colour naming abilities]	think negatively about being obese (societal pressure).	may show more pronounced effects).	at a greater familial risk of MDD (Stevens et al., 2023).

Potential **T**hird **V**ariables	**Nature of Associations** (with the main study variables)	**Obesity & Negative Affect Bi-directional Relationships**		**Negative Affect and Inhibition Bi-directional Relationships**		**Obesity and Inhibition Bi-directional Relationships**	
		Effect of <u>obesity</u> on negative affect	Effect of <u>negative affect</u> on obesity	Effect of <u>negative affect</u> on inhibition	Effect of <u>inhibition</u> on negative affect	Effect of <u>obesity</u> on inhibition	Effect of <u>inhibition</u> on obesity
Ethnicity Potential confounding variable	**Obesity**. White ethnicities appear to show health effects of obesity at higher BMI than some non-white groups e.g., South Asians (NICE, 2013). **Negative Affect**. Black women experience higher rates of depression than white women (no difference in	No known effects of ethnicity.	No known effects of ethnicity.	No known effects of ethnicity.	No known effects of ethnicity.	No known effects of ethnicity.	No known effects of ethnicity.

	males, NHS Digital (2017) [likely socially mediated].						
Socio-economic status Potential moderating variable (inconclusive)	**Obesity**. Lower SES linked to higher obesity rates. **Negative Affect**. Lower SES linked to higher mental health problems [potentially affects the relationship between depression and brain volume (Johns et al., 2025)]. **Inhibition**. Negative association in childhood, little information in adulthood (Ferguson et al., 2021).	SES/ education may partially explain variance in a relationship.	SES/ education may partially explain variance in a relationship.	SES/ education may partially explain variance in a relationship.	No known effects of SES.	SES/ education may partially explain variance in a relationship.	No known effects of SES.
Psychoactive substances (acute effects) Potential Confounding variable	**Inhibition**. Affected by stimulant/ depressive substances.				Potential confound.		Potential confound.

Potential third variables	Nature of **A**ssociations **(**with the main study variables**)**	**Obesity & Negative Affect Bi-directional Relationships**		**Negative Affect and Inhibition Bi-directional Relationships**		**Obesity and Inhibition Bi-directional Relationships**	
		Effect of <u>obesity</u> on negative affect	Effect of <u>negative affect</u> on obesity	Effect of <u>negative affect</u> on inhibition	Effect of <u>inhibition</u> on negative affect	Effect of <u>obesity</u> on inhibition	Effect of <u>inhibition</u> on obesity
Handedness No conclusive third variable effects.	**Inhibition**. No conclusive differences behaviourally, possible effects on verbal inhibition tasks (Marakshina et al., 2017); possible lateralised effects on brain responses (Cherbuin et al., 2011).	No known effects.	No known effects.	No known effects.	No known effects.	No known effects.	No known effects.
General Intelligence No conclusive third variable effects of IQ on health or inhibition.	**Obesity**. Some evidence that general intelligence affects development of obesity in children and education level affects maintenance of obesity in adulthood (Yu et al., 2010). **Inhibition**. General intelligence and inhibition develop together in children. Fluid intelligence does not explain inhibition control in adults (Martin et al., 2021) but may moderate reaction time.	No known effects.	No known effects.	No known effects.	Could moderate reaction time.	No known effects.	Could moderate reaction time.

Appendix D Study 1 Manuscript

Reciprocal Prospective Relationships Between Loneliness and Weight Status in Late Childhood and Early Adolescence

Pamela Qualter*, Ruth Hurley, Alice M. Eccles, Janice Abbott, Michel Boivin, & Richard E. Tremblay.

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https://doi.org/10.1007/s10964-018-0867-9

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This study uses data from the Quebec Longitudinal Study of Child Development (QLSCD). Data collection for the QLSCD was made possible through funding from the *ministère de la Santé et des Services Sociaux (MSSS)* (Ministry of Health and Social Services), the Lucie and André Chagnon Foundation, the *ministère de la Famille* (Ministry of Family), and the *Institut de la statistique du Québec (the Institut)*.

Authors' Contributions

PQ conceived the study, performed statistical analyses, interpreted the findings, drafted the manuscript, and made revisions to the manuscript based on reviewer feedback; RH and AME helped to perform statistical analyses and interpret the findings, and contributed to the draft manuscript; JA, MB, and RET participated in the study design, and edited the manuscript; MB and RET coordinated the original data collection. All authors read and approved the final manuscript.

Data Sharing Declaration.

The dataset analyzed during the current study is not publicly available, but it is available from Institut de la statistique du Québec (Information and Documentation Centre, Institut de la statistique du Québec, 200, chemin Sainte-Foy, 3e étage Québec (Québec, G1R 5T4).

Conflicts of Interest

The authors report no conflict of interests.

Compliance with Ethical Standards

Ethical Approval

All procedures were in accordance with the ethical standards of institutional research committees and with the 1964 Helsinki declaration and its later amendments or comparable

ethical standards. This article does not contain any studies with animals.

Informed Consent

Informed consent was obtained by the all individual participants included in the study.

Abstract

Adolescents who do not conform to weight ideals are vulnerable to disapproval and victimization from peers in school. But, missing from the literature is a prospective examination of weight status and feelings of loneliness that might come from those experiences. Using data from the Québec Longitudinal Study of Child Development, we filled that gap by examining the prospective associations between loneliness and weight status when the sample was aged 10 to 13 years. At ages 10, 12, and 13 years, 1042 youth (572 females; 92% from French speaking homes) reported on their loneliness and were weighed and measured. Family income sufficiency was included in our analyses given its relationship with weight status, but also its possible link with loneliness during early adolescence. The findings showed that (1) weight status and loneliness were not associated concurrently; (2) weight status predicted increases in loneliness from ages 12 to 13 years; and (3) loneliness predicted increases in weight from ages 12 to 13 years among female adolescents, but weight loss among male adolescents. The fact that loneliness was involved in weight gain for females suggests that interventions focused on reducing loneliness and increasing connection with peers during early adolescence could help in reducing obesity.

Keywords: Loneliness, Weight Status, Body Mass Index, childhood, adolescence, Longitudinal, Coping, Gender, Income Sufficiency, Socioeconomic status, Obesity, Overweight, Underweight.

Introduction

Social relationships and weight are prevalent concerns during adolescence (Danneel, Maes, Vanhalst, Bijttebier, & Goosens, 2018; Markey, 2010). Adolescents are vulnerable to peer disapproval of body size (Lawler & Nixon, 2011), feelings of loneliness (Qualter et al., 2015) and they are driven by a need to "fit in" (Reitz, Zimmermann, Hutteman, Specht, & Neyer, 2014). Given the stigma associated with being overweight or obese, peer disapproval is high, creating negative social consequences (Harrist, Swindle, Hubbs-Tait, Topham, Shriver & Page, 2016; Puhl, et al., 2016), heightened loneliness among 10-14 years olds (Hayden-Wade et al., 2005) and weight concerns in pre-adolescents (Sinton et al., 2012). However, the prospective relationship between obesity and loneliness has yet to be examined.

Building on longitudinal work that not only indicates that obesity predicts depression, but that depression also predicts obesity (Goldschmidt, et al., 2010), we hypothesized a bidirectional relationship between loneliness and high weight status. Conversely, feelings of loneliness are reported by those with eating disorders characterized by low body weight (Puhl & Suh, 2015) and a meta-analysis indicated that low weight and depressive symptoms are bidirectionally-related over time (Puccio et al., 2016). Therefore, the current work with adolescents prospectively examines the relationship between (a) loneliness and high weight status and (b) loneliness and low body weight.

The Social Context of Weight Status

The bio-ecological framework highlights two contexts important for adolescent health because they influence beliefs and behavior: (1) the immediate peer group, and (2) the wider social context within which the individual and peers live (Bronfenbrenner, 2005). Social exclusion by peers in school and the accompanying feelings of loneliness during adolescence are recognized as significant influences of adolescent health (Hawkley & Capitanio, 2015).

But, social exclusion based on weight status needs to also be understood within the wider social context, where social norms of the ideal body size create stigma associated with non-ideal weight, influencing adolescents to criticize their peers' appearance (Lawler & Nixon, 2011) and tease them for non-conformation to weight ideals (Mooney et al., 2009).

While male and female adolescents are often criticized about their appearance by their peers to a similar degree (Lawler & Nixon, 2011), the social norms of ideal weight are different for males and females because gender stereotypes of the socio-cultural ideal of beauty emphasize thinness for women and female adolescents (Puhl & Brownell, 2001). Such findings suggest that female adolescents may be more vulnerable to the social context of appearance than male adolescents, receiving greater pressure from peers to conform to the socio-cultural ideals. Empirical evidence shows that females are particularly vulnerable to the negative social effects of high weight status, experiencing more rejection and victimization from the peer group than boys after the age of seven years (Qualter, Murphy, et al., 2015). Thus, gender-based social norms are important to consider when examining the interaction between weight status and loneliness because females may be particularly vulnerable to the negative social effects of not conforming to weight status ideals, experiencing, as a result, more loneliness than boys.

Other society level influences are also important to consider in the current work. Socioeconomic adversity during childhood and adolescence is linked to developmental processes: low income is a known risk factor for obesity earlier in development (Grow et al., 2010) because it leads to stress responses that exacerbate metabolic processes, leading to increased weight status (Wickrama, O'Neal, & Lee, 2013). Further, there is reason to think that low income may also be linked to loneliness. To our knowledge, there is currently no research on socioeconomic status and loneliness during adolescence, but parents who have limited resources and income may not find adequate time to spend with their children,

contributing to increasing distance between parents and their children and child negligence; they also may not have the financial resources to ensure their child's engagement in specific peer group activities that cost money. For those reasons, we include family income sufficiency in our analyses.

Weight Status and Depressive Symptoms

Being at the extremes of weight status (overweight/obese or underweight) is associated with depressive symptoms (Rankin et al., 2016; Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016). Findings show that obese 12-14 year olds have increased chance of developing depression (Eschenbeck, Kohlmann, Dudley, & Schurholz, 2009), but also that depressive symptoms predict weight gain (Goldschmidt et al., 2010). The same bi-directional effects have been found for those with low weight status too, with low weight predicting depressive symptoms, but also depressive symptoms predicting decreases in weight (Puccio et al., 2016). The effects appear to be more pronounced for adolescent girls compared to boys (Anderson, Cohen, Naumova, & Must, 2006).

Mechanisms linking depression with obesity (Goldschmidt et al., 2017) and low weight (Puccio et al, 2016) during adolescents have been examined. That evidence shows that distress caused from the feelings of shame and guilt of not conforming to the body and weight ideal contribute to obesity and low weight being linked over time with depression. Goldschmidt et al also showed that depressive symptoms predict engagement in emotional eating as a way to alleviate distress.

Weight Status and Loneliness

In contrast to the work exploring weight status and depressive symptoms during adolescence, there is a paucity of research examining concurrent and prospective relationships between weight status and loneliness at that same development period. Loneliness is the negative feeling that occurs when a person does not perceive their social

relationships to be as satisfying as they would like (Perlman & Peplau, 1981), sharing one common symptom with depression (negative affect). But, loneliness is determined, specifically, by a negative emotional response to a lack of close affiliative ties to peers during adolescence; depression, in contrast, is attributed to a broader range of causes, including determinants other than deficient social relations (i.e., the individual's genetic makeup, neurological disorders, psychological dysfunctions; Koenig & Abrams, 1999). Thus, an exploration of the prospective association between weight status and loneliness is important given that loneliness specifically taps distress associated with peer group problems, which are hypothesized to be a significant part of the puzzle linking weight status with depression.

Research exploring the mechanisms linking weight status and depression discusses (1) how peer problems are a catalyst for depressive symptoms among adolescents with the highest and lowest weight statuses, but also (2) how coping with emotional distress can lead to emotional eating and increases in weight status. Based on the fact that loneliness includes the same negative affect as depression, we hypothesize a prospective bidirectional association between loneliness and weight status, which we discuss further below.

Weight status predicting loneliness.

It is clear, from empirical research, that there is stigma attached to high weight status. Empirical evidence shows that, in western society, the high degree of social stigma attached to obesity/overweight is evident as early as pre-school (Turnbull, Heaslip, & McLeod, 2000), with increasing negative ratings for "chubby" body types by children aged 5 to 10 years (Brylinsky & Moore, 1994) and peer social rejection of obese children at ages 6-7 years (Harrist et al., 2016). Those negative social circumstances continue into adolescence, with obese and overweight adolescents being regularly stigmatized, socially excluded, and victimized by peers (Puhl & Luedicke, 2012; Puhl, et al., 2016). Thus, high weight status is associated with poorer peer relationships, with overweight 10-14 year olds reporting higher

levels of loneliness (Hayden-Wade et al., 2005). Given that evidence, one could posit that higher weight status would result in less satisfaction with social relationships over time i.e. greater feelings of perceived loneliness. To date, however, the prospective link between higher weight status and feelings of loneliness has not been examined.

In addition to predicting that higher weight status could result in greater loneliness, it is also possible that those with low weight status will report increasing loneliness over time. Empirical work by Wang et al. (2010) shows that male and female adolescents with high weight status are often bullied by their peers, but underweight girls are also often victims. That aggression, and the accompanying feelings of shame and social isolation from peers, is thought to be a consequence of intrasexual competition that is promoted by society's emphasis on thinness among females (Vaillancourt, 2013). Rotenberg et al. (2013) and Rotenberg and Sangha (2014) have also shown that adolescents with low weight status as a result of eating pathologies report higher levels of loneliness compared to their peers, a consequence of stigmatization and alienated from the peer group.

Given the work detailed above, there is a need to examine whether both high and low weight status predict increasing loneliness over the adolescent years. We postulate that there will be concurrent and prospective associations between weight status and loneliness, and that those relationships will be curvilinear in nature, such that individuals with either the highest or lowest weight statuses will feel more loneliness during adolescence compared to their normal weight peers. The current study will examine the prospective curvilinear associations between weight status and loneliness.

Loneliness as a predictor of increasing weight status

Among adults, loneliness has been tentatively implicated as a risk factor for increased weight, but there are few empirical studies. Consistent with the affect-regulation model of binge-eating (Heatherton & Baumeister, 1991), evidence shows that induced loneliness leads

to increased eating as a way to alleviate distress (Rotenberg & Flood, 1999). But, loneliness has also been shown to increase stress hormones such as cortisol that come from heightened physiological stress during episodes of loneliness (Cacioppo & Hawkley, 2003), and those stress hormones affect fat storage and transportation with the body (Dallman, 2010; Moyer et al., 1994).

In addition to the direct weight-related effects of stress hormones, a combination of perceived stress, disturbed sleep, and cognitive rumination resulting from loneliness (Zawadzki, Graham & Gerin, 2013) could affect eating behavior by impairing one's ability to address problems, leading to greater use of passive coping strategies, such as emotional eating (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008). Those loneliness-related stressors are also argued to increase our seeking of easy learned-rewards such as high fat and high sugar foods due to stimulation of the motivation and reward circuits in the brain (Dallman, 2010; Hanlon, Tasali, Leproult, Stuhr, Doncheck, et al., 2016), making it easier to gain weight and harder to lose it.

The work represented above supports the idea that loneliness may lead to increases in weight status, but the prospective link has not yet been examined. The current study examines whether loneliness predicts increases in weight during late childhood/early adolescence.

The Current Study

An examination of the reciprocal prospective relationships between loneliness and weight status among adolescence is important if we want to offer effective intervention solutions for loneliness and obesity among youth. In the current study, feelings of loneliness and weight status are explored in a population sample of Canadian 10-13 year olds, and we examine whether high and low weight status put adolescents at risk of later loneliness, but also whether loneliness scores predict increasing weight status. The findings could have important implications for interventions for obese or underweight youth, and for adolescents

who report loneliness. Guided by the bioecological framework, we also seek to examine gender and income sufficiency as moderators of the prospective relationships between weight status and loneliness.

Method

This study utilized data from the Québec Longitudinal Study of Child Development (QLSCD), a large on-going study which has tracked the health and wellbeing of a random sample of Quebec infants on a range of measures since they were 5 months old (see the study website http://www.iamillbe.stat.gouv.qc.ca/default_an.htm for further details). The representative sample is made up of children born throughout 1998 in the Canadian province of Québec (total population over 7 million, with approximately 70,000 newborns per year). A total of 2,940 infants were selected for QLSCD through a region-based stratified sampling design, of which 2,120 infants (48.8% girls) took part, with parents providing informed consent in 1998. Twins and children with major diseases at birth were not part of the study. In the current study, four waves of data were used from successive data collections when the sample of children was aged 10, 12, and 13 years. We refer to these time points as T1, T2, and T3 respectively of the current study. The QLSCD was approved by the Health Research Ethics Committees of the Québec Statistics Institute and the University of Montreal.

Participants

Loneliness, weight, and height data were collected from 1259 children/early adolescents (667 females, 592 males) at T1-T3 of the current study. Participants with and without all data for the period of the current study were compared using Little's missing completely at random test (Little, 1988) to determine whether data imputation would be possible. That comparison resulted in a significant chi-square value, X^2 (18) = 35.246, p = .009, suggesting that missing values could not be dealt with using data imputation methods. Thus, we used listwise deletion of cases, analyzing data from 1042 participants (572 females, 470 males) for whom complete loneliness, height, and weight data were available at all three

time points[1]. Table 1 shows how the final sample in the current study compares to the original QLSCD sample that had been chosen as a representative sample of children in Québec in 1998. The table shows that, while there were fewer males in the current sample than in the original sample, the reduced sample taking part in the current study was representative of the same children living in the province of Québec in 1998.

Table 1 about here

Measures

Loneliness. Loneliness was measured using the Loneliness and Social Satisfaction questionnaire developed by Rotenberg et al., (2004). This three-item measure is similar to the 3-item short form of the UCLA (University of California, Los Angeles) Loneliness Scale (Hughes, Waite, Hawkley and Cacioppo, 2004), but the word "isolated" was simplified to "alone" in item 3. Items asked the extent to which, in the last two weeks, participants had felt (1) "they had people they could talk to", (2) "left out of things" and, (3) "alone". Item 1 was reverse coded so that higher scores represented higher feeling of loneliness. Participants answered how they best described those feelings (1=never, 2= sometimes, 3= always), with possible total scores ranging from 3 to 9. Cronbach's alpha for the loneliness measure was .66, .68, and .74 at T1, T2, and T3 respectively. Total scores on the loneliness scale were used in our regression analyses looking at the prospective associations between loneliness and weight status, but we also created loneliness groups to examine whether children who

[1] Loneliness, weight, and height data were also available for 926 of the 1042 children at a further time point when the sample was aged 15 years. However, the measure of loneliness was limited to a single item, with response items that did not align with that for the earlier loneliness items; height and weight data were self-reported. In addition, there were so few participants who were classified as underweight (N=20 females; 9 males) or obese (N = 9 females; 5 males) that it would not make the analyses viable.

reported higher levels of loneliness at any given time also had higher weight status scores (higher BMI) at that same time point. Following Yang and Victor (2011) we created a "frequently lonely" group of participants who scored 6 or above on the loneliness scale; all remaining youth were grouped into a "not lonely/occasionally lonely" category. Those groups were created for the purpose of making comparisons on BMI at each time point.

Weight status. Trained research assistants weighed and measured the participants when they were wearing lightweight clothing and no shoes. Two measurements were taken, and, if they varied by more than 0.5 cm for height or 0.2 kg for weight, a third measurement was taken. Where multiple measures were available, we used the average of each measure to calculate BMI (BMI = kg/m2). The computation of the participants' BMI was followed by the creation of a BMI z-score using respondents' BMI, self-reported age, gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007). Those BMI z-scores (referred to here as z-BMI) were used in our regression analyses, exploring the prospective association between weight status (z-BMI) and loneliness. As well as creating a z-BMI score for each participant, we also classified each of them as underweight, overweight, obese, or normal weight, following recommendations from The International Obesity Task Force BMI (IOTF; Cole, Flegal, Nicholls, & Jackson, 2007). Those recommendations classified participants into the following weight categories at each time point: thin grade 3 (BMI $\leq$ 16), thin grade 2 (BMI $\leq$ 17 & > 16), thin grade 1 (BMI $\leq$ 18.5 & > 17), overweight (BMI $\geq$ 25 & < 30), obese (BMI $\geq$ 30) morbid obesity (BMI > 30), and normal weight (BMI $\geq$18.6 & $\leq$ 24.5). Those IOTF cut-offs are recommended for international research and comparison (Lobstein et al., 2004), and are used here to create weight status groups for each time point, enabling us to explore whether different weight categories reported higher levels of loneliness at each time point.

Income sufficiency. Sufficiency of income was determined by low-income cutoffs set by

Statistics Canada in any given year. It takes into account household income, the size of the household, and the size of the residence area. Statistics Canada's low-income cut-offs (LICOs) are income thresholds at which a family would typically spend 20% more of its income than the average family on the necessities of food, shelter, and clothing (https://www.statcan.gc.ca/pub/75f0002m/2012002/lico-sfr-eng.htm). Families are classified as having "sufficient income" when the household income is above the low-income threshold determined by Statistics Canada. When income is between 60% and 90% of the low-income threshold, households are classified as having "insufficient income"; income levels below 60% of the low-income threshold are considered as "very insufficient". Although LICOs are widely used, they do not measure poverty. Unlike the US, Canada does not have a measure of poverty. For example, according to Statistics Canada (https://www.statcan.gc.ca/pub/75f0002m/75f0002m2010003-eng.htm) low-income thresholds are different for a family living in a rural area compared to similar families living in large cities.

Analyses Plan

First, using ANOVA, we examined whether there were differences between people in the different weight categories on loneliness at each time point. Second, using T-tests, we examined differences in BMI between groups of youth identified as either "frequently lonely" or "not lonely/occasionally lonely". We conducted all analyses separately by gender. Third, we conducted a series of chi-square tests to clarify the associations between loneliness and weight status, exploring for males and females separately, membership of any given loneliness group by weight category at each time point. Those chi-square analyses helped us to understand whether adolescents who were "frequently lonely" at any time point were more likely than chance to also be members of the obese or underweight weight groups. Together,

those sets of analyses enabled us to examine concurrent relationships between loneliness and weight status.

Fourth, we examined prospective linear and curvilinear relationships between (1) z-BMI and loneliness, and (2) loneliness and z-BMI using Hierarchical Regression Analyses (HRAs), controlling for gender and income sufficiency. Curvilinear analyses were used to determine the exact associations between weight status at one time point and loneliness at the following data collection waves, and loneliness at one time point and weight status at the other data collection waves. Based on our findings of quadratic effects between z-BMI and loneliness, Structural Equation Modelling was not an appropriate analytic tool and the HRAs are presented as our final statistical analyses.

Results

We examined differences in mean loneliness for young people in different weight categories using ANOVAs, looking at each time point and males and females separately. Because there were so few participants in the three grades of thinness as defined by Cole et al., 2017, we merged those groups to create one group that we defined as "underweight" (BMI < 18.5). We also merged the obese and morbidly obese groups given that there were so few participants classified as morbidly obese.

The one-way ANOVAs investigating loneliness by weight category revealed no significant differences at any of the three time periods: T1 (age 10 years) females, $F(3, 568) = .994$, $p = .395$, males, $F(3, 466) = 1.057$, $p = .367$; T2 (age 12 years) females, $F(3, 568) = .741$, $p = .528$, males, $F(3, 466) = 1.835$, $p = .140$; and T3 (age 13 years) females, $F(3, 568) = .014$, $p = .998$, and males, $F(3, 466) = .393$, $p = .758$. Thus, in the current population sample, it seems weight status is not associated with concurrent reports of higher loneliness during adolescence for males or females; those with high or low weight status did not report higher levels of loneliness compared to normal weight category to report feelings of loneliness.

Table 2 about here

The next set of analyses examined whether adolescents classified as "frequently lonely" were different to peers categorized as "not lonely/sometimes lonely" on weight status at each of the time points. We ran a series of independent t-tests, separately for males and females and found no difference between the "frequently lonely" and "not lonely/sometimes lonely" groups at age 10 years (T1: females, $t(74.78) = 1.88$, $p = .064$; males, $t(465) = 1.12$, $p = .264$) or age 12 years (T2: females, $t(570) = 1.26$, $p = .210$; males, $t(467) = .03$, $p = .974$). At age 13 years (T3), there was a difference between the lonely groups for males ($t(81.38) = 3.04$, $p = .003$), but not females ($t(77.77) = 1.53$, $p = .131$). Findings showed that male adolescents aged 13 years in the "frequently lonely" group had a higher weight status than their male peers in the "not lonely/sometimes lonely" group (Table 3).

Given that the mean BMI scores at T3 for males in the "frequently lonely" group would be considered within the normal range of BMI scores according to The International Obesity Task Force BMI cut-offs (Cole et al., 2007; normal weight = BMI $\geq$18.6 & $\leq$ 24.5), we decided to explore the concurrent association between loneliness and weight status at T3 further. We conducted two Fisher-Freeman-Halton tests, one for males and one for females. Those analyses showed that, at age 13 years (T3), males classified as "frequently lonely" were more likely than chance to be in the normal weight category, while boys in the "not lonely/sometimes" group were more likely to be in the underweight category and less likely than chance to be in be in the normal weight group and overweight weight status groups ($z = 7.817$, $p = .012$). That was not the case for girls at age 13 years ($z = 2.096$, $p = .558$). Thus, it seems that the significant difference in BMI scores between the "frequently lonely" versus "not lonely/sometimes lonely" groups of 13-year old males was driven by the higher numbers

of males from the "not lonely/sometimes lonely group" in the underweight category than we would expect by chance. Taken together with the results from the ANOVA, t-tests, and chi-square analyses show no concurrent relationships between the highest and lowest weight status and loneliness.

Table 3 about here

To examine prospective effects of weight status on loneliness (DV) we ran a series of Hierarchical Multiple Regressions, with predictors on the following steps: (1) gender, income sufficiency, and loneliness (HRAs) from the earlier time point, (2) z-BMI from the earlier time point, (3) z-BMI squared (z-BMI^2), (4) z-BMI x gender interaction, and (5) z-BMI^2 x gender interaction. The squared (^{2}) term serves as a test for a quadratic relation (Cohen et al. 2003). Scores were centered using grand mean subtraction for loneliness, and BMI z-scores. Gender and income sufficiency were dummy coded (gender: −1 = female, and +1 = male; +1 = income sufficiency, and -1= insufficient, with the latter including the categories of insufficient and very insufficient) as recommended by Cohen et al. (2003). We performed bootstrapping, estimating a 95% bias-corrected confidence interval for all values of interest (1000 bootstrap sample).

A further set of HRAs was conducted to examine the longitudinal over-time effects of loneliness on BMI. The regressions followed the pattern and procedure outlined above, with z-BMI at each time point as the independent variable, and loneliness and z-BMI from earlier time points as predictors. Any two-way interactions between z-BMI^2 x Gender were further examined by testing for the linear and quadratic (curvilinear) relations on the measure for each gender separately. Key information from the HRAs are detailed in the manuscript text,

with tables (**Tables S1-S6**) detailing all HRA information included as on-line supplementary information.

Stability of Weight and Loneliness Over Time

As anticipated, over time the strongest predictor of z-BMI and loneliness were previous measures of the same construct, confirming stability. The strongest effects for both constructs were seen over the one-year interval between age 12 and 13 years (T2 to T3). z-BMI stability co-efficients ranged from β= .89 (SE= .02) to .95 (SE=.02), with the highest stability being one year to the next. Loneliness beta weights ranged from β= .27 (SE= .03) to .47 (SE= .04), with moderate to large effects (per Cohen 1988).

Effects of Income Sufficiency on Weight Status and Loneliness

Bootstrapped findings revealed no influence of earlier income sufficiency on weight status (β= .00 [SE= .02] to -.02 [SE=.02]) or loneliness (β= .05 [SE= .06] to -.11 [SE=.07]).

Weight Status Predicting Loneliness

Bootstrapped findings showed only one significant linear effect between weight status at T2 and loneliness at T3 (β= .11 [SE= .04], p = .007), suggesting that higher weight status at age 12 predicted higher loneliness at age 13 years. That effect is detailed in Figure 1. There were no other significant linear or quadratic effects of z-BMI on loneliness over time (See Tables S1-3 for details).

Figure 1 about here

Loneliness Predicting Weight Status

Bootstrapped findings showed a quadratic effect of loneliness at T2 (age 12 years) that interacted significantly with gender to predict z-BMI at T3 (age 13 years), β= .02, SE=.01, p = .007 (see Table S6 for full results). Figure 2 illustrates the quadratic gender-mediated effect,

showing that (a) higher loneliness at age 12 years was related to higher z-BMI at age 13 years for girls, but (b) high loneliness at age 12 years was related to lower z-BMI at age 13 years for boys. This result suggests that loneliness may have a particular role in increasing weight for girls and reducing weight for boys. There were no other significant linear or quadratic effects of loneliness on z-BMI over time (See Tables S4-6 for details).

Figure 2 about here

Discussion

The relationships between weight status and loneliness are complex. Weight status was not associated with concurrent feelings of loneliness, but higher weight at age 12 years predicted increased loneliness from age 12 to 13 years. Interestingly, for those who reported loneliness at age 12 years, we observed a differential effect: weight gain for girls and weight loss for boys. That is likely to have detrimental psychosocial consequences for both girls and boys given that the perceived ideal female body is slim, but the ideal male body shape is muscular (Field, et al. 2014). Findings also suggest a significant health consequence for girls that are lonely, with increases in weight, which for girls already overweight or obese, is a significant health risk.

The Effect of Weight Status on Loneliness

Due to the increasing importance of peer acceptance and body image concerns during early adolescence (Danneel, at el. 2018; Markey, 2010), and the specific peer problems encountered by youth at the extremes of weight status (Puhl, et al., 2016), we investigated whether extremes of weight status would predict greater loneliness. Unlike previous research (Hayden-Wade et al., 2005), we did not find concurrent associations between high weight status and loneliness. We did find prospective associations between high weight status and loneliness, with male and

female adolescents with higher weight status at age 12 years reporting increased loneliness between 12 and 13 years. Such findings that there were no within-time gender differences are consistent with previous research showing that male and female adolescents experience weight related criticism to a similar degree (Lawler & Nixon, 2011). Further, the fact that those with higher weight status reported increasing loneliness highlights the fact that the peer context is important for understanding the social norms surrounding weight status, with society's body ideals communicated and reinforced by peers (Jones & Crawford, 2006); the fact that the prospective effects linking high weight status and loneliness are only evident at ages 12 to13 years and not 10-12 years suggests a sensitive period in development when the peer context becomes particularly influential in delivering messages about ideal body size.

Future research will want to examine whether weight status exerts effects on loneliness only under specific circumstances, such as those where there is a high-level of weight-based victimization (Juvonen et al., 2017) or an internalization of the appearance ideals (Lawler & Nixon, 2011), or when internalizing problems, such as depression, already exist. How loneliness explicitly links to weight-based victimization and self-esteem should also be examined. In addition, time-specific influences of the peer context – in and outside school- that explain the link between high weight status and loneliness should be examined to determine whether there are sensitive periods during which adaptive responses to peer relationship difficulties can be most effective.

Contrary to expectations, in our study low weight was not found to be concurrently associated with, or a significant predictor of, loneliness. The number of participants who were classified as underweight was small, making it more difficult to gauge those effects. That said, it is also possible that having low weight status is less socially stigmatizing than being overweight or obese. It is possible that, due to differences in assumptions made about the

volitional cause of high weight status (Puhl & Heuer, 2010), it is the case that those who are underweight have fewer social problems. Underweight could also be easier to disguise than overweight, leading to fewer negative social repercussions.

The Effect of Loneliness on Weight Status

In line with predictions, loneliness had a significant prospective impact on weight status interacting significantly with gender at age 12 years to predict weight status at age 13 years. Specifically, female adolescents with higher loneliness at age 12 years gained weight from age 12 to 13 years, while male adolescents with higher loneliness lost weight. Those results contribute to a growing body of research indicating that loneliness affects health (Hawkley & Capitanio, 2015), and are consistent with findings that higher loneliness leads to increased food consumption among female older adolescents (Rotenberg & Flood, 1999). While we have not examined mechanisms linking loneliness and weight status, we have provided the first evidence that loneliness is directly related to increasing weight for females upon entry into early adolescence.

The finding that loneliness reduces weight for male adolescents might suggest that loneliness serves as an inhibitor of food consumption among those male adolescents; in contrast, loneliness may have disinhibited food consumption for the female adolescents, a significant problem for those girls already overweight or obese females. The reduction in weight by male adolescents with high weight status in the current sample may be demonstrable of a heightened awareness that they had to lose weight to improve their social connections or were simply more motivated to do so. But, it may be the case that boys in the current sample simply dealt with the stigmatization of overweight by inhibiting eating as a way to copy with negative emotions. It is also possible that boys do not experience psychosocial effects until norms around their ideal male body shape (that of muscle; Field et al, 2014) materialize during puberty. Future research should examine those effects.

The weight gains we saw in girls is likely to be the outcome of engagement in emotional eating, a maladaptive method of alleviating negative emotions (Haedt-Matt & Keel, 2011), that they used to cope with feelings of loneliness. Goldschmidt et al. (2017) found that poor emotional awareness and limited access to adaptive emotion regulation strategies contribute to emotional eating in adolescent females, so further research will want to establish whether emotional eating and poor emotion regulation can help explain the prospective association between loneliness and weight gain for female adolescents.

The recommended next stage of empirical study is the examination of mechanisms that explain how loneliness increases weight status among adolescent females and decreases weight status for adolescent males. The work detailed above may suggest that male adolescents are more motivated to lose weight or more aware that doing so would lead to increased social acceptance needs to be explored, but the possibility that female adolescents were engaged in emotional eating also needs to be examined. Given that among females, loneliness is associated with ruminative cognitions (Vanhalst, Luyckx, Raes & Goossens, 2012), it is possible that rumination directly affects weight by influencing coping strategies (i.e., emotional eating) and planning (i.e., it reduces one's ability to stick to healthy eating intentions). Future work should examine emotion regulation strategies, including rumination, and explore whether alternative, more adaptive strategies for dealing with loneliness might not lead to weight gain. Such work is important for informing interventions focused on reducing loneliness and/or obesity.

Future research will also want to explore how the physiological effects of loneliness could influence eating behavior including increasing the propensity to binge eat palatable foods and reducing one's ability to track food consumption. Such future work should take into account the moderating effects of sleep and physical activity as those factors affect weight metabolism and have been shown to be deficient in lonely adolescents (Harris,

Qualter & Robinson, 2013; Pels & Kleinert, 2016). The impact of our findings for interventions to prevent obesity is clear – targeting early adolescents, particularly females, who are lonely, could help in the fight against obesity.

Stability of Loneliness and Weight Status Over Time

Results also support the stability of weight status and loneliness over time. The relative stability of the constructs over time supports previous research (Pryor et al, 2011; van Dulmen & Goossens, 2013). The strong stability of weight status and loneliness reflects the difficult task that intervention teams face in order to affect changes in those areas of health. After age 13 years, the stability of weight status is likely to change due to physiological changes that accompany puberty, and future work will want to examine how the onset of puberty impacts the prospective associations between weight status and loneliness, exploring the impact for male and female adolescents separately. Given that for females, advanced pubertal maturation is associated with internalizing problems, explained exclusively in terms of environmental influences (Marceau et al., 2012), we might expect that the prospective association between weight status and loneliness is also moderated by pubertal timing for girls. The time interval between data collection waves in the study varied from one to two years, but the effects were seen in the one-year time interval between age 12 to age 13 (T2 to T3). That age could be a key sensitive period for peer relationship problems to impact weight status, but future longitudinal studies may consider one-year time intervals between waves to give a more nuanced picture of magnitude and persistence of effects over time (VanderWeele, Hawkley, Thisted, & Cacioppo & 2011).

Income Sufficiency and Effects on Weight Status and Loneliness

In the current study, we explored the impact of income sufficiency on weight status and loneliness. We did not find any effects linking recent social disadvantage, measured here

in terms of income sufficiency, to high weight status. That finding is consistent with other research (Lee et al., 2014) that found only poverty exposure prior to 2 years of age had a robust association with adolescent obesity.

To our knowledge, this is the first study to examine the longitudinal associations between income sufficiency and youth loneliness. We thought it might be the case that children whose parents had limited income did not have adequate time to spend with their children or have the resources to support peer engagement activities, and children from those families would experience increasing distance from parents and peers, and, thus, report loneliness. But, we did not find that adolescents whose families had insufficient incomes as defined by Statistics Canada, reported higher rates of loneliness. Further empirical work should examine income sufficiency in relation to loneliness, examining whether that effect is found for children whose focus is more on parents as the main source of support (Csikszentmihalyi & Larson, 1984).

Strengths and Limitations

This study has several strengths, including the large population sample, the robust anthropometric techniques used to collect weight and height data, and the prospective nature of the design. The current study followed children over a 3-year period into early adolescence and allowed confirmation of the temporal relationships between loneliness, weight status, and income sufficiency, and provided exploration of effects for male and female adolescents. The time period provided a good test of the stability of the constructs during a period of developmental transition. The simplicity, longitudinal design, and youth sample in the current study add depth to our understanding of the links between weight status and social problems faced by young people during a key period in development.

The study is not without limitations. The difference in interval lengths between data collection points could have introduced confounds into the study. Equal time intervals between measurement points would be preferable (VanderWeele et al., 2011). Missing cases were dealt with through Listwise deletion because the Data Missing Completely At Random (MCAR) tests proved significant. Our chosen method is a less favored method of data cleansing and could have introduced bias in the sample by altering the standard error estimates for sub samples due to non-random data (Allison, 2002). However, the use of bootstrapping helps provide an assurance that the results are not spurious.

Areas for Further study

Throughout this discussion we have highlighted important areas of future work. We noted the need to examine whether weight status exerts effects on loneliness only under specific circumstances, including situations where there is a high-level weight-based victimization or internalization of appearance ideals. We also noted the need to further explore the gendered responses to loneliness, and how any differences between male and female adolescents are related to future gains or reductions in weight.

In addition, in future studies, several further variables could be controlled, including activity levels and onset of puberty. Ethnicity could not be explored as a moderator in the current study because the sample was not ethnically diverse. In future work, ethnicity should also be explored given evidence that girls with high weight status in certain ethnic groups suffer less from the negative effects of weight stigma than other groups (Mustillo, Budd & Hendrix, 2013).

Conclusion

The current study examined the concurrent and prospective reciprocal relationships between weight status and loneliness, controlling for income sufficiency and gender. We found that both male and female adolescents with higher weight status reported increasing loneliness from ages 12 to 13 years, showing that society's views of what constitutes an body ideal size and shape are communicated and reinforced by peers at this point in development.

In addition, loneliness at age 12 years reduced weight for male adolescents from age 12 to 13 years, and increased weight for female adolescents during that same period, suggesting that loneliness may serve as an inhibitor of food consumption among male adolescents, but may disinhibit food consumption for female adolescents, a significant problem for already overweight or obese females. Further work will want to explore the prospective effects further to determine whether the gendered effect is specific to the current sample, and, if not, what that effect tells us about gendered coping and peer group friendships among male and female youth.

The findings suggest early clinical attention to high weight status and loneliness will be important and may have significant effects for adolescent females. Reducing peer-related problems for those with high weight status will help reduce feelings of loneliness, but such problems reflect society's stigma attached to non-conformation to body ideals, making such social norms hard to change. Thus, it will be more important to help adolescents develop resilient strategies of coping with peer criticism of non-ideal body size, reducing the chance that they will engage in emotional eating or experience social stress, both of which would lead to weight gains.

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Appendix C Table 1. Characteristics of the Sample at the Start of the Quebec Longitudinal Study of Child Development (QLSCD) survey and at T1-T3 of the current study.

Time point	Age of participants in months (SD)	n =
Start of QLSCD survey in 1998	4.5 (0.55)	2120
T1 Current Study	121.70 (3.10)	1042
T2 Current Study	145.60 (3.05)	1042
T3 Current Study	157.60 (3.12)	1042

Demographics taken at start of QLSCD for the current sample compared to original sample

	Sample chosen for QLSCD	Sample in current Study
Males %	51	45
French-speaking families %	81	92
Mother's age in years	29	31
Father's age in years	31	33
Mothers did not hold a high school degree %	17	15
Fathers did not hold a high school degree %	20	16
Mother had a university degree %	28	32
Father had a university degree %	26	27
Income Sufficiency at normal levels[2]	81	88
Households headed by single parent %	7	5

[2] Statistics Canada's low-income cut-offs (LICOs) were used to categorize the families of participants on income sufficiency. LICOs are income thresholds at which a family would typically spend 20% more of its income than the average family on the necessities of food, shelter, and clothing. Families are classified as having "sufficient income" when the household income is above the low-income threshold determined by Statistics Canada in any given year. When income is between 60% and 90% of the low-income threshold, households are classified as having "insufficient income"; income levels below 60% of the low-income threshold are considered as "very insufficient". QLSCD = Quebec Longitudinal Study of Child Development.

Appendix C Table 2. Mean (and Standard Deviations) for Loneliness in each Weight Category at Each Time Point for Females and Males

Time (age)	Underweight BMI <18.5		Normal Range 18.51-24.99		Overweight BMI >=25		Obese BMI>=30	
	FEMALES	**MALES**	**FEMALES**	**MALES**	**FEMALES**	**MALES**	**FEMALES**	**MALES**
T1 (10 yr)	3.64 (.93)	43.46 (.95)	3.82 (1.19)	3.88 (1.23)	3.97 (1.33)	3.86 (1.29)	4.03 (1.27)	3.72 (.96)
N=1042	36	26	407	325	99	90	30	29
T2 (12 yr)	3.71 (.86)	3.28 (.61)	3.76 (1.19)	3.81 (1.25)	3.90 (1.17)	3.73 (1.09)	3.97 (1.17)	3.58 (.97)
N=1042	35	25	384	311	116	98	37	36
T3 (13 yr)	3.80 (1.10)	3.93 (1.51)	3.83 (1.31)	3.92 (1.29)	3.85 (1.14)	4.06 (1.43)	3.84 (1.26)	3.82 (1.29)
N=1042	30	28	384	299	120	105	38	38

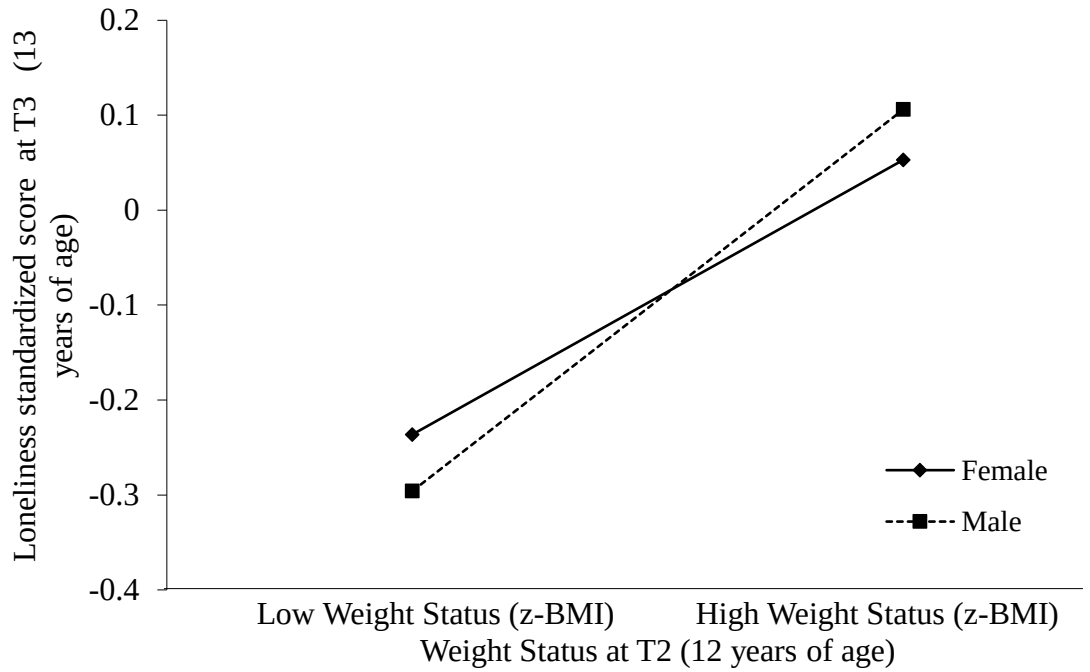
Notes. Sample participants were categorized using the following recommendations from The International Obesity Task Force BMI (IOTF; Cole, Flegal, Nicholls, & Jackson, 2007): thin grade III (BMI $\leq$ 16), thin grade II (BMI $\leq$17 & > 16), thin grade I (BMI $\leq$ 18.5 & > 17), overweight (BMI $\geq$ 25 & < 30), obese (BMI $\geq$ 30), morbid obesity (BMI > 30), and normal weight (BMI $\geq$ 18.6 & $\leq$ 24.5). Underweight = Grade I, II, III were combined due to low numbers in Grades II and III; Obese (obese and morbid obese were combined due to low numbers in the morbid obese category); Possible loneliness scores ranged from 3 to 9. N= 1042 (Female = 572; Male = 470). ANOVAs revealed no differences between males and females on feelings of loneliness at each time point.

Appendix C Table 3. Mean (and Standard Deviations) for BMI by Loneliness Group at Each Time Point for Females and Males

Time	Frequently Lonely		Not Lonely/ Sometimes Lonely	
	FEMALES	**MALES**	**FEMALES**	**MALES**
T1 (10 yr)	19.26 (4.17)	17.76 (2.32)	18.28 (3.11)	18.25 (2.06)
N=1042	66	54	506	416
T2 (12 yr)	20.63 (3.43)	19.86 (3.92)	19.98 (3.88)	19.84 (3.76)
N=1042	60	49	512	421
T3 (13 yr)	21.82 (5.39)	22.52 (5.11)	20.80 (3.84)	20.56 (3.79)
N=1042	69	69	503	401

Notes. Those in the "Frequently Lonely" group scored 6 or above on the loneliness scale; those scoring between 3 and 5 were classified as "Not Lonely/Sometimes Lonely"; N= 1042 (Female = 572; Male = 470); T-tests showed no differences on BMI between same sex peers in the "Frequently Lonely" and "Not Lonely/Sometimes Lonely" groups at ages 10 and 12 years. At age 13 years, males in the "Frequently Lonely" group scored significantly higher on BMI compared to their same sex peers in the "Not Lonely/Sometimes Lonely" group; there were no significant differences between females. All BMI mean scores would be considered in the normal range according to The International Obesity Task Force BMI Cut-offs (IOTF; Cole, Flegal, Nicholls, & Jackson, 2007) where normal weight is considered to be BMI $\geq$ 18.6 & $\leq$ 24.5.

Appendix C Figure 1. Weight Status at T2 (age 12 years) and Loneliness at T3 (13 years).



Appendix C Figure 2. Slopes of the Relation between Loneliness T2 (age 12 years) and BMI T3 (age 13 years) as a Function of Gender.

Appendix C **Supplementary On-line Information**

Reciprocal Prospective Relationships Between Loneliness and Weight Status in Late Childhood and Early Adolescence

Supplementary On-line Information

HRA Bootstrapped 5-Step Results Tables

Table S1. Bootstrapped Hierarchical Regression Analysis: T1 z-BMI as a Predictor of Dependent Variable T2 Loneliness including quadratic effects and interactions.

		Coefficients			Variance explained in each step		
	Predictors	B (SE)	95% CI	*p*	R2	ΔR2	*F* Change
1	GENDER	-.03 (.04)	[-.10, .05]	.398	.09	.09	$F(3,1016)= 31.56$, $p<.001$
	IS_T1	-.11 (.07)	[-.25, .02]	.079			
	T1_Lon	.27 (.04)	[.20, .34]	.001			
2	GENDER	-.03 (.04)	[-.10, .05]	.400	.09	.00	$F(1,1015)= 1.18$, $p=.278$
	IS_T1	-.11 (.07)	[-.25, .02]	.083			
	T1_Lon	.27 (.04)	[.20, .34]	.001			
	T1 z-BMI	.04 (.04)	[-.03, .11]	.302			
3	GENDER	-.03 (.04)	[-.10, .05]	.402	.09	.00	$F(1,1014)=.01$, $p=.915$
	IS_T1	-.11 (.07)	[-.24, .02]	.088			
	T1_Lon	.27 (.04)	[.20, .34]	.001			
	T1 z-BMI	.04 (.04)	[-.04, .13]	.342			
	T1 z-BMI^2	.00 (.02)	[-.05, .03]	.896			
4	GENDER	-.03 (.04)	[-.10, .04]	.390	.09	.00	$F(1,1013)= 1.46$, $p=.228$
	IS_T1	-.11 (.07)	[-.24, .02]	.097			
	T1_Lon	.27 (.04)	[.20, .34]	.001			
	T1 z-BMI	.04 (.04)	[-.04, .12]	.337			
	T1 z-BMI^2	.00 (.02)	[-.05, .03]	.805			
	T1 z-BMI x Gender	-.04 (.04)	[-.11, .04]	.242			
5	GENDER	-.02 (.04)	[-.10, .07]	.679	.09	.00	$F(1,1012)=.33$, $p=.565$
	IS_T1	-.11 (.07)	[-.24, .02]	.093			
	T1_Lon	.27 (.04)	[.20, .34]	.001			
	T1 z-BMI	.05 (.04)	[-.04, .13]	.292			
	T1 z-BMI^2	-.01 (.03)	[-.06, .04]	.684			
	T1 z-BMI x Gender	-.03 (.04)	[-.11, .06]	.521			
	T1 z-BMI^2 x Gender	-.01 (.03)	[-.06, .04]	.604			

Note. T1=10 years old, T2=12 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model:* $F(7,1012)=13.94$, $p<.001$

Table S2. Bootstrapped Hierarchical Regression Analysis: T1 z-BMI as a Predictor of Dependent Variable T3 Loneliness including quadratic effects and interactions.

		Coefficients			Variance explained in each step		
	Predictors	B (SE)	95% CI	*p*	R2	ΔR2	*F* Change
1	GENDER	-.03 (.04)	[-.10, .05]	.458	.08	.08	$F(3,1016)= 29.22$, $p<.001$
	IS_T1	-.07 (.07)	[-.21, .06]	.340			
	T1_Lon	.30 (.03)	[.23, .36]	.001			
2	GENDER	-.03 (.04)	[-.10, .05]	.467	.08	.00	$F(1,1015)= 3.95$, $p=.047$
	IS_T1	-.06 (.07)	[-.20, .07]	.387			
	T1_Lon	.29 (.03)	[.23, .36]	.001			
	T1 z-BMI	.08 (.05)	[-.02, .17]	.094			
3	GENDER	-.03 (.04)	[-.10, .05]	.466	.09	.00	$F(1,1014)= 2.85$, $p=.092$
	IS_T1	-.06 (.07)	[-.20, .07]	.366			
	T1_Lon	.29 (.03)	[.23, .36]	.001			
	T1 z-BMI	.03 (.05)	[-.08, .12]	.607			
	T1 z-BMI^2	.03 (.03)	[-.01, .10]	.125			
4	GENDER	-.03 (.04)	[-.10, .05]	.462	.09	.00	$F(1,1013)=.06$, $p=.807$
	IS_T1	-.06 (.07)	[-.21, .07]	.361			
	T1_Lon	.29 (.03)	[.23, .36]	.001			
	T1 z-BMI	.03 (.05)	[-.08, .12]	.606			
	T1 z-BMI^2	.03 (.03)	[-.01, .10]	.131			
	T1 BMI x Gender	.01 (.05)	[-.08, .11]	.830			
5	GENDER	.00 (.05)	[-.09, .10]	.995	.09	.00	$F(1,1012) = 1.57$ $p=.211$
	IS_T1	-.06 (.07)	[-.20, .07]	.356			
	T1_Lon	.29 (.03)	[.23, .36]	.001			
	T1 z-BMI	.04 (.05)	[-.07, .13]	.436			
	T1 z-BMI^2	.02 (.04)	[-.04, .11]	.533			
	T1 z-BMI x Gender	.05 (.05)	[-.04, .12]	.326			
	T1 z-BMI^2 x Gender	-.03 (.04)	[-.11, .04]	.443			

Note. T1=10 years old; T3=13 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model:* F (7,1019) = 13.78, $p<.001$

Table S3. Bootstrapped Hierarchical Regression Analysis: T2 z-BMI as a Predictor of Dependent Variable T3 Loneliness including quadratic effects and interactions.

		Coefficients			Variance explained in each step		
	Predictors	B (SE)	95% CI	*p*	R2	ΔR2	*F* Change
1	GENDER	.00 (.04)	[-.07, 07]	.933	.19	.19	$F(3,1030)=77.90, p<.001$
	IS_T2	.05 (.06)	[-.08, .17]	.443			
	T2_Lon	.47 (.04)	[.40, .55]	.001			
2	GENDER	.00 (.04)	[-.07, .07]	.920	.19	.01	$F(1,1029)=9.86, p=.002$
	IS_T2	.06 (.06)	[-.06, .18]	.343			
	T2_Lon	.47 (.04)	[.39, .54]	.001			
	T2 z-BMI	.11 (.04)	[.03, .19]	.007			
3	GENDER	.00 (.04)	[-.07, .07]	.916	.20	.00	$F(1,1028)=3.78, p=.052$
	IS_T2	.06 (.06)	[-.06, .18]	.300			
	T2_Lon	.47 (.04)	[.39, .55]	.001			
	T2 z-BMI	.06 (.05)	[-.03, .14]	.195			
	T2z-BMI^2	.04 (.02)	[-.01, .09]	.091			
4	GENDER	.00 (.04)	[-.07, .07]	.918	.20	.00	$F(1,1027)=.28, p=.599$
	IS_T2	.06 (.06)	[-.05, .18]	.289			
	T2_Lon	.47 (.04)	[.39, .55]	.001			
	T2 z-BMI	.06 (.05)	[-.03, .14]	.194			
	T2 z-BMI^2	.04 (.03)	[-.01, .10]	.086			
	T2 z-BMI x Gender	.02 (.04)	[-.07, .10]	.647			
5	GENDER	.02 (.05)	[-.07, .11]	.728	.20	.00	$F(1,1026)=.80, p=.371$
	IS_T2	.06 (.06)	[-.05, .18]	.288			
	T2_Lon	.47 (.04)	[.39, .54]	.001			
	T2 z-BMI	.07 (.05)	[-.03, .15]	.155			
	T2 z-BMI^2	.03 (.03)	[-.02, .10]	.311			
	T2 z-BMI x Gender	.04 (.05)	[-.05, .14]	.343			
	T2 z-BMI^2 x Gender	-.02 (.03)	[-.08, .05]	.527			

Note. T2=12 years old; T3=13 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model:* F (7,1026)= 35.84, $p<.001$

Table S4. Bootstrapped Hierarchical Regression Analysis: T1 Loneliness as a Predictor of Dependent Variable T2 z-BMI including quadratic effects and interactions.

		Coefficients			Variance explained at each step		
	Predictors	B (SE)	95% CI	*p*	R2	ΔR2	F Change
1	GENDER	.00 (.01)	[-.02, .03]	.909	.84	.84	$F(3,1016)=1795.64$, $p<.001$
	IS_T1	.00 (.02)	[-.04, .04]	.910			
	T1 z-BMI	.92 (.02)	[.88, .95]	.001			
2	GENDER	.00 (.01)	[-.02, .03]	.987	.84	.00	$F(1,1015)=.95$, $p=.331$
	IS_T1	.00 (.02)	[-.04, .04]	.988			
	T1 z-BMI	.92 (.02)	[.88, .95]	.001			
	T1_Lon	.01 (.01)	[-.01, .04]	.433			
3	GENDER	.00 (.01)	[-.02, .03]	.982	.84	.00	$F(1,1014)=.47$, $p=.492$
	IS_T1	.00 (.02)	[-.05, .04]	.956			
	T1 z-BMI	.92 (.02)	[.88, .95]	.001			
	T1_Lon	.02 (.02)	[-.02, .05]	.280			
	$T1\_Lon^2$	-.01 (.01)	[-.02, .02]	.625			
4	GENDER	.00 (.01)	[-.02, .03]	.979	.84	.00	$F(1,1013)=.00$, $p=.979$
	IS_T1	.00 (.02)	[-.04, .04]	.947			
	T1 z-BMI	.92 (.02)	[.88, .95]	.001			
	T1_Lon	.02 (.02)	[-.02, .05]	.276			
	$T1\_Lon^2$	-.01 (.01)	[-.02, .02]	.624			
	T1_LonxGender	.00 (.01)	[-.01, .01]	.986			
5	GENDER	-.01 (.01)	[-.04, .02]	.656	.84	.00	$F(1,1012)=1.04$, $p=.308$
	IS_T1	.00(.02)	[-.05, .04]	.926			
	T1 z-BMI	.92 (.02)	[.88, .95]	.001			
	T1_Lon	.02 (.02)	[-.01, .05]	.257			
	$T1\_Lon^2$	-.01 (.01)	[-.02, .01]	.576			
	T1_LonxGender	.00 (.01)	[-.01, .01]	.974			
	$T1_Lon^2$xGender	.00 (.01)	[-.01, .02]	.494			

Note. T1=10 years old, T2=12 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model: F* (7,1012)=768.74, $p<.001$

Table S5. Bootstrapped Hierarchical Regression Analysis: T1 Loneliness as a Predictor of Dependent Variable T3 z-BMI including quadratic effects and interactions.

	Coefficients			Variance explained at each step		
Predictors	B (SE)	95% CI	*p*	R2	ΔR2	*F* Change
1 GENDER	.00 (.01)	[-.03, .03]	.870	.79	.79	$F(3,1016)=1261.25$, $p<.001$
IS_T1	-.02 (.02)	[-.07, .03]	.492			
T1 z-BMI	.89 (.02)	[.85, .93]	.001			
2 GENDER	.00 (.01)	[-.03, .03]	.963	.79	.00	$F(1,1015)=5.26$, $p=.022$
IS_T1	-.01 (.03)	[-.06, .04]	.643			
T1 z-BMI	.89 (.02)	[.85, .93]	.001			
T1_Lon	.03 (.01)	[.00, .06]	.060			
3 GENDER	.00 (.01)	[-.03, .03]	.933	.79	.00	$F(1,1014)=.35$, $p=.555$
IS_T1	-.01 (.03)	[-.06, .04]	.611			
T1 z-BMI	.89 (.02)	[.85, .93]	.001			
T1_Lon	.04 (.02)	[-.01, .08]	.101			
$T1\_Lon^2$	.00 (.01)	[-.02, .02]	.651			
4 GENDER	.00 (.01)	[-.03, .03]	.919	.79	.00	$F(1,1013)=1.66$, $p=.198$
IS_T1	-.01 (.03)	[-.06, .04]	.585			
T1 z-BMI	.89 (.02)	[.85, .93]	.001			
T1_Lon	.04 (.02)	[-.01, .08]	.094			
$T1\_Lon^2$	-.01 (.01)	[-.02, .02]	.633			
T1_LonxGender	.01 (.01)	[-.01, .02]	.313			
5 GENDER	-.01 (.02)	[-.05, .02]	.569	.79	.00	$F(1,1012)=1.32$, $p=.251$
IS_T1	-.01 (.03)	[-.06, .04]	.574			
T1 z-BMI	.89 (.02)	[.86, .93]	.001			
T1_Lon	.04 (.02)	[-.01, .08]	.085			
$T1\_Lon^2$	-.01 (.01)	[-.03, .02]	.577			
T1_LonxGender	.01 (.01)	[-.01, .02]	.322			
$T1_Lon^2$xGender	.01 (.01)	[-.01, .02]	.415			

Note. T1=10 years old; T3=13 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model:* $F(7,1012)=544.20$, $p<.001$

Table S6. Bootstrapped Hierarchical Regression Analysis: T2 Loneliness (Lon) as a Predictor of Dependent Variable T3 z-BMI including quadratic effects and interactions.

		Coefficients			Variance explained at each step		
Predictors		B (SE)	95% CI	*p*	R2	ΔR2	*F* Change
1	GENDER	.00 (.01)	[-.02, .02]	.951	.89	.89	$F(3,1030)=2827.98$, $p<.001$
	IS_T2	.01 (.02)	[-.02, .04]	.529			
	T2 z-BMI	.95 (.02)	[.92, .97]	.001			
2	GENDER	.00 (.01)	[-.02, .02]	.951	.89	.00	$F(1,1029)=1.99$, $p=.158$
	IS_T2	.01 (.02)	[-.02, .04]	.462			
	T2 z-BMI	.94 (.02)	[.91, .97]	.001			
	T2_Lon	.01 (.01)	[-.01, .03]	.253			
3	GENDER	.00 (.01)	[-.02, .02]	.952	.89	.00	$F(1,1028)=6.84$, $p=.009$
	IS_T2	.01 (.02)	[-.02, .04]	.516			
	T2 z-BMI	.95 (.02)	[.92, .97]	.001			
	T2_Lon	-.02 (.02)	[-.04, .02]	.390			
	$T2\_Lon^2$	.02 (.01)	[.00, .03]	.083			
4	GENDER	.00 (.01)	[-.02, .02]	.954	.89	.00	$F(1,1027)=.48$, $p=.490$
	IS_T2	.01 (.02)	[-.02, .04]	.502			
	T2 z-BMI	.95 (.02)	[.92, .97]	.001			
	T2_Lon	-.01 (.02)	[-.04, .02]	.423			
	$T2\_Lon^2$	.02 (.01)	[.00, .03]	.085			
	T2_LonxGender	.01 (.01)	[-.02, .03]	.586			
5	GENDER	-.03 (.01)	[-.05, .00]	.035	.89	.00	$F(1,1026)=11.37$, $p=.001$
	IS_T2	.01 (.02)	[-.02, .04]	.528			
	T2 z-BMI	.94 (.02)	[.91, .97]	.001			
	T2_Lon	-.01 (.02)	[-.04, .02]	.435			
	$T2\_Lon^2$	.01 (.01)	[.00, .02]	.069			
	T2_LonxGender	-.03 (.02)	[-.05, .01]	.044			
	$T2_Lon^2$xGender	.02 (.01)	[.00, .03]	.007			

Note. T2=12 years old; T3=13 years old; z-BMI created using respondents' BMI, self-reported age, Gender, and the external reference sample from WHO (Cole et al., 2000; de Onis et al., 2007); Lon= mean centred loneliness score; IS = Income Sufficiency; Bootstrap results are based on 1000 bootstrap samples*; Model:* $F(7,1026)=1234.70$, $p<.001$.

Appendix E Study 1 QLSCD Questionnaire Items

(Items 27c,27e and 27h were utilised)

Section bien-être de l'enfant – BEE

Objectif : Documenter la présence de troubles dépressifs chez l'E-C.

E11 – enfant de 116 à 127 mois volet 2008	E13– enfant de 140 à 151 mois volet 2010	Sources – justifications – commentaires
27a, 27b, 27c, 27d, 27e, 27f, 27g, 27h	1a, 1b, 1c, 1d, 1e, 1f, 1g, 1h, **1i**, **1j**, **1k**, **1l**, **1m**, **1n**	**Volet 2008:** **27a** à **27h**: nouvelles. Adaptées par M. Boivin et F. Vitaro (GRIP) et tirées du *Children depression Inventory* (CDI – Kovacs, 1985). Cette échelle permet d'évaluer la symptomatologie dépressive des enfants. **Volet 2010 :** **1i** à **1n** : nouvelles. Proposées par l'équipe de l'ÉLDEQ-DELS et tirées du *Children depression Inventory* (CDI – Kovacs, 1985) afin de compléter le CDI-court officiel.

on de l'enfant avec autrui 1/2

Nom de la variable	Source(s)	Catégories	Construction et remarques
VQEED37 *Échelle de somnolence d'Epworth - 2 catégories (tel que rapporté par le jeune)*	QELJ (**E22** : vsonq17a à 17h)	1) Pas de somnolence diurne excessive 2) Somnolence diurne excessive	Variable disponible pour le volet 2019. Cet indice est construit à partir de la variable « *Échelle de somnolence d'Epworth* » (xQEES37) en regroupant le score de la façon suivante : Si 0≤ Score ≤ 10 = 1; Si 11≤ Score ≤ 24 = 2;
Estime de soi			
NQEET13 *Estime de soi de l'enfant (tel que rapporté par l'enfant)*	QIE (**E14** : nednq1a à 1e)	Échelle de 0 à 10	Variable calculée pour le volet 2011 seulement. Si le nombre de réponses valides aux 5 items retenus est égal ou supérieur à 4, alors on calcule le score moyen. Les scores sont ramenés à une échelle variant entre 0 et 10.
Dépression et personnalité			
KQEET10, MQEET10, NQEET10 *Niveau de symptômes dépressifs de l'enfant (tel que rapporté par l'enfant)*	QPAE (**E11** : kqeeq27a à 27h) QIE (**E13** : mbinq1a à 1h) (**E14** : nbinq1a, à 1h)	Échelle de 0 à 10	Variable calculée pour les volets 2008, 2010 et 2011 de la façon suivante : On soustrait 1 de chacun des items afin d'obtenir une valeur minimale de 0 pour chacun d'entre eux. Puis on inverse la valeur de certains items : E11 : q27b, 27c, 27d, 27f, 27g, 27h. E13 : q1b, 1c, 1d, 1f, 1g, 1h. E14 : q1b, 1c, 1d, 1f, 1g, 1h. Si le nombre de réponses valides aux x items retenus est égal ou supérieur à x-y (où y = le nombre possible de variables manquantes déterminé par l'équipe de recherche responsable de la construction de l'échelle), alors on calcule le score moyen obtenu. Les scores sont ramenés à une échelle variant entre 0 et 10. Sinon, xQEET10 est indéterminée.

Appendix F Study 1 Descriptive Data/ Skew and Kurtosis

Total Loneliness (N=1042)	Range	Minimum	Maximum	Mean	SD	Skewness	SE	Kurtosis
T1 AGE 10 (E11)	8.00	1.00	9.00	3.85	1.21	1.449	0.076	1.77
T2 AGE 12 (E13)	9.00	0.00	9.00	3.78	1.17	1.570	0.076	2.19
T3 AGE 13 (E14)	9.00	0.00	9.00	3.88	1.29	1.502	0.076	1.97

Note: Skewness Kurtosis SE = .15

Appendix G Study 2 Methodology

G1 Recruitment Poster

Body Size and Attention Study

uclan
School of Psychology

My name is Ruth Hurley, and I am looking for volunteers to help participate in my PhD study at the University of Central Lancashire, Preston campus.

Are you 25-40 years old with 2 hours free?

Receive a **£10 AMAZON** voucher when you take part! (or 8 SONA points)

The session includes:

1. 3 Button-press computer tasks (while wearing an fNIRS cap to measure brain activity).
2. Questionnaires about your thoughts and emotions.
3. Measures of your weight, height and waist circumference.

To volunteer or for more information, contact Ruth

Email: rhurley2@uclan.ac.uk **Phone:** 01772 893871

This project is being supervised by: Professor Janice Abbott (jabbott@uclan.ac.uk) and Dr. Lea Pilgrim (lpilgrim@uclan.ac.uk), University of Central Lancashire, School of Psychology, Darwin Building, Preston PR12HE (Poster Date: March 2019)

G2 Participant information sheet1

School of Psychology
University of Central Lancashire,
Darwin Building, Preston PRI 2HE

Investigating the Influence of Body Size on Attention

To whom it may concern,

My name is Ruth Hurley, and I am conducting this research as part of my PhD thesis, under the supervision of Professor Janice Abbott and Dr. Lea Pilgrim. This information sheet is intended to give an overview of the study to help you decide if you would like to take part. If there is anything that is not clear or if you would like more information, feel free to talk to me before deciding.

What is the purpose of the study?

The purpose of this study is to gain a greater understanding of whether body size influences attention and which areas of the brain are most and least active during different attention tasks. There is also some research that indicates your mood may affect how much attention you have available to put into a task. We hope that the results of this research can help us to learn more about individual differences in attention whether these differences could be related to body size and mood.

Why have I been invited to participate?

We are inviting people aged 25 to 40 to take part in the study. You must have corrected, or corrected to normal vision and be able to read English. This is because the tasks involve looking at shapes and instructions presented on a computer screen. If you wear glasses, please bring them with you.

You should **not** take part if you have taken recreational drugs, or stimulant/ sedative medication that would affect your attention in the 12 hours before your appointment.

Do I have to take part?

No, it is up to you to decide whether or not to take part. If you decide to, we will book you in for a time slot. On the day of the study you will be asked to sign a consent form, you are still free to withdraw from the study up until you leave, and you can do this without giving a reason.

If you are a current UCLan student, we would like to reassure you that by choosing to either take part or not take part in the study will have no impact on your marks, assessments or future studies.

Will my data be kept confidential?

Yes. All information gathered during this study is kept strictly confidential, and stored securely at the School of Psychology at the University of Central Lancashire in accordance with General Data Protection Regulations.

Ruth Hurley

The Influence of Body Size and Attention

Questionnaire Pack

Participant number:_____

Stimulants/ Sedatives

Have you taken any medication that might affect your attention (stimulants or sedatives) in the last 12 hours? Y/ N (If Y please check with the researcher)

How many caffeine drinks have you had in the last 12 hrs? _____

Demographics

Age [ ] Sex* [M] [F]

*Please circle your sex at birth

Ethnicity

Studies have shown that BMI thresholds are different for people in different ethnic groups due to the natural size and shape of their bodies. Please could you tick the ethnic group that you feel most closely represents you. If you have a mixed family origin, please just choose the group that you feel most closely represents your body type.

Ethnic Group	
☐ African-Caribbean/ Black Caribbean/ Black African	For example, family origin from the Caribbean islands, African nations, sub-Saharan African, African-American.
☐ Asian/ Chinese	For example, family origin from China, Taiwan, Singapore and Hong Kong.
☐ Middle-Eastern	For example, family origin from Egypt, Iran, Iraq, Jordan, Kuwait, Lebanon, Oman, Qatar, Saudi Arabia, Syria, the United Arab Emirates and Yemen.
☐ White European or White Other	For example, family origin from Europe, North America, Australia.
☐ South Asian	For example, family origin from Bangladesh, Bhutan, India, Indian-Caribbean, Maldives, Nepal, Pakistan and Sri Lanka

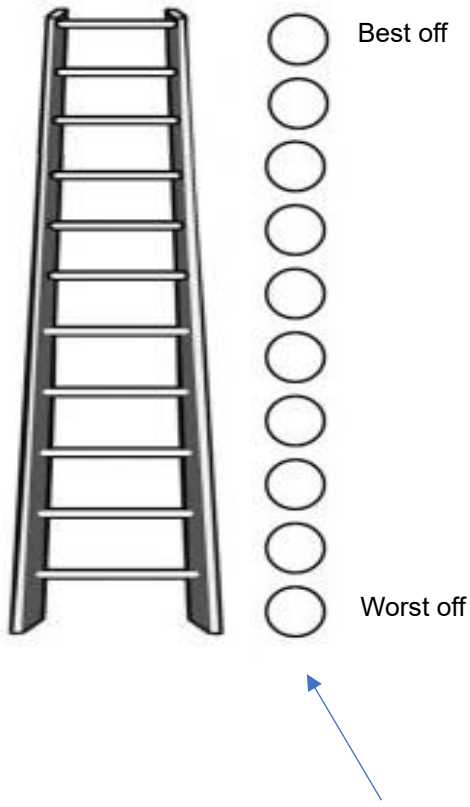
Subjective Social Status, MacArthur Scale

Think of this ladder as representing where people stand in the United Kingdom.

At the top of the ladder are the people who are the best off, those who have the most money, the most education, and the most respected jobs.

At the bottom are the people who are worst off, who have the least money, least education, and the least respected jobs or no job.

The higher up you are on this ladder, the closer you are to the people at the very top and the lower you are, the closer you are to the people at the very bottom.



Where would you place yourself on this ladder? (please tick the preferred circle).

Edinburgh Handedness Inventory[1]

Please indicate with a check (✓) **your preference** in using your left or right hand in the following tasks.

Where the preference is so strong you would **never use the other hand**, unless absolutely forced to, put two checks (✓✓).

If you are **indifferent**, put one check in each column (✓ | ✓).

Some of the activities require both hands. In these cases, the part of the task or object for which hand preference is wanted is indicated in parentheses.

	Task / Object	Left Hand	Right Hand
	1. Writing		
	2. Drawing		
	3. Throwing		
	4. Scissors		
	5. Toothbrush		
	6. Knife (without fork)		
	7. Spoon		
	8. Broom (upper hand)		
	9. Striking a Match (match)		
	10. Opening a Box (lid)		
	Total checks:	LH =	RH =
Researcher to complete	Cumulative Total	CT = LH + RH =	
	Difference	D = RH – LH =	
	Result	R = (D / CT) × 100 =	
	Interpretation: (Left Handed: R < -40) (Ambidextrous: -40 ≤ R ≤ +40) (Right Handed: R > +40)		

[1] Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsycholoolgia*, *9*, 97-113.

Ravens Progressive Matrices (Short form)

(See the researcher for the stimuli for this task)

1. _____ Practice
2. _____ Practice
3. _____ Practice
4. _____
5. _____
6. _____
7. _____
8. _____
9. _____
10. _____
11. _____
12. _____
13. _____
14. _____
15. _____

Practice (Items 1-3) _____

Test (Items 4-15) _____

BRIEF®-A

Behavior Rating Inventory of Executive Function®– Adult Version

SELF-REPORT FORM

Robert M. Roth, PhD, Peter K. Isquith, PhD, and Gerard A. Gioia, PhD

Instructions

On the following pages is a list of statements. We would like to know if you have had problems with these behaviors over the past month. Please answer all the items the best that you can. Please DO NOT SKIP ANY ITEMS. Indicate your response by circling

N	if the behavior is	**Never** a problem
S	if the behavior is	**Sometimes** a problem
O	if the behavior is	**Often** a problem

For example, if you **never** have trouble making decisions, you would circle **N** for this item:

I have trouble making decisions (N) S O

If you make a mistake or want to change your answer, DO NOT ERASE. Draw an "X" through the answer you want to change, and then circle the correct answer:

I have trouble making decisions (X) (S) O

BRIEF_A P1

Level of Education: ☐ Less than High School ☐ High School ☐ College ☐ Degree ☐ Masters/PhD

During the past month, how often has each of the following behaviors been a *problem*?

N = Never S = Sometimes O = Often

1.	I have angry outbursts	N	S	O
2.	I make careless errors when completing tasks	N	S	O
3.	I am disorganized	N	S	O
4.	I have trouble concentrating on tasks (such as chores, reading, or work)	N	S	O
5.	I tap my fingers or bounce my legs	N	S	O
6.	I need to be reminded to begin a task even when I am willing	N	S	O
7.	I have a messy closet	N	S	O
8.	I have trouble changing from one activity or task to another	N	S	O
9.	I get overwhelmed by large tasks	N	S	O
10.	I forget my name	N	S	O
11.	I have trouble with jobs or tasks that have more than one step	N	S	O
12.	I overreact emotionally	N	S	O
13.	I don't notice when I cause others to feel bad or get mad until it is too late	N	S	O
14.	I have trouble getting ready for the day	N	S	O
15.	I have trouble prioritizing activities	N	S	O
16.	I have trouble sitting still	N	S	O
17.	I forget what I am doing in the middle of things	N	S	O
18.	I don't check my work for mistakes	N	S	O
19.	I have emotional outbursts for little reason	N	S	O
20.	I lie around the house a lot	N	S	O
21.	I start tasks (such as cooking, projects) without the right materials	N	S	O
22.	I have trouble accepting different ways to solve problems with work, friends, or tasks	N	S	O
23.	I talk at the wrong time	N	S	O
24.	I misjudge how difficult or easy tasks will be	N	S	O
25.	I have problems getting started on my own	N	S	O
26.	I have trouble staying on the same topic when talking	N	S	O
27.	I get tired	N	S	O
28.	I react more emotionally to situations than my friends	N	S	O
29.	I have problems waiting my turn	N	S	O
30.	People say that I am disorganized	N	S	O
31.	I lose things (such as keys, money, wallet, homework, etc.)	N	S	O
32.	I have trouble thinking of a different way to solve a problem when stuck	N	S	O
33.	I overreact to small problems	N	S	O
34.	I don't plan ahead for future activities	N	S	O
35.	I have a short attention span	N	S	O
36.	I make inappropriate sexual comments	N	S	O
37.	When people seem upset with me, I don't understand why	N	S	O
38.	I have trouble counting to three	N	S	O

During the past month, how often has each of the following behaviors been a *problem*?

N = Never S = Sometimes O = Often

39. I have unrealistic goals	N	S	O
40. I leave the bathroom a mess	N	S	O
41. I make careless mistakes	N	S	O
42. I get emotionally upset easily	N	S	O
43. I make decisions that get me into trouble (legally, financially, socially)	N	S	O
44. I am bothered by having to deal with changes	N	S	O
45. I have difficulty getting excited about things	N	S	O
46. I forget instructions easily	N	S	O
47. I have good ideas but cannot get them on paper	N	S	O
48. I make mistakes	N	S	O
49. I have trouble getting started on tasks	N	S	O
50. I say things without thinking	N	S	O
51. My anger is intense but ends quickly	N	S	O
52. I have trouble finishing tasks (such as chores, work)	N	S	O
53. I start things at the last minute (such as assignments, chores, tasks)	N	S	O
54. I have difficulty finishing a task on my own	N	S	O
55. People say that I am easily distracted	N	S	O
56. I have trouble remembering things, even for a few minutes (such as directions, phone numbers)	N	S	O
57. People say that I am too emotional	N	S	O
58. I rush through things	N	S	O
59. I get annoyed	N	S	O
60. I leave my room or home a mess	N	S	O
61. I get disturbed by unexpected changes in my daily routine	N	S	O
62. I have trouble coming up with ideas for what to do with my free time	N	S	O
63. I don't plan ahead for tasks	N	S	O
64. People say that I don't think before acting	N	S	O
65. I have trouble finding things in my room, closet, or desk	N	S	O
66. I have problems organizing activities	N	S	O
67. After having a problem, I don't get over it easily	N	S	O
68. I have trouble doing more than one thing at a time	N	S	O
69. My mood changes frequently	N	S	O
70. I don't think about consequences before doing something	N	S	O
71. I have trouble organizing work	N	S	O
72. I get upset quickly or easily over little things	N	S	O
73. I am impulsive	N	S	O
74. I don't pick up after myself	N	S	O
75. I have problems completing my work	N	S	O

PHQ-8

Over the last 2 weeks, how often have you been bothered by any of the following problems?

	Not at all	Several days	More than half the days	Nearly every day
1.Little interest or pleasure in doing things	0	1	2	3
2.Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself - or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed? Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3

From the Primary Care Evaluation of Mental Disorders Patient Health Questionnaire (PRIME-MD PHQ). The PHQ was developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues. For research information, contact Dr. Spitzer at rls8@columbia.edu. PRIME-MD® is a trademark of Pfizer Inc. Copyright© 1999 Pfizer Inc. All rights reserved. Reproduced with permission

GAD-7

Over the last 2 weeks, how often have you been bothered by the following problems?

	Not at all	Several days	More than half the days	Nearly every day
1.Feeling nervous, anxious or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?

0 Not difficult at all	1 Somewhat difficult	2 Very difficult	3 Extremely difficult
☐	☐	☐	☐

From the Primary Care Evaluation of Mental Disorders Patient Health Questionnaire (PRIME-MD PHQ). The PHQ was developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues. For research information, contact Dr. Spitzer at rls8@columbia.edu. PRIME-MD® is a trademark of Pfizer Inc. Copyright© 1999 Pfizer Inc. All rights reserved. Reproduced with permission

RRS

Instructions to participants:

People think and do many different things when they feel sad, blue, or depressed. Here are a list of possibilities. Please *circle* if you never, sometimes, often, or always think or do each one when you feel down, sad, or depressed.

Please indicate what you generally do, not what you think you should do.

	Almost Never			Almost Always
1. Think “What am I doing to deserve this?”	1	2	3	4
2. Analyze recent events to try to understand why you are depressed	1	2	3	4
3. Think “Why do I always react this way?”	1	2	3	4
4. Go away by yourself and think about why you feel this way	1	2	3	4
5. Write down what you are thinking and analyze it	1	2	3	4
6. Think about a recent situation, wishing it had gone better	1	2	3	4
7. Think “Why do I have problems other people don’t have?”	1	2	3	4
8. Think “Why can’t I handle things better?”	1	2	3	4
9. Analyze your personality to try to understand why you are depressed	1	2	3	4
10. Go someplace alone to think about your feelings	1	2	3	4

Thank you!

You have now completed the questionnaires and paper-based tasks.

Please hand this pack back to the researcher.

To be completed by the researcher

HCirc:_____ N to I :_____ E to E:_____

Order of completion:

	Resting State Scan
1 / 2	Shapes Task (CPT) with NIRS scan
A / B	Squares Task (ST) with NIRS scan

Optode Issues

Anthropometry

	Height	Weight	Waist
M2			
M3			

Free gift confirmation:

As a thank you for your time participants can chose to receive either a

☐ £10 AMAZON Gift card **OR** ☐ 12 SONA Points (UCLan Psychology students)

Appendix G4 Anthropometric Procedures

The testing took place in a private room and the general procedure was explained. Participants were asked to remove any bulky outer clothing, shoes and belts and empty any pockets. The height and weight measures were taken twice, if measurements varied by more than 0.2 cm for height or 0.2 kg for weight, a third measurement was taken (the measures were averaged) in line with guidance by WHO (2017).

Height. Height was measured using a standiometer (standing measure). Participants were asked to stand up straight and tall with their back (scapulae), feet together, heels in contact with the measure, and weight evenly distributed. They were asked to look straight ahead, and the head angle was checked to be in the Frankfort plain (bottom of the eye socket and the external opening of the ear canal in a horizontal line). The measure was taken while the participant held an in-breath by measuring horizontally at the uppermost point of the head (the crown) to the nearest 0.1cm.

Weight. Was measured using digital weighting scale (Salter Glass Analyser Scale 9141) placed on a firm flat surface and measures were taken per guidance by WHO (2017). The scale was turned on and observed to display 0.0 Kg. Participants were asked to stand still in the centre of the scale, facing forwards with body weight evenly distributed between both feet, hands by their sides (palms facing their thighs). The weight was recorded to the nearest 0.1kg.

Waist Circumference. This was taken using a non-elastic (SECA 201 constant tension measuring tape) at the mid-point between the lower rib and upper hip bone (iliac crest) per NICE and WHO guidelines. If these points were not easily identifiable, waist circumference was taken at the umbilical level. Measures were taken in line with WHO, (2008) and Misra et al., (2005). Participants were asked to stand up straight with their weight evenly distributed and feet close together. The participant was asked to pass the measuring tape behind them, the researcher then took the tape and. The researcher located the lowest rib and the top of the hip bone. If necessary, the participant was asked to help locate these areas. The tape was held midway between the two points positioned snugly (without compressing the skin) parallel to the floor. After a few normal breaths, the measurement was taken at the end of a normal expiration, measuring to the nearest 0.1cm.

Appendix H A Priori Power Analysis (sample size estimation in GPower)

(Mayr et al., 2007)

Figure G1 A Priori Power Analysis for Correlation

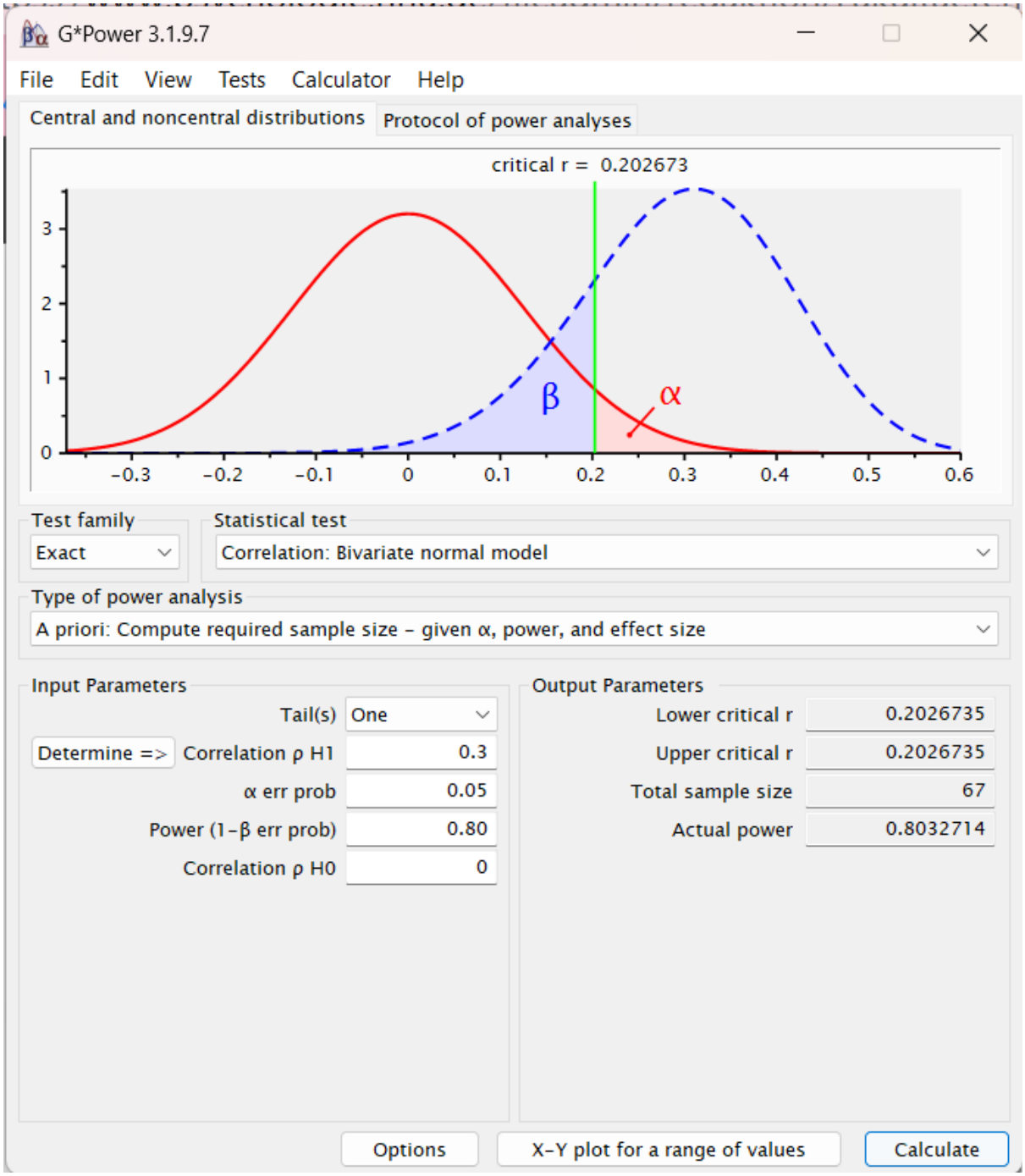


Figure G2 A Priori Power Analysis for Correlation

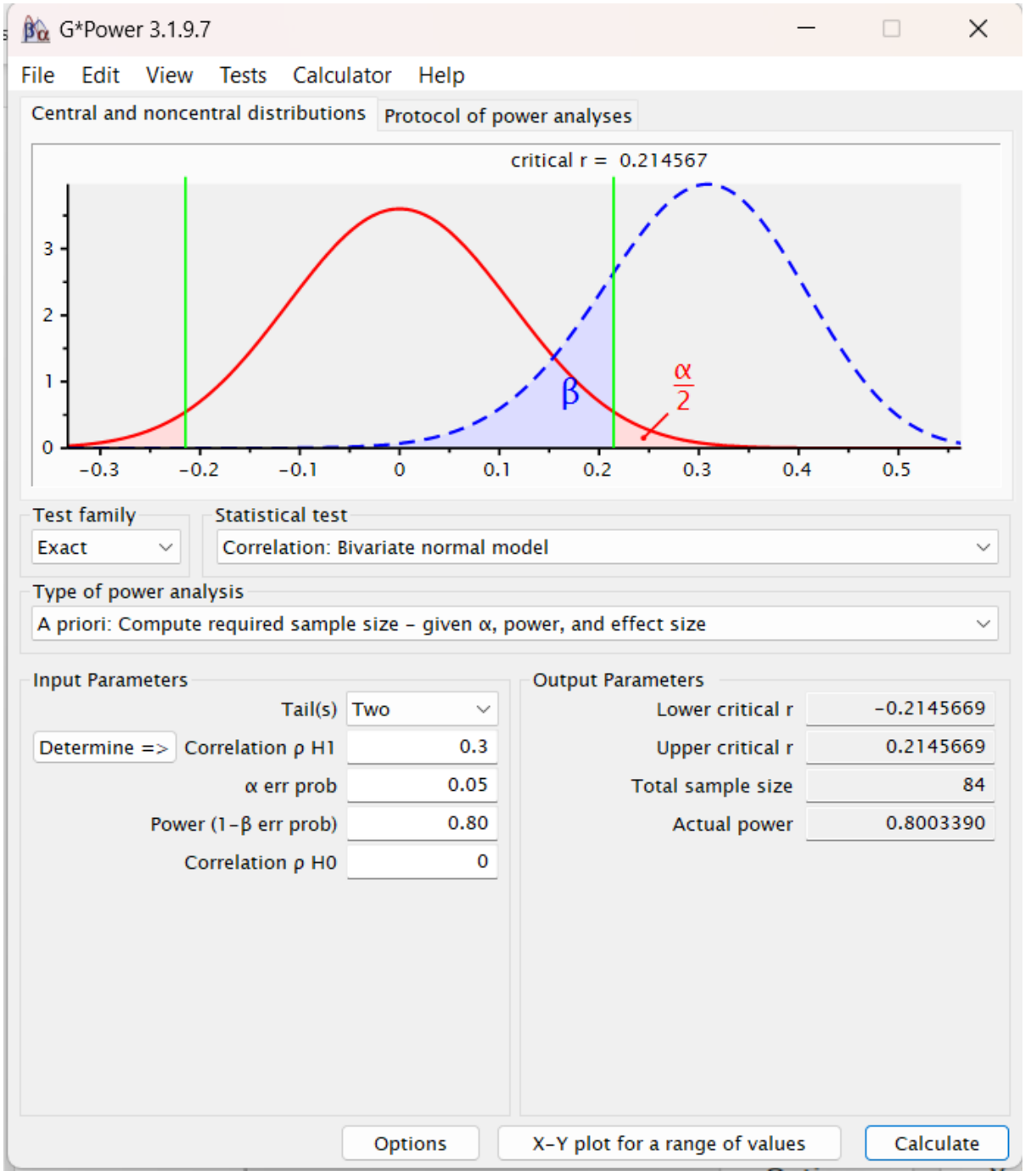
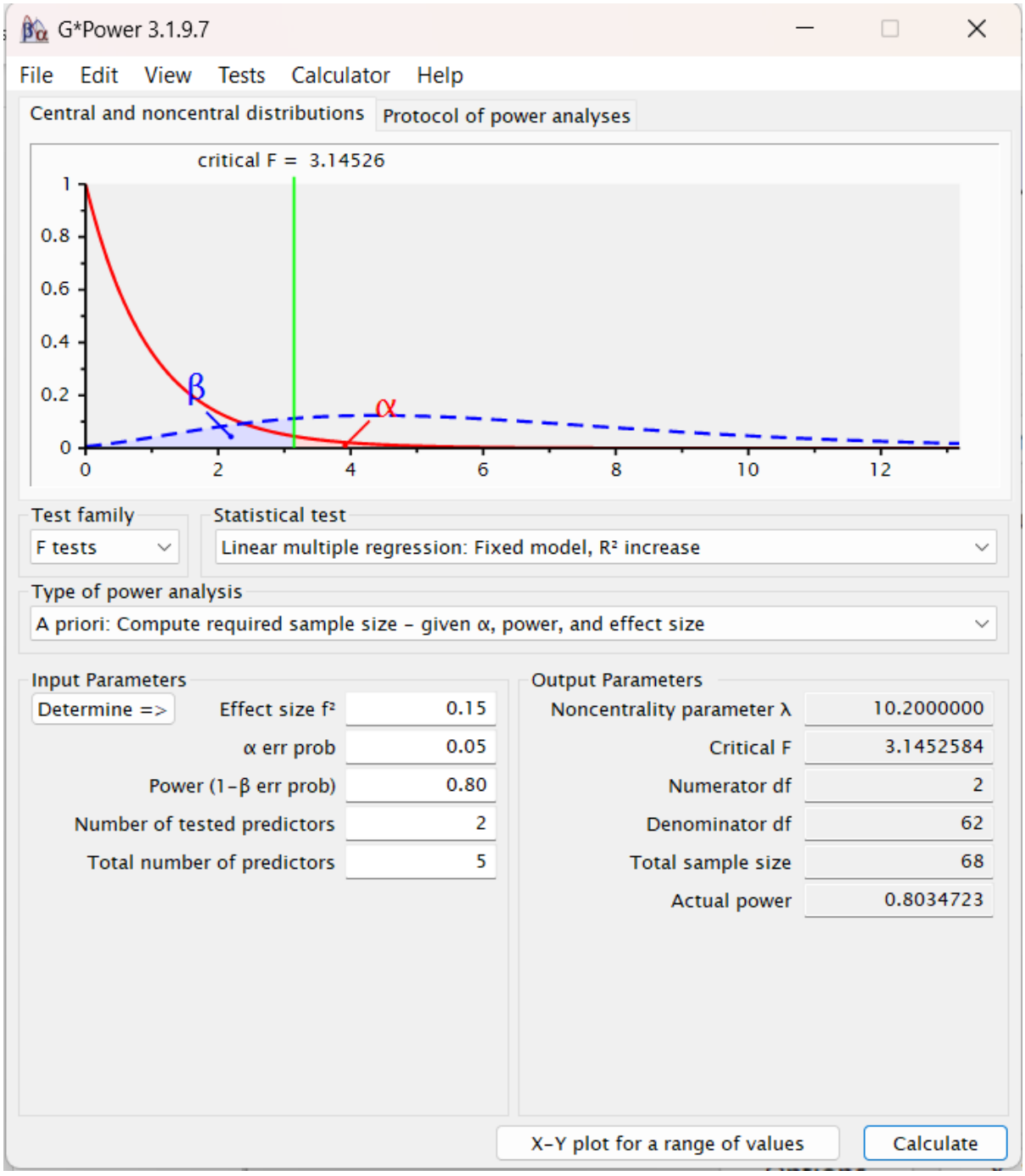


Figure G2 A Priori Power Analysis for Regression



Appendix I Anthropometric Categorical Variables (full sample)

BMI Category. 5 Levels (See Table H1)

BMI Binary. 2 Levels (1=normal BMI<30; 2=Increased 30+)

Waist Circumference Cat. 2 levels: (1=Low/normal or 2=increased risk males >=94cm, females >= 80cm)

Table I1,
Anthropometric Categorical Coding Levels and Participant Numbers Study 2 (N=90)

	UW <18.50	NW 18.50-24.99	OW 25.00-29.99	Ob 30.00 to 39.99	Mob 40.00+
BMI 5 Cat	2 (2%)*	37 (40%)	26 (28%)	21 (23%)	4 (4%)*
BMI 3 Cat	39 (43%)		26 (29%)	25 (28%)	
BMI Binary	Normal-Increased risk of comorbidities (<30) 65 (72%)			High risk of comorbidities (>=30) 25 (28%)	
Waist Circumference Risk (Binary)	Normal 41(46%)		Increased 49(54%)		

**UW and Mob were too small for analysis.*

Table I2

Mental Health Categorical Frequency and Variable Coding (N=90).

N=90	**1=V Low (0-5)**	**2=Low (6-10)**	**3=Moderate Clinical (11-15)**	**4=High Clinical (16 to max)**
ANX_Cat	49 (53%)	21 (23%)	13 (14%)	7 (8%)
DEP_Cat	48 (53)	20 (22%)	12 (13%)	10 (11%)
ANX_Clin (binary)	70 (78%)		20 (22%)	
DEP_Clin (binary)	68 (76%)		22 (24%)	

Table I3

Repetitive Negative Thinking Categorical Frequency and Variable Coding (N=90).

N=90	**1=Low (5-10)**	**2= Moderate (11-15)**	**3=High (6-20)**
RUMb (Brooding)	50 (56%)	24 (27%)	16 (18%)
RUMr (Reflective)	46 (51%)	33 (37%)	11 (12%)
Total Rumination RUMt Binary	**Low 46(51%)**	**Moderate-High 44 (49%)**	
RUM b Binary	**Low 50(56%)**	**Moderate-High** 40(44%)	
RUM r Binary	**Low** 46(51%)	**Moderate-High** 44(49%)	

Table I4

Repetitive Negative Thinking Categorical Frequency and Variable Coding (N=90).

N=90	**1=V Low (not at all)**	**2=Low (Several Days)**	**3=Moderate (> Half the Days)**	**4=High Clinical (Nearly every day)**
Worry	21 (23%)	38(42%)	19(21%)	12(13%)
Worry (Binary)	Low 78(87%)		Moderate-High 12(13%)	

Appendix J BRIEF Subscales Items & Definition

The Subscales of the Behaviour Ratings Inventory of Executive Function Roth et al., (2005)

The scale has 75 statements detailing problems in daily living. Participants were asked to state how often this was a problem in the last month (Never, Sometimes or Often). T-scores of 65 or greater indicate potentially clinically significant problems.

Subscales
Inhibitory Control. Items 5,16,29,36,43,55,58,73 Ability to inhibit, resist, or not act on impulse. Core deficit in ADD, underlying deficit in executive dysfunction, disinhibited behaviour as a result of brain injury.
Shift. Items 8, 22, 32, 44, 61, 67 Ability to make transitions of thought, problem solve flexibly, switch or alternate attention and behaviour. Core deficit in Aspergers (High functioning autism) e.g. perseverative problem-solving (getting 'stuck' on a specific idea and unable to move past it).
Emotional Control. Items 1, 12, 19, 28, 33, 42, 51, 57, 69, 72 Ability to regulate emotional responses. Excessive emotional reactions or changeability in emotions (lability).
Self-Monitor. Items. 13, 23, 37, 50, 64, 70 Awareness of one's own social behaviour and comprehension of its effect on others.
Initiate. Items 6,14,20,25,45,49,53,62 Ability to initiate tasks or problem-solving strategies (not by opposition). Issue in those with frontal lobe injury (requiring extra cues and prompts).
Working Memory. Items 4,11,17,26,35, 46,56,68 Ability to carry out multistep activities or implement a sequence of actions/instructions. Core component of cognitive dysfunction (Barkley 1997a). Strongly related to sustained attention ability.
Plan/Organise. Items 9, 15, 21, 34, 39, 47, 54, 63, 66, 71 Ability to plan, prioritise and anticipate future events and prepare for an activity ahead of time to meet a goal. Impacts learning, memory, ability to utilise information in different contexts. Integral in executive dysfunction.
Task Monitor. 2, 18, 24, 41, 52, 75 Awareness of the extent of one's errors in problem solving /activities.
Org. of Materials. Items 3, 7, 30, 31, 40, 60, 65, 74 Ability to maintain orderliness in one's physical environment (working and living spaces).

Behavioural Regulation Index Maintain regulatory control of behaviour and emotion. Composed of Inhibit, Shift, Emotional Control, Self Monitor.
Metacognition Index Ability to systematically problem solve via planning and organisation. Composed of Initiate, Plan/Organise, Working Memory Task monitor and Organisation of materials.

Appendix K Study 2 Treatment of Data

Table K1 Coding and Calculation of variables

1=African-Caribbean/ Black Caribbean/ Black African; 2= Asian/ Chinese; 3=

Middle-Eastern; 4= White European or White Other; 5= South Asian 6= Other

Demographic Variables	
Age (years)	Level of Education (5 Levels coded 1-5) 1=High School; 2=College; 3=Degree; 4=Masters; 5=Doctoral
Gender (1=Male; 2=Female)	Fluid Intelligence (Ravens total task items correct 0-12).
Ethnicity (6 Levels coded 1-6) 1=African-Caribbean/ Black Caribbean/ Black African; 2= Asian/ Chinese; 3= Middle-Eastern; 4= White European or White Other; 5= South Asian 6= Other	No. Caffeine drinks (in the last 12 hours)
Subjective Social Status (MacArthur Scale) Worst off to Best off, coded 1-10, (10 is high)	

Anthropometric		
BMI Raw	Continuous	Raw (non-normalised)
BMI Category	4 Levels	-1=UW, 1=NW, 2=OW, 3=Ob, 4=Mob NB UW and Mob are too small for analysis
BMI 3Cat	3 Levels	1=UW/NW, 2=OW, 3=Ob/Mob
BMI Binary	2 Levels	1=normal/OW BMI<30; 2=Increased 30+
WC	Continuous	
WC Risk Category	2 Levels	1=Low/normal, 2=increased risk (males>94cm; females >80cm)
BMI High Low15	2 Levels	1=Low/normal <35, 2=increased risk 35+
Mental Health		
Depression **Anxiety**	Continuous	PHQ-8 Dep Total Score 0-24 GAD-7 Anx Total Score 0-21

Depression Cat **Anxiety** Cat	4 Levels	1= V Low (0-5); 2=Low (6-10); 3=Moderate Clinical (11-15) 4=High Clinical (16+)
Depression Clinical **Anxiety Clinical**	2 Levels	Above and below clinical cut off (1= <10; 2=11+)
Repetitive Negative Thinking		
Worry	Continuous	GAD item 2 and 3 summed (total 0-6) CODING: 0=none, 1=some, 2= >half days, 3=nearly every day
Worry Cat	4 levels	0=none, 1=1-2=some, 2=3-4 >half days, 3=5-6=nearly every day
Worry Binary Cat	2 Levels	1 low (none & some, 0-2), 2= high (>half days & nearly every day, 4-6)
Rumination Total, Brooding, Reflective	Continuous	Total score (1-40) Sub Scales brooding / reflective (1-20)
Rumination Cat RUMt, RUMb, RUMr	3 Levels	Total (1 mild =10-20, 2 moderate =21-30, 3 high =31-40) Sub Scale brooding or reflective (1 mild=5-10, 2 moderate=11-15, 3 high=16-20) *NB low n for high rumination responses.*
Rumination Binary Cat RUMt, RUMb, RUMr	2 Levels	1= mild (10-20), 2=moderate/high (21-40)
Cognitive Performance		
Task-Related Variables /Counterbalancing		
Handedness	3 Levels	Total score (R): 3= Left Handed: R < -40; 2= Ambidextrous: -40 ≤ R ≤ +40 1= Right Handed: R > +40)
Response version	2 Levels	1=Version A (target red square on left of screen), 2=Version B (target red square on right or screen)
Task order	2 Levels	1= CPT first, 2=Simons Task first
Simons Task (DV)		
E-Prime data for reaction time, accuracy and congruences was extracted and used to calculate the following in Excel. RT<150 were removed. Accuracy converted to		

Error and Error was converted to percentage Error. Analyses were completed on accurate trials.		
RT Block Mean	2 Levels	Block 1; Block 2 Average RT for accurate trials RT>150ms from stimuli onset.
RT Overall Mean	Continuous	Average RT for accurate trials in Block 1 & Block 2
RT I-C Block Mean	2 Levels	(If Incongruent trials are disadvantaged e.g. higher errors, the total will be a positive figure)
RT I-C Overall Mean	Continuous	
% Err Block Total	2 Levels	Total errors per block as a percentage of trials
Continuous Performance Task (DV)		
RT Block Mean	2 Levels	Average RT for accurate trials RT>150ms from stimuli onset
RT Overall Mean	Continuous	
% Err Block Total	2 Levels	Total errors per block as a percentage of trials
% Err Overall Mean	Continuous	
%FTP Error per block	2 Levels	Omission or 'Fail to press' errors
%FTP Error Overall	Continuous	
%SHP Error per block	2 Levels	Commission or 'Should not have pressed' errors
%SHP Error Overall	Continuous	
Self-Report: Behaviour Ratings of Executive Function (BRIEF) T-Score		
Participants rated how often they experience 75 Daily living problems (1= Never, 2=Sometimes or 3=Often. Sub scores were totals of relevant items (see Appendix J BRIEF Subscales Items & Definition)		
Inhibition T-Score	Continuous	Total items and participant age used to extract t-scores from handbook
Inhibition Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Shift	Continuous	
Shift Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Emotional control	Continuous	
Emotional control Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Self-Monitor	Continuous	
Self-Monitor Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Initiate	Continuous	
Initiate Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical

Working Memory	Continuous	
Working Memory Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Plan/Organise	Continuous	
Plan/Organise Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Task Monitor	Continuous	
Task Monitor Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Org. of Materials	Continuous	
Org. of Materials Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Behavioural Regulation Index	Continuous	
Behavioural Regulation Index Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Metacognition **Index**	Continuous	
Metacognition **Index Clinical**	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Global Executive **Composite**	Continuous	
Global Executive Composite Clinical	2 Levels	0=<65 Nonclinical; 1=65+ Clinical
Neurocognitive		
fNIRS RS Scan (DV)		
Oxyhaemoglobin (HbO2) Average (7 min) Right Frontal Channels 1-5 Left Frontal Channels 1-5 Right Temporal Channels 1-3 Left Temporal Channels 1-3	Continuous	Brain tissue changes in oxygenated haemoglobin concentration (HbO_2) Brain tissue changes in deoxygenated haemoglobin concentration (HHb)
Regional Oxyhaemoglobin Right Frontal Mean Left Frontal Mean Right Temporal Mean Left Temporal Mean	4 Levels	**Regional average – regional average baseline**

fNIRS CPT Task Scan (DV)		
fNIRS Simons Task Scan (DV) Baseline was the 20 second rest period immediately before the task block. Average rest minus average task was calculated for each measurement channel. Regional averages were calculated for RF, LF, RT, LT.		
Oxyhaemoglobin (HbO2) Average over task Epochs **Right Frontal Channels 1-5** **Left Frontal Channels 1-5** **Right Temporal Channels 1-3** **Left Temporal Channels 1-3**	Continuous	**Brain tissue changes in oxygenated haemoglobin concentration (HbO2)** **Brain tissue changes in deoxygenated haemoglobin concentration (HHb)**
Regional Oxyhaemoglobin **Right Frontal Mean** **Left Frontal Mean** **Right Temporal Mean** **Left Temporal Mean**	4 Levels	**Regional average – regional average baseline**

Appendix L Neurocognitive Task Measurement Epochs

Table L1 CPT Task measurement epochs (for fNIRS)

Participant activity	Variable	Definition	Marker or stimuli onset
Resting	BASE_R1	20 second baseline between practice and block1 of the task	R1 to R1+20
	R2	20 sec between block1 and block2	R2 to R2+20
	R3	20 sec between block2 and block3	R3 to R3+20
	R4	20 sec between block3 and block4	R4 to R4+20
	BaseTOT	Total of activity during all baseline (rest) measures	
	BaseMEAN	Mean of activity during all baseline (rest) measures	
On task	BLOCK1	Stimuli 20 to 100 = 81	O20 to R2
	BLOCK2	Stimuli 101 to 181 = 81	O101 to R3
	BLOCK3	Stimuli 182 to 262 = 81	O182 to R4
	BLOCK4	Stimuli 263 to 343 = 81	O263 to R5 or o343+1.6
	BlockTOT	Total of activity during all task (experiment) measures	
	BlockMEAN	Mean of activity during all task (experiment) measures	

Table L2 ST Task Measurement Epochs

Resting	BASE_R1	20 second baseline between practice and block1 of the task	R1 to R1+20
	R2	20 sec between block1 and block2	R2 to R2+20
	R3	20 sec between block2 and block3	R3 to R3+20
	R4	20 sec between block3 and block4	R4 to R4+20
	R5	20 sec after block 4	R5 to R5+20
	BaseTOT	Total of activity during all baseline (rest) measures	
	BaseMEAN	Mean of activity during all baseline (rest) measures	

On task	BLOCK1	Stimuli 17 to 96 (80)	O20 to R2
	BLOCK2	Stimuli 97 to 176 (80)	O101 to R3
	BLOCK3	Stimuli 177 to 256 (80)	O182 to R4
	BLOCK4	Stimuli 257 to 336 (80)	O20 to R2
	BlockTOT	Total of activity during all task (experiment) measures	
	BlockMEAN	Mean of activity during all task (experiment) measures	

Appendix M Optode Positioning (fNIRS)

Unlike fMRI where you can scan the whole brain in 3d to model exactly where different brain structures lie in relation to one another, fNIRS has to rely on measurements derived from external landmarks on the head surface (see Chapter 6 for measurement procedure). These are measurements and external landmarks are matched as far as possible with average locations of internal brain areas. The location of key brain areas are summarised in various brain atlas' (derived from average measurements of participant brain structures) such as Brodmann's areas (Brodmann, 1908), the Talairach atlas (Talairach and Tournoux, 1988) Talairach daemon (Lancaster et al., 2000) and Montreal Neurological Institute (MNI) atlas.

By its nature, the process of spatial registration has scope for imprecision (head anatomy is individual), but several studies have assured that the methods based on head surface landmarks do reliably measure the required regions of interest on the cortex. Tsuzuki and Dan (2014) provide an overview of various ways to 'spatially register' the brain; matching the location of electrodes on the scalp to the desired locations to measure cortical activity spatially. Digitizers can be used to map the surface of the brain in 3D by registering set reference points on the surface. The advantage is a more personalised spatial understanding of where the optodes will lie on the head, however there are drawbacks that make digitizers less reliable. These include measurement bias, failed registration errors and interference by external equipment.

The International 10-20 system is the most common method of placing electrodes on the scalp for EEG and is frequently used to place optodes for EEG per

(Jasper, 1958) as well a fNIRS. This system uses visible landmarks on the head using simple measurements to find Cz at 0.0 (at the top and centre of the head) which can then be used to find key neurological structures relative to this point. The International 10:20 system has been found to agree with Brodmann's mapping system (Homan et al., 1987). Although few techniques can provide the level of accuracy attained by fMRI, the International 10:20 system has been found to be affordable and reliable enough to isolate larger cortical areas such as the Dorsolateral Prefrontal Cortex (F3) e.g. in TMS positioning (Herwig et al., 2003).

Regions of interested related to the DMN and the frontal cortex (attention control and inhibition) were researched. Potential areas were cross referenced with the fOLD (fNIRS Optodes Location Decider v2.2) brain ATLAS as this is specifically designed with fNIRS coverage (Zimeo Marais, Balardin & Sato 2017) which has limited depth. The fOLD atlas uses a 10:10 system so this does not map on exactly to the 10:20 locations but gives an idea of which brain areas are within a realistic depth for fNIRS. The most promising regions are listed below and Table XX shows the names/ locations of the relevant regions per popular brain atlases adapted from (Okamoto et al., 2004).

Attention/ Inhibition:

Dorsolateral Prefrontal Cortex, Brodmann's Areas 9 and 46 (particularly the right) (Dores et al., 2017; Garavan et al., 2002; Peterson et al., 2002); (Fox et al., 2005)

Inferior frontal lobe, Brodmann's Area 45 (Peterson et al., 2002)

Right superior frontal gyrus Brodmann's Area 45 (Lavagnino, Mwangi, et al., 2016)

Default Mode Network:

Medial Prefrontal cortex, Brodmann's area 46 (Durantin et al., 2015; Spreng et al., 2010),

Lateral Temporal Cortex, Brodmann's area 21 (Spreng et al., 2010)

Table L1 Names and Locations of the Regions of Interest per Popular Brain Atlases and percentage coverage adapted from Okamoto et al. (2004)

International 10:20	**Anatomy Talairach Daemon**	% Coverage	**Brodmann's Area**	% Coverage	**Anatomy Manual Labelling**	% Coverage	**Brodmann's Area**	% Coverage
Frontal Lobe (FL) Areas Associated with Attention and Inhibition Function								
	LEFT FL							
F3	Superior frontal G	56	10	47	Middle frontal G	81	**9**	63
	Middle frontal G	44	**9**	43	Superior frontal G	19	10	31
F7	Inferior frontal G	84	47	81	Inferior frontal G	88	47	63
	Middle frontal G	16	**45**	13	Middle frontal G	13	**45**	19
	RIGHT FL							
F4	Middle frontal G	60	10	49	Middle frontal G	98	**9**	52
	Superior frontal G	40	**9**	34	Superior frontal G	2	**46**	25
F8	Inferior frontal G	94	47	94	Inferior frontal G	100	47	60
	Middle frontal G	6	**45**	6	Middle frontal G		**45**	29
Temporal Lobe Areas Associated with the Default Mode Network								
	LEFT TL							
T3	L TL Middle temporal G	94	**21**	94	L TL Middle temporal G	88	**21**	88
	L TL Superior temporal G	6	22	6	L TL Superior temporal G	12	22	12
	RIGH TL							
T4	R TL Middle temporal G	96	**21**	95	R TL Middle temporal G	85	**21**	85
	R TL Superior temporal G	4	22	5	R TL Superior temporal G	15	22	15

Note: G=Gyrus

The channel names and locations are recorded below. At each channel measures were taken of the change in the concentration of oxygenated and deoxygenated haemoglobin over time (these two signals added together give the total change in oxygenation over time).

Table L2

Receiver to Transmitter pairs for each measurement channels and their nearest 10:20 regions.

Right Hemisphere			**Left Hemisphere**		
Channel names	**Receiver-Transmitter pairs**	Nearest 10:20	**Channel names**	**Receiver-Transmitter pairs**	Nearest 10:20
RF1	Rx3a - Tx3	F8	**LF1**	Rx3b - Tx7	F7
RF2	Rx1a - Tx3		**LF2**	Rx3b - Tx6	
RF3	Rx1a - Tx2		**LF3**	Rx1b - Tx6	
RF4	Rx4a - Tx2		**LF4**	Rx1b - Tx5	
RF5	Rx4a - Tx4	F4	**LF5**	Rx2b - Tx5	F3
RT1	Rx1a - Tx1	T4	**LT1**	Rx4b - Tx6	T3
RT2	Rx2a - Tx1	T4	**LT2**	Rx4b - Tx8	T3
RT3	Rx2b - Tx3	T4	**LT3**	Rx3b - Tx8	T3

Note: RF=Right frontal; RT=Right Temporal; LF=Left Frontal; LT=Left Temporal; *xa*/*xb* indicates the same receiver is used at more than one channel.

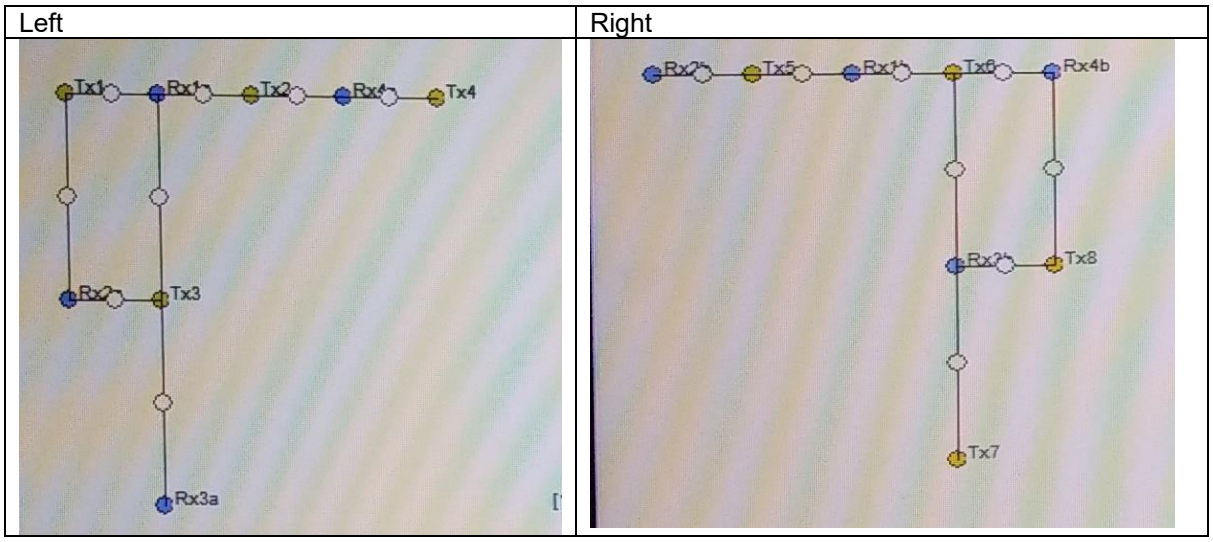


Figure L1

Artinis visualisation showing the receiver (blue) and transmitters (Yellow) and channels

Appendix N Study 2 Scale Reliability (Chronbach's Alpha)

Case Processing Summary

		N	%
Cases	Valid	92	100.0
	Excluded[a]	0	.0
	Total	92	100.0

a. Listwise deletion based on all variables in the procedure.

N1 Scale: Ravens Matrices

Reliability Statistics

Cronbach's Alpha	N of Items
.765	12

N2 Scale: GAD-7

Reliability Statistics

Cronbach's Alpha	N of Items
.897	7

N3 Scale: PHQ-8

Reliability Statistics

Cronbach's Alpha	N of Items
.894	8

N4 Scale: RSS

Reliability Statistics

Cronbach's Alpha	N of Items
.794	10

N5 Scale: RSS - Brooding

Reliability Statistics

Cronbach's Alpha	N of Items
.724	5

N6 Scale: RSS - Reflection

Reliability Statistics

Cronbach's Alpha	N of Items
.756	5

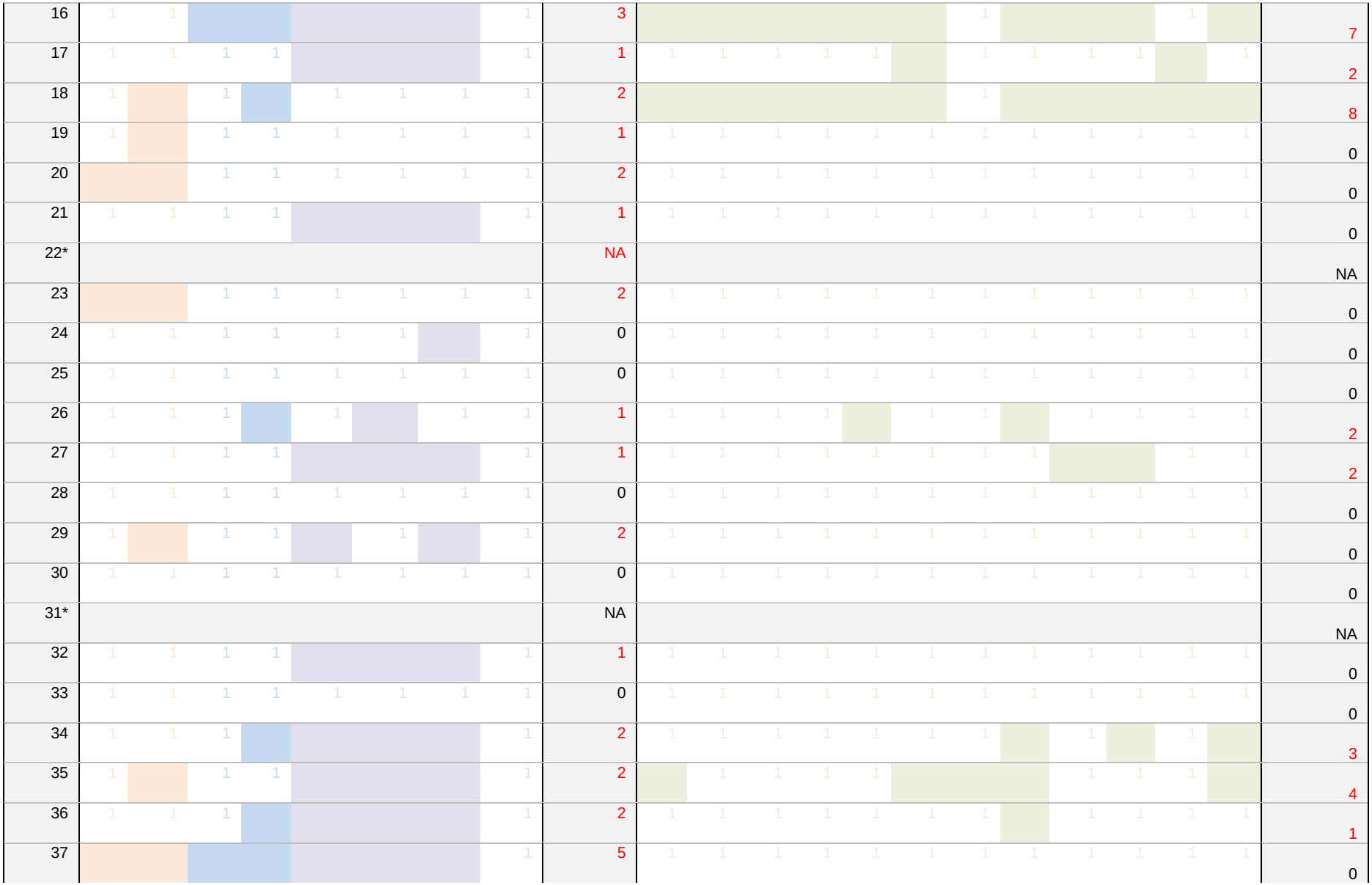
N7 Scale: BRIEF - All Items

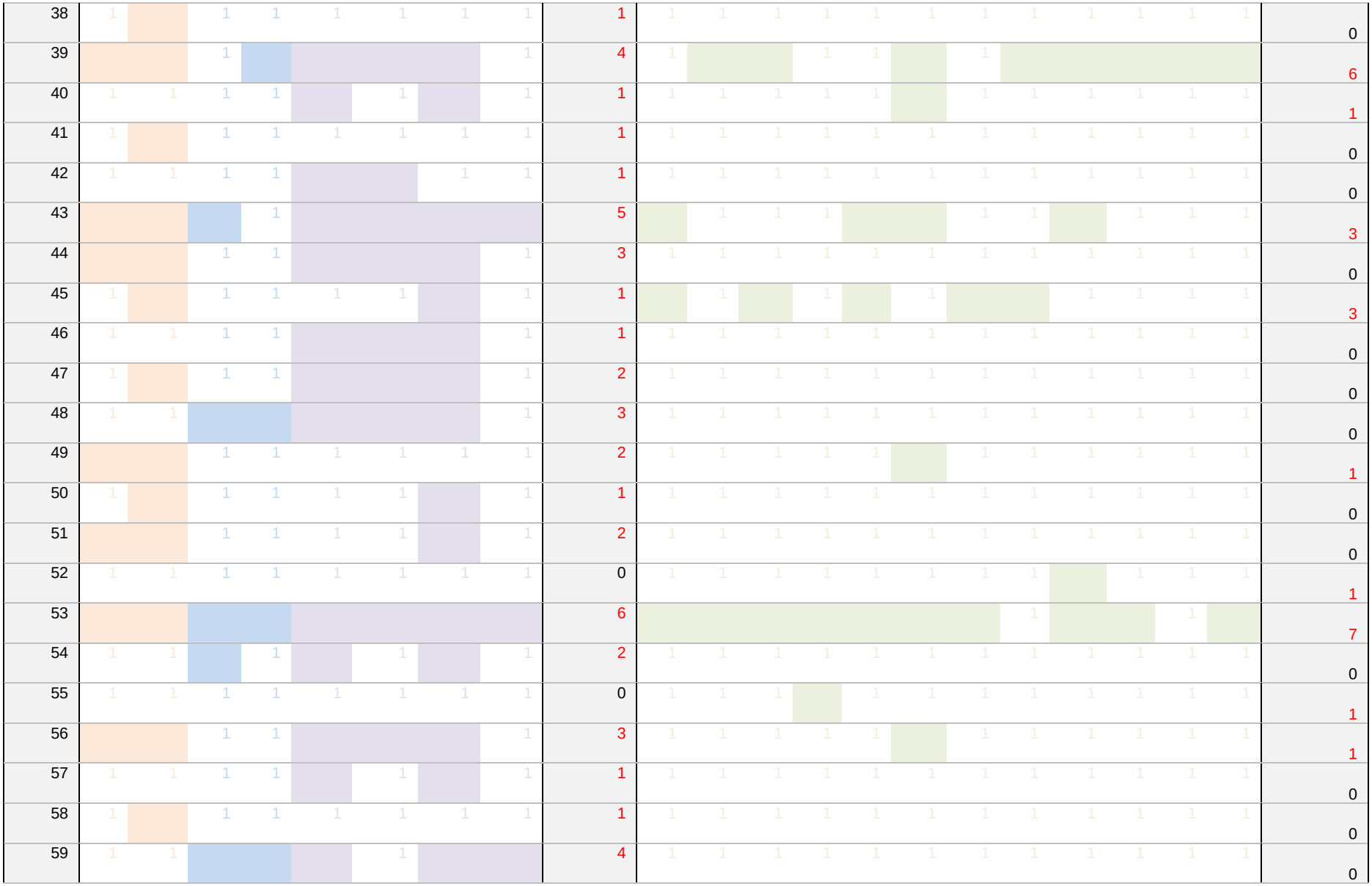
Reliability Statistics

Cronbach's Alpha	N of Items
.960	75

Appendix O Participant Comorbidities Profile

PPN (N=93)	BMI	Waist	ANX	Dep	RUMt	RUMb	RUMr	Worry	No. of health issues /6	BRI _SS	Mi _SS	GEC _SS	Inh	Shi	EmC	SeM	Ini	WkM	Pla	OrM	TKM	No. of EF problems /9
	Norm /OW	Low/ Norm	NClin	NClin	Low	Low	Low	Low		NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	NClin <65	
1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	0
2*									NA													NA
3	1	1	1	1	1	1	1	1	0	1	1	1	1		1	1	1	1		1	1	2
4	1						1	1	4		1	1	1				1	1	1	1	1	3
5	1	1	1	1				1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
6	1		1	1	1	1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
7	1	1	1	1				1	1	1				1	1		1		1		1	4
8									6		1	1	1	1		1	1	1	1	1	1	1
9	1		1	1				1	2	1	1	1	1	1		1	1	1	1	1	1	1
10									6											1		8
11	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	0
12*			1			1		1	4	1	1	1	1	1	1		1	1	1	1		2
13			1	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	0
14			1	1	1	1	1	1	2	1	1	1	1	1	1	1	1		1	1		2
15	1							1	4	1			1	1	1	1	1		1		1	2

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51 2 0
52 0 1
53 6 7
54 2 0
55 0 1
56 3 1
57 1 0
58 1 0
59 4 0


60 5 3
61* 4 0
62 0 0
63 3 0
64* 3 0
65 0 0
66 0 0
67 0 0
68 1 0
69 3 4
70 3 2
71 0 1
72 0 0
73 3 1
74 2 0
75 3 8
76 2 0
77 4 5
78 1 0
79 2 0
80 1 0
81 4 9

82	1		1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
83	1	1	1	1	1	1	1		1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
84	1	1	1	1	1	1	1	1	0		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
85	1		1	1					2		1	1	1				1	1	1		1	1	1	1	3
86	1		1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
87	1		1					1	1		3	1	1	1	1		1	1	1		1	1	1	1	2
88			1	1	1	1		1	1		2	1	1	1	1	1	1	1	1	1	1	1	1	1	0
89	1		1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
90	1	1	1	1				1	1		1	1	1	1	1	1	1	1	1	1	1	1	1	1	0
91			1	1	1	1	1	1	1		2	1	1	1	1	1	1	1	1	1	1	1		1	1
92							1		6		1	1	1	1	1	1	1		1	1	1	1	1	1	1
93			1	1	1	1	1	1	2		1	1	1	1	1	1	1	1		1	1	1	1	1	1
Total Low (High)	65 (25)	41 (49)	70 (20)	68 (22)	46 (44)	50 (40)	46 (44)	78 (12)	2		78 (12)	80 (10)	79 (11)	79 (11)	74 (16)	72 (18)	81 (9)	75 (15)	70 (20)	79 (11)	82 (8)	79 (11)			1

Note: 1=Low group; Colour=High group; *=Incomplete or unusable data in some measures

Appendix P Study 2 RESULTS

P1 SPSS fNIRS During Resting State (Oxygenated Hb)

Regional Differences During Resting State

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Epsilon[b] Huynh-Feldt	Epsilon[b] Lower-bound
Region	.648	37.175	5	<.001	.809	.834	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Region	Sphericity Assumed	1559.435	3	519.812	4.209	.006
	Greenhouse-Geisser	1559.435	2.427	642.666	4.209	.011
	Huynh-Feldt	1559.435	2.501	623.481	4.209	.010
	Lower-bound	1559.435	1.000	1559.435	4.209	.043
Error(Region)	Sphericity Assumed	32235.140	261	123.506		
	Greenhouse-Geisser	32235.140	211.107	152.696		
	Huynh-Feldt	32235.140	217.602	148.138		
	Lower-bound	32235.140	87.000	370.519		

Estimates

Measure: OHb

Region	Mean	Std. Error	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
1	-4.735	1.287	-7.292	-2.178
2	-5.870	1.375	-8.602	-3.137
3	-8.651	1.659	-11.949	-5.354
4	-9.997	1.827	-13.627	-6.366

Pairwise Comparisons

Measure: OHb

(I) Region	(J) Region	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b] Lower Bound	95% Confidence Interval for Difference[b] Upper Bound
1	2	1.134	1.649	1.000	-3.319	5.588

	3	3.916*	1.277	.017	.468	7.364
	4	5.261	2.084	.080	-.364	10.887
2	1	-1.134	1.649	1.000	-5.588	3.319
	3	2.782	1.651	.574	-1.677	7.241
	4	4.127	1.669	.092	-.380	8.634
3 RT	1 RF	-3.916*	1.277	.017	-7.364	-.468
	2	-2.782	1.651	.574	-7.241	1.677
	4	1.345	1.623	1.000	-3.038	5.728
4	1	-5.261	2.084	.080	-10.887	.364
	2	-4.127	1.669	.092	-8.634	.380
	3	-1.345	1.623	1.000	-5.728	3.038

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

BMI Resting State

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
BMI_Binary_Cat	1.00	NormalRisk	65
	2.00	IncreasedRisk	23

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.648	36.808	5	<.001	.808

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	1710.555	3	570.185
	Greenhouse-Geisser	1710.555	2.424	705.781
	Huynh-Feldt	1710.555	2.528	676.647
	Lower-bound	1710.555	1.000	1710.555
Region * BMI_Binary_Cat	Sphericity Assumed	272.679	3	90.893
	Greenhouse-Geisser	272.679	2.424	112.508
	Huynh-Feldt	272.679	2.528	107.864
	Lower-bound	272.679	1.000	272.679
Error(Region)	Sphericity Assumed	31962.461	258	123.886
	Greenhouse-Geisser	31962.461	208.433	153.347
	Huynh-Feldt	31962.461	217.407	147.017
	Lower-bound	31962.461	86.000	371.657

Tests of Within-Subjects Effects

Measure: OxyHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	4.603	.004	.051
	Greenhouse-Geisser	4.603	.007	.051
	Huynh-Feldt	4.603	.006	.051
	Lower-bound	4.603	.035	.051
Region * BMI_Binary_Cat	Sphericity Assumed	.734	.533	.008
	Greenhouse-Geisser	.734	.506	.008
	Huynh-Feldt	.734	.511	.008
	Lower-bound	.734	.394	.008
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

Tests of Between-Subjects Effects

Measure: OxyHb

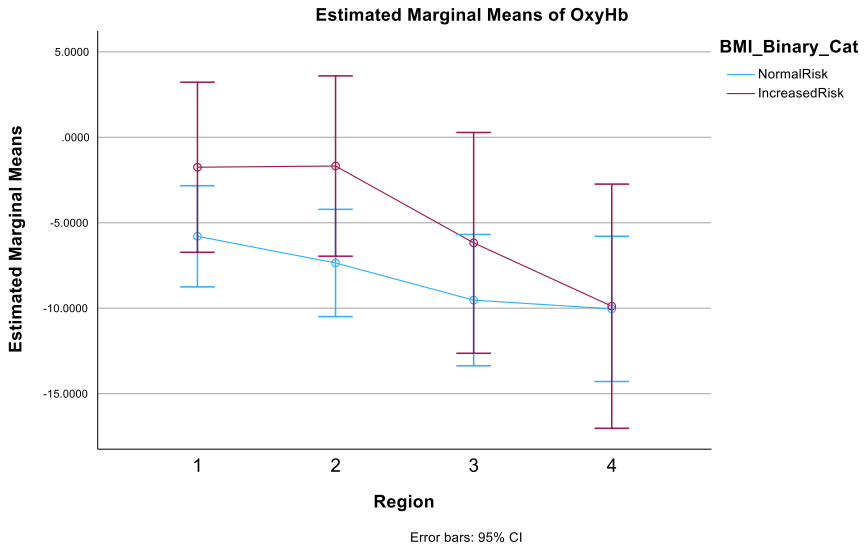
Transformed Variable: Average

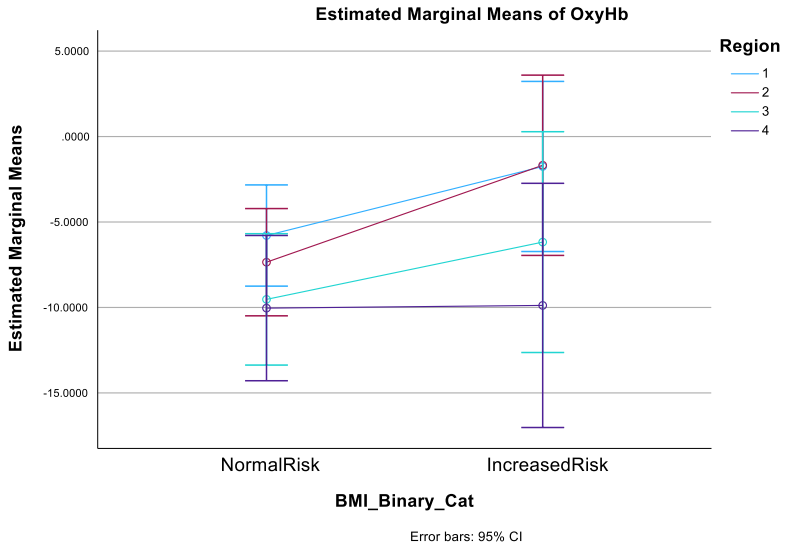
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	11571.005	1	11571.005	24.399	<.001	.221
BMI_Binary_Cat	742.291	1	742.291	1.565	.214	.018
Error	40784.995	86	474.244			

Measure: OxyHb

BMI_Binary_Cat	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
NormalRisk	-8.177	1.351	-10.862	-5.492
IncreasedRisk	-4.872	2.270	-9.385	-.359

Profile Plots





Resting State WAIST BINARY

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
Waist_Cat_MF	1.00	Low	41
	2.00	Increased	47

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.648	36.704	5	<.001	.808

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	1503.693	3	501.231
	Greenhouse-Geisser	1503.693	2.424	620.310
	Huynh-Feldt	1503.693	2.528	594.700
	Lower-bound	1503.693	1.000	1503.693
Region * Waist_Cat_MF	Sphericity Assumed	119.375	3	39.792
	Greenhouse-Geisser	119.375	2.424	49.245
	Huynh-Feldt	119.375	2.528	47.212
	Lower-bound	119.375	1.000	119.375
Error(Region)	Sphericity Assumed	32115.764	258	124.480
	Greenhouse-Geisser	32115.764	208.473	154.053
	Huynh-Feldt	32115.764	217.450	147.693
	Lower-bound	32115.764	86.000	373.439

Tests of Within-Subjects Effects

Measure: OxyHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	4.027	.008	.045
	Greenhouse-Geisser	4.027	.013	.045
	Huynh-Feldt	4.027	.012	.045
	Lower-bound	4.027	.048	.045
Region * Waist_Cat_MF	Sphericity Assumed	.320	.811	.004
	Greenhouse-Geisser	.320	.768	.004
	Huynh-Feldt	.320	.777	.004
	Lower-bound	.320	.573	.004
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			

Lower-bound				

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	19292.760	1	19292.760	40.808	<.001	.322
Waist_Cat_MF	869.114	1	869.114	1.838	.179	.021
Error	40658.173	86	472.769			

Depression Resting State

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
Dep_Clin2	1.00	LowNonClin	68
	2.00	Clinical	20

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.648	36.771	5	<.001	.809

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square	F
Region	Sphericity Assumed	1284.378	3	428.126	3.431
	Greenhouse-Geisser	1284.378	2.426	529.524	3.431
	Huynh-Feldt	1284.378	2.530	507.651	3.431
	Lower-bound	1284.378	1.000	1284.378	3.431
Region * DEP_Clin	Sphericity Assumed	41.845	3	13.948	.112
	Greenhouse-Geisser	41.845	2.426	17.252	.112
	Huynh-Feldt	41.845	2.530	16.539	.112
	Lower-bound	41.845	1.000	41.845	.112
Error(Region)	Sphericity Assumed	32193.295	258	124.780	
	Greenhouse-Geisser	32193.295	208.596	154.333	
	Huynh-Feldt	32193.295	217.584	147.958	
	Lower-bound	32193.295	86.000	374.341	

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Sig.	Partial Eta Squared
Region	Sphericity Assumed	.018	.038
	Greenhouse-Geisser	.026	.038
	Huynh-Feldt	.024	.038
	Lower-bound	.067	.038
Region * DEP_Clin	Sphericity Assumed	.953	.001
	Greenhouse-Geisser	.926	.001
	Huynh-Feldt	.932	.001
	Lower-bound	.739	.001
Error(Region)	Sphericity Assumed		

	Greenhouse-Geisser		
	Huynh-Feldt		
	Lower-bound		

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	16403.184	1	16403.184	34.446	<.001	.286
DEP_Clin	574.659	1	574.659	1.207	.275	.014
Error	40952.627	86	476.193			

Resting State ANXIETY

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
ANX_Clin2	1.00	LowNonClin	69
	2.00	Clinical	19

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.648	36.794	5	<.001	.808

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square	F
Region	Sphericity Assumed	1409.497	3	469.832	3.773
	Greenhouse-Geisser	1409.497	2.423	581.641	3.773
	Huynh-Feldt	1409.497	2.528	557.635	3.773
	Lower-bound	1409.497	1.000	1409.497	3.773
Region * ANX_Clin	Sphericity Assumed	109.755	3	36.585	.294
	Greenhouse-Geisser	109.755	2.423	45.291	.294
	Huynh-Feldt	109.755	2.528	43.422	.294
	Lower-bound	109.755	1.000	109.755	.294
Error(Region)	Sphericity Assumed	32125.385	258	124.517	

	Greenhouse-Geisser	32125.385	208.405	154.149	
	Huynh-Feldt	32125.385	217.377	147.787	
	Lower-bound	32125.385	86.000	373.551	

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Sig.	Partial Eta Squared
Region	Sphericity Assumed	.011	.042
	Greenhouse-Geisser	.018	.042
	Huynh-Feldt	.016	.042
	Lower-bound	.055	.042
Region * ANX_Clin	Sphericity Assumed	.830	.003
	Greenhouse-Geisser	.787	.003
	Huynh-Feldt	.796	.003
	Lower-bound	.589	.003
Error(Region)	Sphericity Assumed		
	Greenhouse-Geisser		
	Huynh-Feldt		
	Lower-bound		

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	16351.903	1	16351.903	34.439	<.001	.286
ANX_Clin	693.803	1	693.803	1.461	.230	.017
Error	40833.484	86	474.808			

Resting State TOTAL RUMINATION

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		N
RUMt_Binary_Cat	1.00	45
	2.00	43

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.634	38.635	5	<.001	.806

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	1604.140	3	534.713
	Greenhouse-Geisser	1604.140	2.418	663.330
	Huynh-Feldt	1604.140	2.522	636.003
	Lower-bound	1604.140	1.000	1604.140
Region * RUMt_Binary_Cat	Sphericity Assumed	831.444	3	277.148
	Greenhouse-Geisser	831.444	2.418	343.811
	Huynh-Feldt	831.444	2.522	329.647
	Lower-bound	831.444	1.000	831.444
Error(Region)	Sphericity Assumed	31403.696	258	121.720
	Greenhouse-Geisser	31403.696	207.975	150.997
	Huynh-Feldt	31403.696	216.911	144.777
	Lower-bound	31403.696	86.000	365.159

Tests of Within-Subjects Effects

Measure: OxyHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	4.393	.005	.049
	Greenhouse-Geisser	4.393	.009	.049
	Huynh-Feldt	4.393	.008	.049
	Lower-bound	4.393	.039	.049
Region * RUMt_Binary_Cat	Sphericity Assumed	2.277	.080	.026
	Greenhouse-Geisser	2.277	.094	.026
	Huynh-Feldt	2.277	.092	.026
	Lower-bound	2.277	.135	.026
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	19099.639	1	19099.639	41.609	<.001
RUMt_Binary_Cat	2050.957	1	2050.957	4.468	.037
Error	39476.330	86	459.027		

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Partial Eta Squared
Intercept	.326
RUMt_Binary_Cat	.049
Error	

Resting State BROODING RUMINATION

with Bonferroni correction

Within-Subjects Factors

Measure: OHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
RUMb_Binary_Cat	1.00	Low	49
	2.00	High	39

Mauchly's Test of Sphericity[a]

Measure: OHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	.631	38.949	5	<.001	.802	.837	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + RUMb_Binary_Cat

Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: OHb

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Region	Sphericity Assumed	1794.495	3	598.165	4.915	.002
	Greenhouse-Geisser	1794.495	2.407	745.384	4.915	.005
	Huynh-Feldt	1794.495	2.510	714.800	4.915	.004

	Lower-bound	1794.495	1.000	1794.495	4.915	.029
Region * RUMb_Binary_Cat	Sphericity Assumed	836.957	3	278.986	2.292	.079
	Greenhouse-Geisser	836.957	2.407	347.649	2.292	.093
	Huynh-Feldt	836.957	2.510	333.384	2.292	.090
	Lower-bound	836.957	1.000	836.957	2.292	.134
Error(Region)	Sphericity Assumed	31398.182	258	121.698		
	Greenhouse-Geisser	31398.182	207.043	151.651		
	Huynh-Feldt	31398.182	215.902	145.428		
	Lower-bound	31398.182	86.000	365.095		

Tests of Between-Subjects Effects

Measure: OHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	20604.746	1	20604.746	47.272	<.001
RUMb_Binary_Cat	4042.064	1	4042.064	9.273	.003
Error	37485.222	86	435.875		

Estimated Marginal Means

1. Region

Estimates

Measure: OHb

Region	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-4.865	1.297	-7.442	-2.287
2	-6.190	1.358	-8.890	-3.489
3	-9.210	1.594	-12.378	-6.042
4	-10.539	1.776	-14.069	-7.008

Pairwise Comparisons

Measure: OHb

(I) Region	(J) Region	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	1.325	1.660	1.000	-3.158	5.808
	3	4.346*	1.227	.004	1.032	7.659

	4	5.674*	2.073	.045	.076	11.271
2	1	-1.325	1.660	1.000	-5.808	3.158
	3	3.021	1.656	.430	-1.453	7.494
	4	4.349	1.677	.067	-.179	8.877
3	1	-4.346*	1.227	.004	-7.659	-1.032
	2	-3.021	1.656	.430	-7.494	1.453
	4	1.328	1.643	1.000	-3.110	5.766
4	1	-5.674*	2.073	.045	-11.271	-.076
	2	-4.349	1.677	.067	-8.877	.179
	3	-1.328	1.643	1.000	-5.766	3.110

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

2. RUMb_Binary_Cat

Estimates

Measure: OHb

			95% Confidence Interval	
RUMb_Binary_Cat	Mean	Std. Error	Lower Bound	Upper Bound
Low	-4.290	1.491	-7.255	-1.325
High	-11.112	1.672	-14.434	-7.789

Pairwise Comparisons

Measure: OHb

					95% Confidence Interval for Difference[b]	
(I) RUMb_Binary_Cat	(J) RUMb_Binary_Cat	Mean Difference (I-J)	Std. Error	Sig.[b]	Lower Bound	Upper Bound
Low	High	6.822*	2.240	.003	2.368	11.275
High	Low	-6.822*	2.240	.003	-11.275	-2.368

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

3. RUMb_Binary_Cat * Region

Estimates

Measure: OHb

				95% Confidence Interval	
RUMb_Binary_Cat	Region	Mean	Std. Error	Lower Bound	Upper Bound
Low	1	-3.726	1.726	-7.158	-.294

	2	-3.373	1.809	-6.969	.222
	3	-4.292	2.122	-8.510	-.074
	4	-5.769	2.365	-10.470	-1.068
High	1	-6.003	1.935	-9.850	-2.156
	2	-9.006	2.027	-13.036	-4.976
	3	-14.129	2.378	-18.857	-9.400
	4	-15.308	2.651	-20.578	-10.039

Pairwise Comparisons

Measure: OHb

	(I)	(J)	Mean	Std.		95% Confidence Interval for Difference[b]	
Region	RUMb_Binary_Cat	RUMb_Binary_Cat	Difference (I-J)	Error	Sig.[b]	Lower Bound	Upper Bound
1	Low	High	2.277	2.593	.382	-2.878	7.433
	High	Low	-2.277	2.593	.382	-7.433	2.878
2	Low	High	5.633*	2.717	.041	.232	11.033
	High	Low	-5.633*	2.717	.041	-11.033	-.232
3	Low	High	9.837*	3.187	.003	3.500	16.173
	High	Low	-9.837*	3.187	.003	-16.173	-3.500
4	Low	High	9.539*	3.552	.009	2.478	16.601
	High	Low	-9.539*	3.552	.009	-16.601	-2.478

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

4. RUMb_Binary_Cat * Region

Estimates

Measure: OHb

				95% Confidence Interval	
RUMb_Binary_Cat	Region	Mean	Std. Error	Lower Bound	Upper Bound
Low	1	-3.726	1.726	-7.158	-.294
	2	-3.373	1.809	-6.969	.222
	3	-4.292	2.122	-8.510	-.074
	4	-5.769	2.365	-10.470	-1.068
High	1	-6.003	1.935	-9.850	-2.156
	2	-9.006	2.027	-13.036	-4.976
	3	-14.129	2.378	-18.857	-9.400
	4	-15.308	2.651	-20.578	-10.039

Pairwise Comparisons

Measure: OHb

RUMb_Binary_Cat	(I) Region	(J) Region	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b] Lower Bound	Upper Bound
Low	1	2	-.353	2.210	1.000	-6.322	5.616
		3	.566	1.634	1.000	-3.846	4.978
		4	2.043	2.760	1.000	-5.410	9.495
	2	1	.353	2.210	1.000	-5.616	6.322
		3	.919	2.205	1.000	-5.037	6.875
		4	2.396	2.232	1.000	-3.633	8.425
	3	1	-.566	1.634	1.000	-4.978	3.846
		2	-.919	2.205	1.000	-6.875	5.037
		4	1.477	2.188	1.000	-4.432	7.386
	4	1	-2.043	2.760	1.000	-9.495	5.410
		2	-2.396	2.232	1.000	-8.425	3.633
		3	-1.477	2.188	1.000	-7.386	4.432
High	1	2	3.003	2.477	1.000	-3.688	9.693
		3	8.125*	1.831	<.001	3.180	13.071
		4	9.305*	3.093	.021	.951	17.659
	2	1	-3.003	2.477	1.000	-9.693	3.688
		3	5.123	2.472	.247	-1.554	11.799
		4	6.302	2.502	.082	-.455	13.060
	3	1	-8.125*	1.831	<.001	-13.071	-3.180
		2	-5.123	2.472	.247	-11.799	1.554
		4	1.180	2.452	1.000	-5.443	7.803
	4	1	-9.305*	3.093	.021	-17.659	-.951
		2	-6.302	2.502	.082	-13.060	.455
		3	-1.180	2.452	1.000	-7.803	5.443

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

Resting State BROODING RUMINATION AND GENDER COVARIATE

Within-Subjects Factors

Measure: OHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		Value Label	N
RUMb_Binary_Cat	1.00	Low	49
	2.00	High	39

Mauchly's Test of Sphericity[a]

Measure: OHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.631	38.523	5	<.001	.801

Tests of Within-Subjects Effects

Measure: OHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	605.474	3	201.825
	Greenhouse-Geisser	605.474	2.404	251.883
	Huynh-Feldt	605.474	2.537	238.672
	Lower-bound	605.474	1.000	605.474
Region * Gender	Sphericity Assumed	1470.548	3	490.183
	Greenhouse-Geisser	1470.548	2.404	611.762
	Huynh-Feldt	1470.548	2.537	579.676
	Lower-bound	1470.548	1.000	1470.548
Region * RUMb_Binary_Cat	Sphericity Assumed	774.880	3	258.293
	Greenhouse-Geisser	774.880	2.404	322.358
	Huynh-Feldt	774.880	2.537	305.451
	Lower-bound	774.880	1.000	774.880
Error(Region)	Sphericity Assumed	29927.635	255	117.363
	Greenhouse-Geisser	29927.635	204.322	146.473
	Huynh-Feldt	29927.635	215.632	138.791
	Lower-bound	29927.635	85.000	352.090

Tests of Within-Subjects Effects

Measure: OHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	1.720	.163	.020
	Greenhouse-Geisser	1.720	.175	.020
	Huynh-Feldt	1.720	.172	.020
	Lower-bound	1.720	.193	.020
Region * Gender	Sphericity Assumed	4.177	.007	.047
	Greenhouse-Geisser	4.177	.012	.047
	Huynh-Feldt	4.177	.010	.047
	Lower-bound	4.177	.044	.047
Region * RUMb_Binary_Cat	Sphericity Assumed	2.201	.088	.025
	Greenhouse-Geisser	2.201	.103	.025
	Huynh-Feldt	2.201	.100	.025
	Lower-bound	2.201	.142	.025
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

Tests of Between-Subjects Effects

Measure: OHb
Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	30.453	1	30.453	.074	.787
Gender	2375.786	1	2375.786	5.752	.019
RUMb_Binary_Cat	3857.658	1	3857.658	9.339	.003
Error	35109.436	85	413.052		

Tests of Between-Subjects Effects

Measure: OHb

Transformed Variable: Average

Source	Partial Eta Squared
Intercept	.001
Gender	.063
RUMb_Binary_Cat	.099
Error	

Resting State Reflective Rumination

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		N
RUMr_Binary_Cat	1.00	45
	2.00	43

Descriptive Statistics

	RUMr_Binary_Cat	Mean	Std. Deviation	N
RF_ORS_Av	1.00	-4.611201	12.9761396	45
	2.00	-4.865147	11.1934783	43
	Total	-4.735288	12.0690032	88
LF_ORS_Av	1.00	-3.914012	9.0351133	45
	2.00	-7.916064	15.8332517	43
	Total	-5.869560	12.8979644	88
RT_ORS_Av	1.00	-6.269566	12.5148858	45
	2.00	-11.143889	18.0348922	43
	Total	-8.651338	15.5639655	88
LT_ORS_Av	1.00	-6.744744	11.6120663	45
	2.00	-13.399660	21.0641971	43
	Total	-9.996578	17.1344282	88

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.650	36.501	5	<.001	.812

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	1594.756	3	531.585
	Greenhouse-Geisser	1594.756	2.435	655.043
	Huynh-Feldt	1594.756	2.540	627.895
	Lower-bound	1594.756	1.000	1594.756
Region * RUMr_Binary_Cat	Sphericity Assumed	480.109	3	160.036
	Greenhouse-Geisser	480.109	2.435	197.204
	Huynh-Feldt	480.109	2.540	189.031
	Lower-bound	480.109	1.000	480.109
Error(Region)	Sphericity Assumed	31755.031	258	123.082
	Greenhouse-Geisser	31755.031	209.374	151.667
	Huynh-Feldt	31755.031	218.427	145.381
	Lower-bound	31755.031	86.000	369.245

Tests of Within-Subjects Effects

Measure: OxyHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	4.319	.005	.048
	Greenhouse-Geisser	4.319	.010	.048
	Huynh-Feldt	4.319	.009	.048
	Lower-bound	4.319	.041	.048
Region * RUMr_Binary_Cat	Sphericity Assumed	1.300	.275	.015
	Greenhouse-Geisser	1.300	.276	.015
	Huynh-Feldt	1.300	.276	.015
	Lower-bound	1.300	.257	.015
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	19047.677	1	19047.677	40.792	<.001
RUMr_Binary_Cat	1369.747	1	1369.747	2.933	.090
Error	40157.539	86	466.948		

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Partial Eta Squared
Intercept	.322
RUMr_Binary_Cat	.033
Error	

Resting State WORRY

Within-Subjects Factors

Measure: OxyHb

Region	Dependent Variable
1	RF_ORS_Av
2	LF_ORS_Av
3	RT_ORS_Av
4	LT_ORS_Av

Between-Subjects Factors

		N
Worry_Binary_Cat	1.00	76
	2.00	12

Descriptive Statistics

Worry_Binary_Cat	Mean	Std. Deviation	N

RF_ORS_Av	1.00	-4.626242	12.4068962	76
	2.00	-5.425913	10.0951613	12
	Total	-4.735288	12.0690032	88
LF_ORS_Av	1.00	-6.093619	12.9230456	76
	2.00	-4.450517	13.2106657	12
	Total	-5.869560	12.8979644	88
RT_ORS_Av	1.00	-7.656411	14.7886623	76
	2.00	-14.952536	19.3533857	12
	Total	-8.651338	15.5639655	88
LT_ORS_Av	1.00	-9.689588	17.1547985	76
	2.00	-11.940847	17.6278524	12
	Total	-9.996578	17.1344282	88

Mauchly's Test of Sphericity[a]

Measure: OxyHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser
Region	.637	38.172	5	<.001	.801

Mauchly's Test of Sphericity[a]

Measure: OxyHb

	Epsilon	
Within Subjects Effect	Huynh-Feldt	Lower-bound
Region	.836	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.[a]

a. Design: Intercept + Worry_Binary_Cat
Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: OxyHb

Source		Type III Sum of Squares	df	Mean Square
Region	Sphericity Assumed	1454.523	3	484.841
	Greenhouse-Geisser	1454.523	2.404	604.924
	Huynh-Feldt	1454.523	2.507	580.130
	Lower-bound	1454.523	1.000	1454.523
Region * Worry_Binary_Cat	Sphericity Assumed	442.539	3	147.513
	Greenhouse-Geisser	442.539	2.404	184.048
	Huynh-Feldt	442.539	2.507	176.505
	Lower-bound	442.539	1.000	442.539
Error(Region)	Sphericity Assumed	31792.600	258	123.227
	Greenhouse-Geisser	31792.600	206.785	153.747
	Huynh-Feldt	31792.600	215.622	147.446
	Lower-bound	31792.600	86.000	369.681

Tests of Within-Subjects Effects

Measure: OxyHb

Source		F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	3.935	.009	.044
	Greenhouse-Geisser	3.935	.015	.044
	Huynh-Feldt	3.935	.014	.044
	Lower-bound	3.935	.050	.044
Region * Worry_Binary_Cat	Sphericity Assumed	1.197	.311	.014
	Greenhouse-Geisser	1.197	.309	.014
	Huynh-Feldt	1.197	.309	.014
	Lower-bound	1.197	.277	.014
Error(Region)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	10891.313	1	10891.313	22.662	<.001
Worry_Binary_Cat	196.284	1	196.284	.408	.524
Error	41331.002	86	480.593		

Tests of Between-Subjects Effects

Measure: OxyHb

Transformed Variable: Average

Source	Partial Eta Squared
Intercept	.209
Worry_Binary_Cat	.005
Error	

P2 SPSS fNIRS During CT Task (attention) ANOVA

CT/ Regional Differences in OHb During the CT Task (attention)

Within-Subjects Factors

Measure: OHb

Region	Dependent Variable
1	RF_OC_L2Avg B1_2
2	LF_OC_L2Avg B1_2
3	RT_OC_L2Avg B1_2
4	LT_OC_L2Avg B1_2

Descriptive Statistics

	Mean	Std. Deviation	N
RF_OC_L2AvgB1_2	-1.3113	4.46547	87
LF_OC_L2AvgB1_2	-.4956	3.75355	87
RT_OC_L2AvgB1_2	-1.0372	3.11612	87
LT_OC_L2AvgB1_2	-.8774	3.63870	87

Mauchly's Test of Sphericity[a]

Measure: OHb

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	.645	37.192	5	<.001	.779	.802	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: OHb

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	30.307	3	10.102	1.186	.316	.014
	Greenhouse-Geisser	30.307	2.337	12.966	1.186	.312	.014
	Huynh-Feldt	30.307	2.407	12.593	1.186	.312	.014
	Lower-bound	30.307	1.000	30.307	1.186	.279	.014
Error(Region)	Sphericity Assumed	2198.304	258	8.521			
	Greenhouse-Geisser	2198.304	201.010	10.936			
	Huynh-Feldt	2198.304	206.973	10.621			
	Lower-bound	2198.304	86.000	25.562			

CT/ BMI risk and OHb During the CT Task (attention) – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
BMI_Binary_Cat	1.00	NormalRisk	65
	2.00	IncreasedRisk	22

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.645	36.745	5	0.000	0.778	0.810	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + BMI_Binary_Cat
Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	32.305	3	10.768	1.261	0.288
	Greenhouse-Geisser	32.305	2.333	13.847	1.261	0.288
	Huynh-Feldt	32.305	2.431	13.288	1.261	0.288
	Lower-bound	32.305	1.000	32.305	1.261	0.265
Region * BMI_Binary_Cat	Sphericity Assumed	20.537	3	6.846	0.802	0.494
	Greenhouse-Geisser	20.537	2.333	8.803	0.802	0.467
	Huynh-Feldt	20.537	2.431	8.448	0.802	0.471
	Lower-bound	20.537	1.000	20.537	0.802	0.373
Error(Region)	Sphericity Assumed	2177.767	255	8.540		
	Greenhouse-Geisser	2177.767	198.306	10.982		
	Huynh-Feldt	2177.767	206.644	10.539		
	Lower-bound	2177.767	85.000	25.621		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	212.597	1	212.597	6.691	0.011
BMI_Binary_Cat	1.053	1	1.053	0.033	0.856
Error	2700.919	85	31.776		

CT/ WAIST risk and OHb During CT Task (attention) Sig with Post hocs

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Waist_Cat_MF	1.00	Low	41
	2.00	Increased	46

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.638	37.682	5	0.000	0.777	0.810	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Waist_Cat_MF
Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	27.452	3	9.151	1.075	0.360
	Greenhouse-Geisser	27.452	2.331	11.775	1.075	0.351
	Huynh-Feldt	27.452	2.429	11.301	1.075	0.352
	Lower-bound	27.452	1.000	27.452	1.075	0.303
Region * Waist_Cat_MF	Sphericity Assumed	26.883	3	8.961	1.052	0.370
	Greenhouse-Geisser	26.883	2.331	11.531	1.052	0.359
	Huynh-Feldt	26.883	2.429	11.066	1.052	0.361
	Lower-bound	26.883	1.000	26.883	1.052	0.308
Error(Region)	Sphericity Assumed	2171.421	255	8.515		
	Greenhouse-Geisser	2171.421	198.162	10.958		
	Huynh-Feldt	2171.421	206.488	10.516		
	Lower-bound	2171.421	85.000	25.546		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	274.319	1	274.319	9.235	0.003
Waist_Cat_MF	177.100	1	177.100	5.962	0.017
Error	2524.872	85	29.704		

Estimated Marginal Means

1. Region

Estimates

Measure:

Region	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-1.000	0.329	-1.655	-0.346
2	-0.846	0.388	-1.618	-0.073
3	-1.243	0.465	-2.168	-0.319
4	-0.468	0.402	-1.268	0.331

Pairwise Comparisons

Measure:

(I) Region		Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-0.155	0.468	1.000	-1.419	1.109
	3	0.243	0.337	1.000	-0.668	1.154
	4	-0.532	0.414	1.000	-1.649	0.585
2	1	0.155	0.468	1.000	-1.109	1.419
	3	0.398	0.565	1.000	-1.129	1.925
	4	-0.377	0.430	1.000	-1.539	0.784
3	1	-0.243	0.337	1.000	-1.154	0.668
	2	-0.398	0.565	1.000	-1.925	1.129
	4	-0.775	0.413	0.383	-1.890	0.340
4	1	0.532	0.414	1.000	-0.585	1.649
	2	0.377	0.430	1.000	-0.784	1.539
	3	0.775	0.413	0.383	-0.340	1.890

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

2. Waist_Cat_MF * Region

Estimates

Measure:

Waist_Cat_MF		Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Low	1	-0.355	0.479	-1.307	0.597
	2	-0.291	0.565	-1.414	0.832
	3	-0.058	0.676	-1.402	1.286
	4	0.005	0.585	-1.158	1.168
Increased	1	-1.646	0.452	-2.544	-0.747
	2	-1.400	0.533	-2.460	-0.340
	3	-2.428	0.638	-3.697	-1.159
	4	-0.942	0.552	-2.040	0.156

Pairwise Comparisons

Measure:

Region			Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
						Lower Bound	Upper Bound
1	Low	Increased	1.291	0.658	0.053	-0.018	2.600
	Increased	Low	-1.291	0.658	0.053	-2.600	0.018
2	Low	Increased	1.109	0.777	0.157	-0.436	2.654
	Increased	Low	-1.109	0.777	0.157	-2.654	0.436
3	Low	Increased	2.370*	0.930	0.013	0.521	4.219
	Increased	Low	-2.370*	0.930	0.013	-4.219	-0.521

4	Low	Increased	0.947	0.804	0.243	-0.653	2.546
	Increased	Low	-0.947	0.804	0.243	-2.546	0.653

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

3. Waist_Cat_MF * Region

Estimates

Measure:

Waist_Cat_MF		Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Low	1	-0.355	0.479	-1.307	0.597
	2	-0.291	0.565	-1.414	0.832
	3	-0.058	0.676	-1.402	1.286
	4	0.005	0.585	-1.158	1.168
Increased	1	-1.646	0.452	-2.544	-0.747
	2	-1.400	0.533	-2.460	-0.340
	3	-2.428	0.638	-3.697	-1.159
	4	-0.942	0.552	-2.040	0.156

Pairwise Comparisons

Measure:

Waist_Cat_MF			Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
						Lower Bound	Upper Bound
Low	1	2	-0.064	0.681	1.000	-1.902	1.775
		3	-0.297	0.490	1.000	-1.621	1.028
		4	-0.360	0.601	1.000	-1.984	1.265
	2	1	0.064	0.681	1.000	-1.775	1.902

		3	-0.233	0.822	1.000	-2.454	1.988
		4	-0.296	0.625	1.000	-1.985	1.393
	3	1	0.297	0.490	1.000	-1.028	1.621
		2	0.233	0.822	1.000	-1.988	2.454
		4	-0.063	0.600	1.000	-1.684	1.558
	4	1	0.360	0.601	1.000	-1.265	1.984
		2	0.296	0.625	1.000	-1.393	1.985
		3	0.063	0.600	1.000	-1.558	1.684
Increased	1	2	-0.246	0.642	1.000	-1.981	1.490
		3	0.783	0.463	0.568	-0.468	2.034
		4	-0.704	0.568	1.000	-2.238	0.830
	2	1	0.246	0.642	1.000	-1.490	1.981
		3	1.028	0.776	1.000	-1.068	3.125
		4	-0.458	0.590	1.000	-2.053	1.136
	3	1	-0.783	0.463	0.568	-2.034	0.468
		2	-1.028	0.776	1.000	-3.125	1.068
		4	-1.487	0.567	0.062	-3.017	0.044
	4	1	0.704	0.568	1.000	-0.830	2.238
		2	0.458	0.590	1.000	-1.136	2.053
		3	1.487	0.567	0.062	-0.044	3.017

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

CT/ Anxiety and OHb During CT Task (attention) During CT Attention Task

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
ANX_Clin	1.00	LowNonClin	69
	2.00	Clinical	18

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.638	37.562	5	0.000	0.775	0.808	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + ANX_Clin
Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	12.681	3	4.227	0.493	0.687
	Greenhouse-Geisser	12.681	2.325	5.453	0.493	0.640
	Huynh-Feldt	12.681	2.423	5.234	0.493	0.647
	Lower-bound	12.681	1.000	12.681	0.493	0.484
Region * ANX_Clin	Sphericity Assumed	13.971	3	4.657	0.544	0.653
	Greenhouse-Geisser	13.971	2.325	6.008	0.544	0.608
	Huynh-Feldt	13.971	2.423	5.766	0.544	0.615
	Lower-bound	13.971	1.000	13.971	0.544	0.463
Error(Region)	Sphericity Assumed	2184.334	255	8.566		
	Greenhouse-Geisser	2184.334	197.653	11.051		
	Huynh-Feldt	2184.334	205.937	10.607		
	Lower-bound	2184.334	85.000	25.698		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	145.106	1	145.106	4.585	0.035
ANX_Clin	11.818	1	11.818	0.373	0.543
Error	2690.154	85	31.649		

CT/ Depression and OHb During CT Task (attention) Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Dep_Clin	1.00	LowNonClin	67
	2.00	Clinical	20

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.644	36.872	5	0.000	0.778	0.810	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + DEP_Clin Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	18.614	3	6.205	0.723	0.539
	Greenhouse-Geisser	18.614	2.333	7.980	0.723	0.507
	Huynh-Feldt	18.614	2.431	7.658	0.723	0.512
	Lower-bound	18.614	1.000	18.614	0.723	0.398
Region * DEP_Clin	Sphericity Assumed	9.471	3	3.157	0.368	0.776
	Greenhouse-Geisser	9.471	2.333	4.060	0.368	0.725
	Huynh-Feldt	9.471	2.431	3.897	0.368	0.734
	Lower-bound	9.471	1.000	9.471	0.368	0.546
Error(Region)	Sphericity Assumed	2188.834	255	8.584		
	Greenhouse-Geisser	2188.834	198.265	11.040		
	Huynh-Feldt	2188.834	206.599	10.595		
	Lower-bound	2188.834	85.000	25.751		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	167.144	1	167.144	5.277	0.024
DEP_Clin	9.637	1	9.637	0.304	0.583
Error	2692.335	85	31.675		

CT/Total Rumination and OHb During CT Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMt_Binary Low_Mod+	1.00	Low	44
	2.00	Mod/High	43

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.653	35.743	5	0.000	0.784	0.817	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + RUMt_Binary Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Region	Sphericity Assumed	30.811	3	10.270	1.205	0.308

	Greenhouse-Geisser	30.811	2.353	13.096	1.205	0.305
	Huynh-Feldt	30.811	2.452	12.564	1.205	0.306
	Lower-bound	30.811	1.000	30.811	1.205	0.275
Region * RUMt_Binary	Sphericity Assumed	25.738	3	8.579	1.007	0.390
	Greenhouse-Geisser	25.738	2.353	10.940	1.007	0.377
	Huynh-Feldt	25.738	2.452	10.495	1.007	0.379
	Lower-bound	25.738	1.000	25.738	1.007	0.318
Error(Region)	Sphericity Assumed	2172.567	255	8.520		
	Greenhouse-Geisser	2172.567	199.975	10.864		
	Huynh-Feldt	2172.567	208.450	10.422		
	Lower-bound	2172.567	85.000	25.560		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	298.616	1	298.616	9.542	0.003
RUMt_Binary	41.925	1	41.925	1.340	0.250
Error	2660.047	85	31.295		

CT/Brooding Rumination and OHb During CT Attention Task

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMb_Binary Low_Mod+	1.00	Low	49
	2.00	Mod/High	38

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.656	35.339	5	0.000	0.787	0.820	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + RUMb_Binary
Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Region	Sphericity Assumed	36.154	3	12.051	1.420	0.237

	Greenhouse-Geisser	36.154	2.360	15.321	1.420	0.243
	Huynh-Feldt	36.154	2.460	14.696	1.420	0.242
	Lower-bound	36.154	1.000	36.154	1.420	0.237
Region * RUMb_Binary	Sphericity Assumed	34.525	3	11.508	1.356	0.257
	Greenhouse-Geisser	34.525	2.360	14.631	1.356	0.260
	Huynh-Feldt	34.525	2.460	14.034	1.356	0.260
	Lower-bound	34.525	1.000	34.525	1.356	0.247
Error(Region)	Sphericity Assumed	2163.779	255	8.485		
	Greenhouse-Geisser	2163.779	200.581	10.788		
	Huynh-Feldt	2163.779	209.106	10.348		
	Lower-bound	2163.779	85.000	25.456		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	279.724	1	279.724	8.849	0.004
RUMb_Binary	15.133	1	15.133	0.479	0.491
Error	2686.839	85	31.610		

CT/ Reflective Rumination and OHb During CT Attention Task

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMr_Binary Low_Mod+	1.00	Low	44
	2.00	Mod/High	43

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.642	37.095	5	0.000	0.778	0.811	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + RUMr_Binary Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	30.524	3	10.175	1.189	0.314
	Greenhouse-Geisser	30.524	2.335	13.074	1.189	0.311
	Huynh-Feldt	30.524	2.433	12.546	1.189	0.312
	Lower-bound	30.524	1.000	30.524	1.189	0.279
Region * RUMr_Binary	Sphericity Assumed	15.899	3	5.300	0.619	0.603
	Greenhouse-Geisser	15.899	2.335	6.810	0.619	0.564
	Huynh-Feldt	15.899	2.433	6.535	0.619	0.570
	Lower-bound	15.899	1.000	15.899	0.619	0.434
Error(Region)	Sphericity Assumed	2182.405	255	8.558		
	Greenhouse-Geisser	2182.405	198.446	10.997		
	Huynh-Feldt	2182.405	206.795	10.553		
	Lower-bound	2182.405	85.000	25.675		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	299.811	1	299.811	9.474	0.003
RUMr_Binary	12.038	1	12.038	0.380	0.539
Error	2689.934	85	31.646		

CT/ Worry and OHb During CT Task (attention)- Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RT_OC_L2AvgB1_2
2	LT_OC_L2AvgB1_2
3	RF_OC_L2AvgB1_2
4	LF_OC_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Worry_Binary	1.00	Low/none	58
	2.00	Mod/High	29

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.643	36.927	5	0.000	0.777	0.810	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Worry_Binary
Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Region	Sphericity Assumed	23.613	3	7.871	0.918	0.433
	Greenhouse-Geisser	23.613	2.331	10.131	0.918	0.413
	Huynh-Feldt	23.613	2.429	9.723	0.918	0.417
	Lower-bound	23.613	1.000	23.613	0.918	0.341
Region * Worry_Binary	Sphericity Assumed	12.079	3	4.026	0.470	0.704
	Greenhouse-Geisser	12.079	2.331	5.183	0.470	0.655
	Huynh-Feldt	12.079	2.429	4.974	0.470	0.663
	Lower-bound	12.079	1.000	12.079	0.470	0.495
Error(Region)	Sphericity Assumed	2186.225	255	8.573		
	Greenhouse-Geisser	2186.225	198.114	11.035		
	Huynh-Feldt	2186.225	206.436	10.590		
	Lower-bound	2186.225	85.000	25.720		

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	256.689	1	256.689	8.078	0.006
Worry_Binary	1.052	1	1.052	0.033	0.856
Error	2700.920	85	31.776		

P3 SPSS fNIRS During ST Task (Inhibition) ANOVA SPSS

ST/Regional Differences and OHb During ST Task (Inhibition)

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Descriptive Statistics

	Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.869	12.068	5	0.034	0.911	0.943	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: Region

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	19.707	3	6.569	1.707	0.166	0.019

	Greenhouse-Geisser	19.707	2.732	7.213	1.707	0.171	0.019
	Huynh-Feldt	19.707	2.829	6.965	1.707	0.169	0.019
	Lower-bound	19.707	1.000	19.707	1.707	0.195	0.019
Error(Region)	Sphericity Assumed	1004.131	261	3.847			
	Greenhouse-Geisser	1004.131	237.696	4.224			
	Huynh-Feldt	1004.131	246.158	4.079			
	Lower-bound	1004.131	87.000	11.542			

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	50.435	1	50.435	2.735	0.102	0.030
Error	1604.143	87	18.438			

ST/BMI risk and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
BMI_Binary_Cat	1.00	NormalRisk	65
	2.00	Increased Risk	23

Descriptive Statistics

BMI_Binary_Cat		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	NormalRisk	-0.65153	3.045578	65
	IncreasedRisk	-0.63230	2.756012	23
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	NormalRisk	-0.52216	3.408764	65
	IncreasedRisk	-0.73243	2.211947	23
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	NormalRisk	-0.29584	1.931229	65
	IncreasedRisk	0.44810	1.830940	23
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	NormalRisk	-0.28102	3.157258	65
	IncreasedRisk	0.07076	1.235023	23
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Mauchly's W		df	Sig.	Epsilon[b]

Within Subjects Effect		Approx. Chi-Square			Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.868	11.953	5	0.035	0.910	0.954	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + BMI_Binary_Cat
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	27.254	3	9.085	2.355	0.072	0.027
	Greenhouse-Geisser	27.254	2.731	9.980	2.355	0.079	0.027
	Huynh-Feldt	27.254	2.862	9.522	2.355	0.076	0.027
	Lower-bound	27.254	1.000	27.254	2.355	0.129	0.027
Region * BMI_Binary_Cat	Sphericity Assumed	8.786	3	2.929	0.759	0.518	0.009
	Greenhouse-Geisser	8.786	2.731	3.217	0.759	0.507	0.009
	Huynh-Feldt	8.786	2.862	3.070	0.759	0.512	0.009
	Lower-bound	8.786	1.000	8.786	0.759	0.386	0.009
Error(Region)	Sphericity Assumed	995.345	258	3.858			
	Greenhouse-Geisser	995.345	234.862	4.238			
	Huynh-Feldt	995.345	246.141	4.044			
	Lower-bound	995.345	86.000	11.574			

Tests of Between-Subjects Effects

Measure:
Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	28.632	1	28.632	1.538	0.218	0.018
BMI_Binary_Cat	3.476	1	3.476	0.187	0.667	0.002
Error	1600.667	86	18.612			

ST/ WAIST risk and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Waist_Cat_MF	1.00	Low	41
	2.00	Increased	47

Descriptive Statistics

Waist_Cat_MF		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	Low	-0.30658	2.203439	41
	Increased	-0.94304	3.481842	47
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	Low	-0.31043	3.520025	41
	Increased	-0.80976	2.762194	47
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	Low	-0.12699	2.066035	41
	Increased	-0.07908	1.812163	47
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	Low	0.18553	2.061401	41
	Increased	-0.51586	3.273348	47
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.861	12.634	5	0.027	0.906	0.950	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Waist_Cat_MF
Within Subjects Design: Region

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	18.754	3	6.251	1.618	0.185	0.018
	Greenhouse-Geisser	18.754	2.719	6.898	1.618	0.190	0.018
	Huynh-Feldt	18.754	2.849	6.583	1.618	0.188	0.018
	Lower-bound	18.754	1.000	18.754	1.618	0.207	0.018
Region * Waist_Cat_MF	Sphericity Assumed	7.627	3	2.542	0.658	0.578	0.008
	Greenhouse-Geisser	7.627	2.719	2.805	0.658	0.564	0.008
	Huynh-Feldt	7.627	2.849	2.677	0.658	0.571	0.008
	Lower-bound	7.627	1.000	7.627	0.658	0.419	0.008
Error(Region)	Sphericity Assumed	996.504	258	3.862			
	Greenhouse-Geisser	996.504	233.802	4.262			
	Huynh-Feldt	996.504	244.983	4.068			
	Lower-bound	996.504	86.000	11.587			

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	46.237	1	46.237	2.506	0.117	0.028
Waist_Cat_MF	17.526	1	17.526	0.950	0.332	0.011
Error	1586.617	86	18.449			

ST/ ANXIETY and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
ANX_Clin	1.00	LowNonClin	69
	2.00	Clinical	19

Descriptive Statistics

ANX_Clin		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	LowNonClin	-0.84706	3.014026	69
	Clinical	0.08183	2.689009	19
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	LowNonClin	-0.70385	3.283834	69
	Clinical	-0.11686	2.512572	19
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	LowNonClin	-0.34871	1.778162	69
	Clinical	0.79672	2.202257	19
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	LowNonClin	-0.38896	3.019794	69
	Clinical	0.53679	1.504779	19
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Mauchly's W		df	Sig.	Epsilon[b]

Within Subjects Effect		Approx. Chi-Square			Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.868	11.950	5	0.035	0.910	0.954	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + ANX_Clin
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	18.423	3	6.141	1.582	0.194	0.018
	Greenhouse-Geisser	18.423	2.731	6.746	1.582	0.198	0.018
	Huynh-Feldt	18.423	2.862	6.437	1.582	0.196	0.018
	Lower-bound	18.423	1.000	18.423	1.582	0.212	0.018
Region * ANX_Clin	Sphericity Assumed	2.379	3	0.793	0.204	0.893	0.002
	Greenhouse-Geisser	2.379	2.731	0.871	0.204	0.877	0.002
	Huynh-Feldt	2.379	2.862	0.831	0.204	0.885	0.002
	Lower-bound	2.379	1.000	2.379	0.204	0.652	0.002
Error(Region)	Sphericity Assumed	1001.752	258	3.883			
	Greenhouse-Geisser	1001.752	234.869	4.265			
	Huynh-Feldt	1001.752	246.148	4.070			
	Lower-bound	1001.752	86.000	11.648			

Tests of Between-Subjects Effects

Measure:
Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	3.651	1	3.651	0.202	0.654	0.002
ANX_Clin	47.922	1	47.922	2.648	0.107	0.030
Error	1556.221	86	18.096			

Estimated Marginal Means

1. Region

Measure:

Region	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-0.383	0.382	-1.142	0.377
2	-0.410	0.407	-1.218	0.398
3	0.224	0.243	-0.259	0.707
4	0.074	0.359	-0.640	0.788

2. ANX_Clin

Measure:

ANX_Clin	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
LowNonClin	-0.572	0.256	-1.081	-0.063
Clinical	0.325	0.488	-0.645	1.295

3. ANX_Clin * Region

Measure:

ANX_Clin		Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
LowNonClin	1	-0.847	0.355	-1.553	-0.141
	2	-0.704	0.378	-1.455	0.047
	3	-0.349	0.226	-0.797	0.100
	4	-0.389	0.334	-1.052	0.274
Clinical	1	0.082	0.677	-1.263	1.427
	2	-0.117	0.720	-1.548	1.314
	3	0.797	0.430	-0.058	1.652
	4	0.537	0.636	-0.727	1.801

Profile Plots

ST/ DEPRESSION and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Dep_Clin	1.00	LowNonClin	68
	2.00	Clinical	20

Descriptive Statistics

Dep_Clin		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	LowNonClin	-0.87495	2.980348	68
	Clinical	0.13020	2.809799	20
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	LowNonClin	-0.80942	3.290256	68
	Clinical	0.21272	2.412419	20
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	LowNonClin	-0.31861	1.834406	68
	Clinical	0.63710	2.080955	20
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	LowNonClin	-0.38741	3.012031	68
	Clinical	0.48524	1.686063	20
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Mauchly's W		df	Sig.	Epsilon[b]

Within Subjects Effect		Approx. Chi-Square			Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.869	11.912	5	0.036	0.911	0.955	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + DEP_Clin
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	12.483	3	4.161	1.069	0.363	0.012
	Greenhouse-Geisser	12.483	2.732	4.569	1.069	0.359	0.012
	Huynh-Feldt	12.483	2.864	4.359	1.069	0.361	0.012
	Lower-bound	12.483	1.000	12.483	1.069	0.304	0.012
Region * DEP_Clin	Sphericity Assumed	0.208	3	0.069	0.018	0.997	0.000
	Greenhouse-Geisser	0.208	2.732	0.076	0.018	0.995	0.000
	Huynh-Feldt	0.208	2.864	0.073	0.018	0.996	0.000
	Lower-bound	0.208	1.000	0.208	0.018	0.894	0.000
Error(Region)	Sphericity Assumed	1003.922	258	3.891			
	Greenhouse-Geisser	1003.922	234.992	4.272			
	Huynh-Feldt	1003.922	246.282	4.076			
	Lower-bound	1003.922	86.000	11.674			

Tests of Between-Subjects Effects

Measure:
Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	3.307	1	3.307	0.184	0.669	0.002
DEP_Clin	57.437	1	57.437	3.194	0.077	0.036
Error	1546.706	86	17.985			

ST/RUMINATION and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMt_Binary Low_Mod+	1.00	Low	45
	2.00	Mod/High	43

Descriptive Statistics

RUMt_Binary Low_Mod+		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	Low	-0.97640	3.053214	45
	Mod/High	-0.30126	2.847508	43
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	Low	-1.30911	3.120622	45
	Mod/High	0.18893	2.984963	43
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	Low	-0.56240	1.909437	45
	Mod/High	0.38104	1.837816	43
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	Low	-0.27762	1.690777	45
	Mod/High	-0.09642	3.609256	43
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.868	11.977	5	0.035	0.911	0.955	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + RUMt_Binary
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	19.342	3	6.447	1.690	0.170	0.019
	Greenhouse-Geisser	19.342	2.734	7.074	1.690	0.174	0.019
	Huynh-Feldt	19.342	2.866	6.750	1.690	0.172	0.019
	Lower-bound	19.342	1.000	19.342	1.690	0.197	0.019
Region * RUMt_Binary	Sphericity Assumed	19.877	3	6.626	1.737	0.160	0.020
	Greenhouse-Geisser	19.877	2.734	7.270	1.737	0.165	0.020
	Huynh-Feldt	19.877	2.866	6.936	1.737	0.162	0.020
	Lower-bound	19.877	1.000	19.877	1.737	0.191	0.020
Error(Region)	Sphericity Assumed	984.254	258	3.815			
	Greenhouse-Geisser	984.254	235.139	4.186			
	Huynh-Feldt	984.254	246.443	3.994			
	Lower-bound	984.254	86.000	11.445			

Tests of Between-Subjects Effects

Measure:
Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	47.945	1	47.945	2.670	0.106	0.030
RUMt_Binary	59.785	1	59.785	3.329	0.072	0.037
Error	1544.358	86	17.958			

Estimated Marginal Means

1. Region

Measure:

Region	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-0.639	0.315	-1.265	-0.013
2	-0.560	0.326	-1.208	0.087
3	-0.091	0.200	-0.488	0.307
4	-0.187	0.298	-0.780	0.406

2. RUMt_Binary Low_Mod+

Measure:

RUMt_Binary Low_Mod+	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
Low	-0.781	0.316	-1.409	-0.153
Mod/High	0.043	0.323	-0.599	0.685

3. RUMt_Binary Low_Mod+ * Region

Measure:

RUMt_Binary Low_Mod+		Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Low	1	-0.976	0.440	-1.852	-0.101
	2	-1.309	0.455	-2.214	-0.404
	3	-0.562	0.279	-1.118	-0.007
	4	-0.278	0.417	-1.107	0.551
Mod/High	1	-0.301	0.451	-1.197	0.594
	2	0.189	0.466	-0.737	1.115
	3	0.381	0.286	-0.187	0.949
	4	-0.096	0.427	-0.944	0.752

ST/ Brooding Rumination and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMb_Binary Low_Mod+	1.00	Low	49
	2.00	Mod/High	39

Descriptive Statistics

RUMb_Binary Low_Mod+		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	Low	-0.86039	2.925125	49
	Mod/High	-0.37778	3.013048	39
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	Low	-1.07594	3.180057	49
	Mod/High	0.04961	2.987369	39
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	Low	-0.27556	1.963253	49
	Mod/High	0.11741	1.874259	39
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	Low	-0.10889	1.745638	49
	Mod/High	-0.28983	3.722794	39
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.876	11.185	5	0.048	0.916	0.961	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + RUMb_Binary
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	16.947	3	5.649	1.479	0.221	0.017
	Greenhouse-Geisser	16.947	2.749	6.165	1.479	0.224	0.017
	Huynh-Feldt	16.947	2.882	5.881	1.479	0.222	0.017
	Lower-bound	16.947	1.000	16.947	1.479	0.227	0.017
Region * RUMb_Binary	Sphericity Assumed	18.647	3	6.216	1.627	0.183	0.019
	Greenhouse-Geisser	18.647	2.749	6.783	1.627	0.188	0.019
	Huynh-Feldt	18.647	2.882	6.471	1.627	0.185	0.019
	Lower-bound	18.647	1.000	18.647	1.627	0.206	0.019
Error(Region)	Sphericity Assumed	985.484	258	3.820			
	Greenhouse-Geisser	985.484	236.402	4.169			
	Huynh-Feldt	985.484	247.820	3.977			
	Lower-bound	985.484	86.000	11.459			

Tests of Between-Subjects Effects

Measure:
Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	43.215	1	43.215	2.343	0.130	0.027
RUMb_Binary	17.987	1	17.987	0.975	0.326	0.011
Error	1586.157	86	18.444			

ST/ Reflective Rumination and OHb During The Inhibition Task with post hocs

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
RUMr_Binary Low_Mod+	1.00	Low	45
	2.00	Mod/High	43

Descriptive Statistics

RUMr_Binary Low_Mod+		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	Low	-0.96620	3.074060	45
	Mod/High	-0.31194	2.826486	43
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	Low	-1.42673	3.053166	45
	Mod/High	0.31202	2.989713	43
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	Low	-0.66011	1.984026	45
	Mod/High	0.48329	1.689636	43
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	Low	-0.23351	1.813304	45
	Mod/High	-0.14258	3.548211	43
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.861	12.645	5	0.027	0.908	0.952	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + RUMr_Binary
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	19.340	3	6.447	1.712	0.165	0.020
	Greenhouse-Geisser	19.340	2.724	7.100	1.712	0.170	0.020
	Huynh-Feldt	19.340	2.855	6.775	1.712	0.168	0.020
	Lower-bound	19.340	1.000	19.340	1.712	0.194	0.020
Region * RUMr_Binary	Sphericity Assumed	32.490	3	10.830	2.876	0.037	0.032
	Greenhouse-Geisser	32.490	2.724	11.926	2.876	0.042	0.032
	Huynh-Feldt	32.490	2.855	11.381	2.876	0.039	0.032
	Lower-bound	32.490	1.000	32.490	2.876	0.094	0.032
Error(Region)	Sphericity Assumed	971.641	258	3.766			
	Greenhouse-Geisser	971.641	234.279	4.147			
	Huynh-Feldt	971.641	245.504	3.958			
	Lower-bound	971.641	86.000	11.298			

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	47.702	1	47.702	2.678	0.105	0.030
RUMr_Binary	72.329	1	72.329	4.061	0.047	0.045
Error	1531.814	86	17.812			

Estimated Marginal Means

1. Region

Estimates

Measure:

Region	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-0.639	0.315	-1.266	-0.013
2	-0.557	0.322	-1.198	0.083
3	-0.088	0.197	-0.480	0.303
4	-0.188	0.298	-0.781	0.405

Pairwise Comparisons

Measure:

(I) Region		Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-0.082	0.258	1.000	-0.779	0.615
	3	-0.551	0.266	0.251	-1.270	0.169
	4	-0.451	0.331	1.000	-1.344	0.442
2	1	0.082	0.258	1.000	-0.615	0.779
	3	-0.469	0.292	0.675	-1.259	0.321
	4	-0.369	0.323	1.000	-1.241	0.502
3	1	0.551	0.266	0.251	-0.169	1.270
	2	0.469	0.292	0.675	-0.321	1.259
	4	0.100	0.278	1.000	-0.650	0.850
4	1	0.451	0.331	1.000	-0.442	1.344
	2	0.369	0.323	1.000	-0.502	1.241
	3	-0.100	0.278	1.000	-0.850	0.650

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Pillai's trace	0.051	1.509[a]	3.000	84.000	0.218	0.051
Wilks' lambda	0.949	1.509[a]	3.000	84.000	0.218	0.051
Hotelling's trace	0.054	1.509[a]	3.000	84.000	0.218	0.051

Roy's largest root	0.054	1.509[a]	3.000	84.000	0.218	0.051

Each F tests the multivariate effect of Region. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.
a. Exact statistic

2. RUMr_Binary Low_Mod+

Estimates

Measure:

RUMr_Binary Low_Mod+	Mean	Std. Error	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
Low	-0.822	0.315	-1.447	-0.196
Mod/High	0.085	0.322	-0.555	0.725

Pairwise Comparisons

Measure:

(I) RUMr_Binary Low_Mod+		Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b] Lower Bound	95% Confidence Interval for Difference[b] Upper Bound
Low	Mod/High	-.907*	0.450	0.047	-1.801	-0.012
Mod/High	Low	.907*	0.450	0.047	0.012	1.801

Based on estimated marginal means
*. The mean difference is significant at the .05 level.
b. Adjustment for multiple comparisons: Bonferroni.

3. RUMr_Binary Low_Mod+ * Region

Estimates

Measure:

RUMr_Binary Low_Mod+		Mean	Std. Error	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
Low	1	-0.966	0.441	-1.842	-0.090
	2	-1.427	0.451	-2.322	-0.531
	3	-0.660	0.275	-1.207	-0.113

	4	-0.234	0.417	-1.063	0.596
Mod/High	1	-0.312	0.451	-1.208	0.584
	2	0.312	0.461	-0.604	1.228
	3	0.483	0.282	-0.076	1.043
	4	-0.143	0.427	-0.991	0.706

Pairwise Comparisons

Measure:

Region			Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
						Lower Bound	Upper Bound
1	Low	Mod/High	-0.654	0.630	0.302	-1.907	0.599
	Mod/High	Low	0.654	0.630	0.302	-0.599	1.907
2	Low	Mod/High	-1.739*	0.645	0.008	-3.020	-0.457
	Mod/High	Low	1.739*	0.645	0.008	0.457	3.020
3	Low	Mod/High	-1.143*	0.394	0.005	-1.926	-0.361
	Mod/High	Low	1.143*	0.394	0.005	0.361	1.926
4	Low	Mod/High	-0.091	0.597	0.879	-1.277	1.095
	Mod/High	Low	0.091	0.597	0.879	-1.095	1.277

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

4. RUMr_Binary Low_Mod+ * Region

Estimates

Measure:

RUMr_Binary Low_Mod+		Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Low	1	-0.966	0.441	-1.842	-0.090
	2	-1.427	0.451	-2.322	-0.531
	3	-0.660	0.275	-1.207	-0.113
	4	-0.234	0.417	-1.063	0.596

Mod/High	1	-0.312	0.451	-1.208	0.584
	2	0.312	0.461	-0.604	1.228
	3	0.483	0.282	-0.076	1.043
	4	-0.143	0.427	-0.991	0.706

Pairwise Comparisons

Measure:

RUMr_Binary Low_Mod+			Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a] Lower Bound	Upper Bound
Low	1	2	0.461	0.361	1.000	-0.514	1.435
		3	-0.306	0.373	1.000	-1.312	0.700
		4	-0.733	0.462	0.701	-1.982	0.516
	2	1	-0.461	0.361	1.000	-1.435	0.514
		3	-0.767	0.409	0.385	-1.871	0.338
		4	-1.193	0.451	0.058	-2.412	0.025
	3	1	0.306	0.373	1.000	-0.700	1.312
		2	0.767	0.409	0.385	-0.338	1.871
		4	-0.427	0.388	1.000	-1.475	0.622
	4	1	0.733	0.462	0.701	-0.516	1.982
		2	1.193	0.451	0.058	-0.025	2.412
		3	0.427	0.388	1.000	-0.622	1.475
Mod/High	1	2	-0.624	0.369	0.567	-1.621	0.373
		3	-0.795	0.381	0.239	-1.824	0.234
		4	-0.169	0.473	1.000	-1.447	1.108
	2	1	0.624	0.369	0.567	-0.373	1.621
		3	-0.171	0.418	1.000	-1.301	0.958
		4	0.455	0.462	1.000	-0.792	1.701
	3	1	0.795	0.381	0.239	-0.234	1.824
		2	0.171	0.418	1.000	-0.958	1.301
		4	0.626	0.397	0.712	-0.447	1.698
	4	1	0.169	0.473	1.000	-1.108	1.447
		2	-0.455	0.462	1.000	-1.701	0.792
		3	-0.626	0.397	0.712	-1.698	0.447

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

ST/WORRY and OHb During the Inhibition Task – Non Sig

Within-Subjects Factors

Measure:

Region	Dependent Variable
1	RF_OS_L2AvgB1_2
2	LF_OS_L2AvgB1_2
3	RT_OS_L2AvgB1_2
4	LT_OS_L2AvgB1_2

Between-Subjects Factors

		Value Label	N
Worry_Binary	1.00	Low/none	58
	2.00	Mod/High	30

Descriptive Statistics

Worry_Binary		Mean	Std. Deviation	N
RF_OS_L2AvgB1_2	Low/none	-0.87288	3.071755	58
	Mod/High	-0.20886	2.717710	30
	Total	-0.64651	2.957057	88
LF_OS_L2AvgB1_2	Low/none	-0.82376	3.396128	58
	Mod/High	-0.10027	2.520810	30
	Total	-0.57712	3.129485	88
RT_OS_L2AvgB1_2	Low/none	-0.40794	1.806259	58
	Mod/High	0.49123	2.033328	30
	Total	-0.10140	1.923393	88
LT_OS_L2AvgB1_2	Low/none	-0.13272	1.882237	58
	Mod/High	-0.29804	4.030691	30
	Total	-0.18908	2.782599	88

Mauchly's Test of Sphericity[a]

Measure:

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Region	0.870	11.825	5	0.037	0.912	0.955	0.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.
a. Design: Intercept + Worry_Binary
Within Subjects Design: Region
b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure:

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Region	Sphericity Assumed	16.452	3	5.484	1.428	0.235	0.016
	Greenhouse-Geisser	16.452	2.735	6.015	1.428	0.238	0.016
	Huynh-Feldt	16.452	2.866	5.739	1.428	0.236	0.016
	Lower-bound	16.452	1.000	16.452	1.428	0.235	0.016
Region * Worry_Binary	Sphericity Assumed	13.350	3	4.450	1.159	0.326	0.013
	Greenhouse-Geisser	13.350	2.735	4.881	1.159	0.324	0.013
	Huynh-Feldt	13.350	2.866	4.657	1.159	0.325	0.013
	Lower-bound	13.350	1.000	13.350	1.159	0.285	0.013
Error(Region)	Sphericity Assumed	990.781	258	3.840			
	Greenhouse-Geisser	990.781	235.205	4.212			
	Huynh-Feldt	990.781	246.515	4.019			
	Lower-bound	990.781	86.000	11.521			

Tests of Between-Subjects Effects

Measure:

Transformed Variable:

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	27.374	1	27.374	1.488	0.226	0.017
Worry_Binary	22.245	1	22.245	1.209	0.275	0.014
Error	1581.898	86	18.394			

P4 Functional Connectivity Pearson Correlation Matrix SPSS

Brooding Rumination Resting State Functional Connectivity Correlation

Correlations

Low_Mod+		RT1	RT2	RT3	RF1	RF2	RF3	RF4	RF5	LT1	LT2	LT3	LF1	LF2	LF3	LF4	LF5
Low																	
RT1	r	1	.796**	.605**	0.121	.801**	.850**	.586**	.456**	-0.023	.500**	.449**	-0.101	-0.023	-0.070	-0.062	-0.185
	p		0.000	0.000	0.407	0.000	0.000	0.000	0.001	0.877	0.000	0.001	0.488	0.875	0.633	0.670	0.202
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT2	r	.796**	1	.731**	.403**	.891**	.840**	.744**	.616**	-0.060	.403**	.535**	-0.070	-0.110	-0.102	0.015	-0.046
	p	0.000		0.000	0.004	0.000	0.000	0.000	0.000	0.683	0.004	0.000	0.630	0.453	0.486	0.917	0.753
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT3	r	.605**	.731**	1	.391**	.829**	.706**	.797**	.725**	-.401**	0.208	.778**	0.057	-0.082	-0.095	0.059	0.047
	p	0.000	0.000		0.005	0.000	0.000	0.000	0.000	0.004	0.152	0.000	0.698	0.574	0.515	0.686	0.749
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF1	r	0.121	.403**	.391**	1	.376**	.371**	.352*	0.186	-0.021	0.004	.512**	.427**	0.161	0.031	-0.004	0.107
	p	0.407	0.004	0.005		0.008	0.009	0.013	0.200	0.883	0.978	0.000	0.002	0.268	0.832	0.979	0.462
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF2	r	.801**	.891**	.829**	.376**	1	.913**	.724**	.687**	-0.149	.400**	.609**	-0.058	-0.064	-0.094	-0.004	-0.072
	p	0.000	0.000	0.000	0.008		0.000	0.000	0.000	0.307	0.004	0.000	0.693	0.663	0.518	0.976	0.625
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF3	r	.850**	.840**	.706**	.371**	.913**	1	.754**	.577**	-0.128	.376**	.605**	0.001	-0.015	-0.101	-0.087	-0.118
	p	0.000	0.000	0.000	0.009	0.000		0.000	0.000	0.382	0.008	0.000	0.996	0.916	0.492	0.554	0.418
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF4	r	.586**	.744**	.797**	.352*	.724**	.754**	1	.741**	-.363*	0.240	.696**	0.010	-0.139	-0.065	0.117	0.015
	p	0.000	0.000	0.000	0.013	0.000	0.000		0.000	0.010	0.097	0.000	0.943	0.341	0.658	0.422	0.916

	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF5	r	.456**	.616**	.725**	0.186	.687**	.577**	.741**	1	-.344*	0.234	.526**	-0.024	-0.188	-0.026	0.235	0.061
	p	0.001	0.000	0.000	0.200	0.000	0.000	0.000		0.016	0.106	0.000	0.868	0.195	0.859	0.104	0.675
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT1	r	-0.023	-0.060	-.401**	-0.021	-0.149	-0.128	-.363*	-.344*	1	.622**	-0.202	-0.027	.375**	.341*	0.103	-0.115
	p	0.877	0.683	0.004	0.883	0.307	0.382	0.010	0.016		0.000	0.165	0.854	0.008	0.017	0.483	0.432
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT2	r	.500**	.403**	0.208	0.004	.400**	.376**	0.240	0.234	.622**	1	.352*	-0.116	0.011	0.029	-0.010	-0.235
	p	0.000	0.004	0.152	0.978	0.004	0.008	0.097	0.106	0.000		0.013	0.426	0.940	0.842	0.946	0.104
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT3	r	.449**	.535**	.778**	.512**	.609**	.605**	.696**	.526**	-0.202	.352*	1	0.152	-0.040	-0.125	-0.073	0.051
	p	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.165	0.013		0.297	0.783	0.392	0.618	0.726
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF1	r	-0.101	-0.070	0.057	.427**	-0.058	0.001	0.010	-0.024	-0.027	-0.116	0.152	1	0.112	0.008	0.056	0.059
	p	0.488	0.630	0.698	0.002	0.693	0.996	0.943	0.868	0.854	0.426	0.297		0.444	0.956	0.703	0.689
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF2	r	-0.023	-0.110	-0.082	0.161	-0.064	-0.015	-0.139	-0.188	.375**	0.011	-0.040	0.112	1	.888**	.563**	.312*
	p	0.875	0.453	0.574	0.268	0.663	0.916	0.341	0.195	0.008	0.940	0.783	0.444		0.000	0.000	0.029
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF3	r	-0.070	-0.102	-0.095	0.031	-0.094	-0.101	-0.065	-0.026	.341*	0.029	-0.125	0.008	.888**	1	.828**	.325*
	p	0.633	0.486	0.515	0.832	0.518	0.492	0.658	0.859	0.017	0.842	0.392	0.956	0.000		0.000	0.023
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF4	r	-0.062	0.015	0.059	-0.004	-0.004	-0.087	0.117	0.235	0.103	-0.010	-0.073	0.056	.563**	.828**	1	.314*
	p	0.670	0.917	0.686	0.979	0.976	0.554	0.422	0.104	0.483	0.946	0.618	0.703	0.000	0.000		0.028
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF5	r	-0.185	-0.046	0.047	0.107	-0.072	-0.118	0.015	0.061	-0.115	-0.235	0.051	0.059	.312*	.325*	.314*	1
	p	0.202	0.753	0.749	0.462	0.625	0.418	0.916	0.675	0.432	0.104	0.726	0.689	0.029	0.023	0.028	

	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
Mod/High																	
RT1	r	1	.924**	.431**	.458**	.578**	.570**	0.226	0.250	.494**	.509**	.508**	.331*	.383*	.443**	.534**	.448**
	p		0.000	0.006	0.003	0.000	0.000	0.167	0.125	0.001	0.001	0.001	0.039	0.016	0.005	0.000	0.004
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RT2	r	.924**	1	.468**	.480**	.521**	.508**	0.134	0.145	.529**	.538**	.549**	0.290	.473**	.507**	.598**	.562**
	p	0.000		0.003	0.002	0.001	0.001	0.416	0.378	0.001	0.000	0.000	0.073	0.002	0.001	0.000	0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RT3	r	.431**	.468**	1	.568**	.494**	.472**	.530**	.400*	.354*	.395*	.502**	.324*	0.303	0.284	0.075	0.115
	p	0.006	0.003		0.000	0.001	0.002	0.001	0.012	0.027	0.013	0.001	0.044	0.061	0.080	0.649	0.487
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF1	r	.458**	.480**	.568**	1	.662**	.630**	.519**	.406*	0.081	0.206	0.274	0.248	0.170	0.304	0.159	-0.046
	p	0.003	0.002	0.000		0.000	0.000	0.001	0.010	0.623	0.208	0.091	0.128	0.302	0.060	0.334	0.779
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF2	r	.578**	.521**	.494**	.662**	1	.946**	.623**	.517**	0.111	0.068	0.082	0.194	0.120	.340*	.435**	-0.035
	p	0.000	0.001	0.001	0.000		0.000	0.000	0.001	0.500	0.681	0.621	0.236	0.466	0.034	0.006	0.833
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF3	r	.570**	.508**	.472**	.630**	.946**	1	.740**	.666**	0.129	0.053	0.118	0.135	0.161	.423**	.557**	0.068
	p	0.000	0.001	0.002	0.000	0.000		0.000	0.000	0.433	0.749	0.476	0.413	0.328	0.007	0.000	0.682
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF4	r	0.226	0.134	.530**	.519**	.623**	.740**	1	.802**	-0.043	-0.089	-0.027	0.157	-0.021	0.202	0.241	-0.065
	p	0.167	0.416	0.001	0.001	0.000	0.000		0.000	0.796	0.591	0.872	0.340	0.901	0.218	0.140	0.694
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF5	r	0.250	0.145	.400*	.406*	.517**	.666**	.802**	1	0.005	-0.087	0.127	0.229	0.182	.345*	.430**	0.115
	p	0.125	0.378	0.012	0.010	0.001	0.000	0.000		0.976	0.598	0.441	0.161	0.269	0.031	0.006	0.487
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39

LT1	r	.494**	.529**	.354*	0.081	0.111	0.129	-0.043	0.005	1	.827**	.438**	0.203	.548**	.469**	.365*	.418**
	p	0.001	0.001	0.027	0.623	0.500	0.433	0.796	0.976		0.000	0.005	0.215	0.000	0.003	0.022	0.008
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LT2	r	.509**	.538**	.395*	0.206	0.068	0.053	-0.089	-0.087	.827**	1	.602**	0.139	.591**	.492**	0.241	.346*
	p	0.001	0.000	0.013	0.208	0.681	0.749	0.591	0.598	0.000		0.000	0.398	0.000	0.001	0.139	0.031
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LT3	r	.508**	.549**	.502**	0.274	0.082	0.118	-0.027	0.127	.438**	.602**	1	0.220	.655**	.481**	0.290	.513**
	p	0.001	0.000	0.001	0.091	0.621	0.476	0.872	0.441	0.005	0.000		0.179	0.000	0.002	0.073	0.001
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF1	r	.331*	0.290	.324*	0.248	0.194	0.135	0.157	0.229	0.203	0.139	0.220	1	.329*	0.232	0.175	0.208
	p	0.039	0.073	0.044	0.128	0.236	0.413	0.340	0.161	0.215	0.398	0.179		0.041	0.156	0.285	0.204
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF2	r	.383*	.473**	0.303	0.170	0.120	0.161	-0.021	0.182	.548**	.591**	.655**	.329*	1	.884**	.508**	.557**
	p	0.016	0.002	0.061	0.302	0.466	0.328	0.901	0.269	0.000	0.000	0.000	0.041		0.000	0.001	0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF3	r	.443**	.507**	0.284	0.304	.340*	.423**	0.202	.345*	.469**	.492**	.481**	0.232	.884**	1	.666**	.526**
	p	0.005	0.001	0.080	0.060	0.034	0.007	0.218	0.031	0.003	0.001	0.002	0.156	0.000		0.000	0.001
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF4	r	.534**	.598**	0.075	0.159	.435**	.557**	0.241	.430**	.365*	0.241	0.290	0.175	.508**	.666**	1	.674**
	p	0.000	0.000	0.649	0.334	0.006	0.000	0.140	0.006	0.022	0.139	0.073	0.285	0.001	0.000		0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF5	r	.448**	.562**	0.115	-0.046	-0.035	0.068	-0.065	0.115	.418**	.346*	.513**	0.208	.557**	.526**	.674**	1
	p	0.004	0.000	0.487	0.779	0.833	0.682	0.694	0.487	0.008	0.031	0.001	0.204	0.000	0.001	0.000	
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39

Brooding Rumination During Attention Task Functional Connectivity Correlation

Low RUMb		RF1_OC	RF2_OC	RF3_OC	RF4_OC	RF5_OC	LF1_OC	LF2_OC	LF3_OC	LF4_OC	LF5_OC	RT1_OC	RT2_OC	RT3_OC	LT1_OC	LT2_OC	LT3_OC
RF1_OC	r	1	.639**	0.270	.507**	.518**	.770**	.735**	.561**	.335*	0.145	0.179	.421**	.668**	0.124	.293*	.805**
	p		0.000	0.060	0.000	0.000	0.000	0.000	0.000	0.019	0.319	0.219	0.003	0.000	0.398	0.041	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF2_OC	r	.639**	1	.819**	.317*	.491**	.589**	.625**	.471**	.300*	-0.014	.664**	.508**	.683**	0.064	0.200	.619**
	p	0.000		0.000	0.027	0.000	0.000	0.000	0.001	0.036	0.923	0.000	0.000	0.000	0.661	0.168	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF3_OC	r	0.270	.819**	1	0.184	0.221	.316*	.333*	0.281	0.218	-0.011	.893**	.371**	.488**	-0.009	0.094	.322*
	p	0.060	0.000		0.207	0.127	0.027	0.019	0.050	0.133	0.938	0.000	0.009	0.000	0.950	0.519	0.024
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF4_OC	r	.507**	.317*	0.184	1	.658**	.523**	.472**	.448**	.656**	-0.042	0.069	.396**	.468**	0.094	0.131	.531**
	p	0.000	0.027	0.207		0.000	0.000	0.001	0.001	0.000	0.774	0.636	0.005	0.001	0.520	0.370	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF5_OC	r	.518**	.491**	0.221	.658**	1	.438**	.541**	.517**	.410**	-0.008	0.048	0.213	.477**	0.043	0.170	.533**
	p	0.000	0.000	0.127	0.000		0.002	0.000	0.000	0.003	0.957	0.742	0.142	0.001	0.768	0.243	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF1_OC	r	.770**	.589**	.316*	.523**	.438**	1	.691**	.621**	.501**	0.081	0.193	.433**	.595**	.348*	.492**	.785**
	p	0.000	0.000	0.027	0.000	0.002		0.000	0.000	0.000	0.582	0.185	0.002	0.000	0.014	0.000	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49

LF2_OC	r	.735**	.625**	.333*	.472**	.541**	.691**	1	.878**	.385**	0.027	0.191	.371**	.628**	0.059	0.261	.757**
	p	0.000	0.000	0.019	0.001	0.000	0.000		0.000	0.006	0.852	0.188	0.009	0.000	0.686	0.070	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF3_OC	r	.561**	.471**	0.281	.448**	.517**	.621**	.878**	1	.426**	-0.001	0.125	0.225	.531**	0.195	.459**	.628**
	p	0.000	0.001	0.050	0.001	0.000	0.000	0.000		0.002	0.997	0.394	0.120	0.000	0.179	0.001	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF4_OC	r	.335*	.300*	0.218	.656**	.410**	.501**	.385**	.426**	1	-.347*	0.083	0.153	.338*	0.082	0.162	.385**
	p	0.019	0.036	0.133	0.000	0.003	0.000	0.006	0.002		0.014	0.573	0.294	0.017	0.573	0.267	0.006
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF5_OC	r	0.145	-0.014	-0.011	-0.042	-0.008	0.081	0.027	-0.001	-.347*	1	0.026	0.101	0.014	-0.024	0.050	0.148
	p	0.319	0.923	0.938	0.774	0.957	0.582	0.852	0.997	0.014		0.860	0.488	0.926	0.868	0.734	0.310
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT1_OC	r	0.179	.664**	.893**	0.069	0.048	0.193	0.191	0.125	0.083	0.026	1	.439**	.449**	0.043	-0.004	.284*
	p	0.219	0.000	0.000	0.636	0.742	0.185	0.188	0.394	0.573	0.860		0.002	0.001	0.768	0.977	0.048
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT2_OC	r	.421**	.508**	.371**	.396**	0.213	.433**	.371**	0.225	0.153	0.101	.439**	1	.443**	0.065	0.045	.526**
	p	0.003	0.000	0.009	0.005	0.142	0.002	0.009	0.120	0.294	0.488	0.002		0.001	0.656	0.758	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT3_OC	r	.668**	.683**	.488**	.468**	.477**	.595**	.628**	.531**	.338*	0.014	.449**	.443**	1	0.263	0.259	.617**
	p	0.000	0.000	0.000	0.001	0.001	0.000	0.000	0.000	0.017	0.926	0.001	0.001		0.067	0.072	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49

LT1_OC	r	0.124	0.064	-0.009	0.094	0.043	.348*	0.059	0.195	0.082	-0.024	0.043	0.065	0.263	1	.675**	0.232
	p	0.398	0.661	0.950	0.520	0.768	0.014	0.686	0.179	0.573	0.868	0.768	0.656	0.067		0.000	0.109
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT2_OC	r	.293*	0.200	0.094	0.131	0.170	.492**	0.261	.459**	0.162	0.050	-0.004	0.045	0.259	.675**	1	0.210
	p	0.041	0.168	0.519	0.370	0.243	0.000	0.070	0.001	0.267	0.734	0.977	0.758	0.072	0.000		0.148
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT3_OC	r	.805**	.619**	.322*	.531**	.533**	.785**	.757**	.628**	.385**	0.148	.284*	.526**	.617**	0.232	0.210	1
	p	0.000	0.000	0.024	0.000	0.000	0.000	0.000	0.000	0.006	0.310	0.048	0.000	0.000	0.109	0.148	
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49

Mod/High RUMb (Attention Task)

		RF1_OC	RF2_OC	RF3_OC	RF4_OC	RF5_OC	LF1_OC	LF2_OC	LF3_OC	LF4_OC	LF5_OC	RT1_OC	RT2_OC	RT3_OC	LT1_OC	LT2_OC	LT3_OC
RF1_OC	r	1	0.173	0.102	-0.070	-0.263	.561**	0.035	-0.015	-0.203	-0.169	0.023	0.091	.360*	0.041	-0.121	0.138
	p		0.299	0.541	0.676	0.111	0.000	0.836	0.930	0.221	0.310	0.891	0.588	0.026	0.806	0.468	0.410
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RF2_OC	r	0.173	1	.653**	0.222	-0.131	-0.032	0.169	-0.063	.323*	0.132	.612**	0.155	-0.020	-0.151	-0.007	-0.121
	p	0.299		0.000	0.181	0.433	0.849	0.310	0.707	0.048	0.430	0.000	0.352	0.906	0.367	0.968	0.471
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RF3_OC	r	0.102	.653**	1	.812**	0.122	0.057	0.223	0.138	.476**	0.204	.562**	.694**	0.102	-0.005	-0.001	.372*
	p	0.541	0.000		0.000	0.464	0.733	0.178	0.410	0.003	0.220	0.000	0.000	0.543	0.978	0.997	0.021

	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RF4_OC	r	-0.070	0.222	.812**	1	0.244	0.114	.324*	0.224	.403*	0.180	0.236	.754**	0.270	0.057	0.071	.630**
	p	0.676	0.181	0.000		0.140	0.495	0.047	0.176	0.012	0.278	0.154	0.000	0.102	0.735	0.670	0.000
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RF5_OC	r	-0.263	-0.131	0.122	0.244	1	-0.086	-0.076	0.145	-0.011	0.074	-0.078	0.311	-0.132	0.003	-0.120	0.056
	p	0.111	0.433	0.464	0.140		0.608	0.650	0.384	0.946	0.661	0.641	0.058	0.429	0.987	0.473	0.740
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LF1_OC	r	.561**	-0.032	0.057	0.114	-0.086	1	.520**	.454**	-0.171	-0.163	-0.107	0.202	0.185	.404*	0.001	.503**
	p	0.000	0.849	0.733	0.495	0.608		0.001	0.004	0.306	0.328	0.523	0.224	0.267	0.012	0.993	0.001
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LF2_OC	r	0.035	0.169	0.223	.324*	-0.076	.520**	1	.611**	0.101	0.041	0.239	.371*	0.127	0.090	0.284	.587**
	p	0.836	0.310	0.178	0.047	0.650	0.001		0.000	0.548	0.806	0.149	0.022	0.447	0.592	0.084	0.000
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LF3_OC	r	-0.015	-0.063	0.138	0.224	0.145	.454**	.611**	1	0.105	-0.139	-0.016	.341*	-0.094	.358*	-0.170	.348*
	p	0.930	0.707	0.410	0.176	0.384	0.004	0.000		0.529	0.404	0.923	0.036	0.574	0.027	0.308	0.033
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LF4_OC	r	-0.203	.323*	.476**	.403*	-0.011	-0.171	0.101	0.105	1	.744**	0.313	0.314	-0.064	-0.029	-.347*	.322*
	p	0.221	0.048	0.003	0.012	0.946	0.306	0.548	0.529		0.000	0.056	0.054	0.701	0.864	0.033	0.049
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LF5_OC	r	-0.169	0.132	0.204	0.180	0.074	-0.163	0.041	-0.139	.744**	1	0.279	0.254	-0.007	-0.219	-0.100	0.270
	p	0.310	0.430	0.220	0.278	0.661	0.328	0.806	0.404	0.000		0.090	0.124	0.966	0.186	0.551	0.101

	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RT1_OC	r	0.023	.612**	.562**	0.236	-0.078	-0.107	0.239	-0.016	0.313	0.279	1	.473**	0.053	-0.078	0.218	0.079
	p	0.891	0.000	0.000	0.154	0.641	0.523	0.149	0.923	0.056	0.090		0.003	0.753	0.642	0.188	0.637
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RT2_OC	r	0.091	0.155	.694**	.754**	0.311	0.202	.371*	.341*	0.314	0.254	.473**	1	0.314	0.147	0.036	.654**
	p	0.588	0.352	0.000	0.000	0.058	0.224	0.022	0.036	0.054	0.124	0.003		0.055	0.377	0.832	0.000
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
RT3_OC	r	.360*	-0.020	0.102	0.270	-0.132	0.185	0.127	-0.094	-0.064	-0.007	0.053	0.314	1	.408*	-0.058	.510**
	p	0.026	0.906	0.543	0.102	0.429	0.267	0.447	0.574	0.701	0.966	0.753	0.055		0.011	0.728	0.001
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LT1_OC	r	0.041	-0.151	-0.005	0.057	0.003	.404*	0.090	.358*	-0.029	-0.219	-0.078	0.147	.408*	1	-0.288	.440**
	p	0.806	0.367	0.978	0.735	0.987	0.012	0.592	0.027	0.864	0.186	0.642	0.377	0.011		0.080	0.006
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LT2_OC	r	-0.121	-0.007	-0.001	0.071	-0.120	0.001	0.284	-0.170	-.347*	-0.100	0.218	0.036	-0.058	-0.288	1	-0.012
	p	0.468	0.968	0.997	0.670	0.473	0.993	0.084	0.308	0.033	0.551	0.188	0.832	0.728	0.080		0.942
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38
LT3_OC	r	0.138	-0.121	.372*	.630**	0.056	.503**	.587**	.348*	.322*	0.270	0.079	.654**	.510**	.440**	-0.012	1
	p	0.410	0.471	0.021	0.000	0.740	0.001	0.000	0.033	0.049	0.101	0.637	0.000	0.001	0.006	0.942	
	N	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38	38

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

a. RUMb_Binary Low_Mod+ = Mod/High

Brooding Rumination During the Inhibition task Functional Connectivity Correlation

Low Rumination		RF1_OS	RF2_OS	RF3_OS	RF4_OS	RF5_OS	LF1_OS	LF2_OS	LF3_OS	LF4_OS	LF5_OS	RT1_OS	RT2_OS	RT3_OS	LT1_OS	LT2_OS	LT3_OS
RF1_OS	r	1	0.257	.320*	.577**	0.204	.602**	.532**	.442**	.403**	0.018	.454**	.693**	.739**	.428**	.408**	.300*
	p		0.075	0.025	0.000	0.160	0.000	0.000	0.001	0.004	0.905	0.001	0.000	0.000	0.002	0.004	0.037
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF2_OS	r	0.257	1	.685**	0.186	.406**	.320*	.509**	.368**	.282*	.485**	.529**	-0.230	.360*	.337*	0.263	.429**
	p	0.075		0.000	0.200	0.004	0.025	0.000	0.009	0.050	0.000	0.000	0.112	0.011	0.018	0.068	0.002
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF3_OS	r	.320*	.685**	1	.300*	.549**	0.248	.357*	.403**	.393**	.381**	.541**	0.028	0.214	.409**	.289*	0.104
	p	0.025	0.000		0.037	0.000	0.086	0.012	0.004	0.005	0.007	0.000	0.850	0.141	0.004	0.044	0.476
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF4_OS	r	.577**	0.186	.300*	1	.475**	.361*	.434**	.421**	.554**	0.253	.395**	.628**	.460**	.425**	.349*	.354*
	p	0.000	0.200	0.037		0.001	0.011	0.002	0.003	0.000	0.080	0.005	0.000	0.001	0.002	0.014	0.013
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RF5_OS	r	0.204	.406**	.549**	.475**	1	0.267	.316*	.375**	.621**	.643**	.472**	0.226	0.228	.321*	.385**	0.187
	p	0.160	0.004	0.000	0.001		0.063	0.027	0.008	0.000	0.000	0.001	0.119	0.116	0.024	0.006	0.197
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF1_OS	r	.602**	.320*	0.248	.361*	0.267	1	.637**	.812**	.734**	0.219	.500**	.350*	.506**	0.181	.605**	.350*
	p	0.000	0.025	0.086	0.011	0.063		0.000	0.000	0.000	0.130	0.000	0.014	0.000	0.212	0.000	0.014
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF2_OS	r	.532**	.509**	.357*	.434**	.316*	.637**	1	.653**	.457**	0.266	.604**	.323*	.429**	.653**	.565**	.658**
	p	0.000	0.000	0.012	0.002	0.027	0.000		0.000	0.001	0.064	0.000	0.024	0.002	0.000	0.000	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF3_OS	r	.442**	.368**	.403**	.421**	.375**	.812**	.653**	1	.717**	.301*	.569**	0.265	.338*	0.240	.700**	.290*
	p	0.001	0.009	0.004	0.003	0.008	0.000	0.000		0.000	0.035	0.000	0.065	0.018	0.097	0.000	0.043
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF4_OS	r	.403**	.282*	.393**	.554**	.621**	.734**	.457**	.717**	1	.410**	.447**	.425**	.353*	0.256	.642**	0.200
	p	0.004	0.050	0.005	0.000	0.000	0.000	0.001	0.000		0.003	0.001	0.002	0.013	0.076	0.000	0.169

	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LF5_OS	r	0.018	.485**	.381**	0.253	.643**	0.219	0.266	.301*	.410**	1	0.226	-0.194	-0.071	0.108	0.144	.407**
	p	0.905	0.000	0.007	0.080	0.000	0.130	0.064	0.035	0.003		0.118	0.183	0.630	0.461	0.324	0.004
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT1_OS	r	.454**	.529**	.541**	.395**	.472**	.500**	.604**	.569**	.447**	0.226	1	.293*	.548**	.525**	.467**	.392**
	p	0.001	0.000	0.000	0.005	0.001	0.000	0.000	0.000	0.001	0.118		0.041	0.000	0.000	0.001	0.005
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT2_OS	r	.693**	-0.230	0.028	.628**	0.226	.350*	.323*	0.265	.425**	-0.194	.293*	1	.569**	.415**	.469**	0.122
	p	0.000	0.112	0.850	0.000	0.119	0.014	0.024	0.065	0.002	0.183	0.041		0.000	0.003	0.001	0.402
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
RT3_OS	r	.739**	.360*	0.214	.460**	0.228	.506**	.429**	.338*	.353*	-0.071	.548**	.569**	1	.375**	.480**	.330*
	p	0.000	0.011	0.141	0.001	0.116	0.000	0.002	0.018	0.013	0.630	0.000	0.000		0.008	0.000	0.021
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT1_OS	r	.428**	.337*	.409**	.425**	.321*	0.181	.653**	0.240	0.256	0.108	.525**	.415**	.375**	1	.424**	.600**
	p	0.002	0.018	0.004	0.002	0.024	0.212	0.000	0.097	0.076	0.461	0.000	0.003	0.008		0.002	0.000
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT2_OS	r	.408**	0.263	.289*	.349*	.385**	.605**	.565**	.700**	.642**	0.144	.467**	.469**	.480**	.424**	1	.385**
	p	0.004	0.068	0.044	0.014	0.006	0.000	0.000	0.000	0.000	0.324	0.001	0.001	0.000	0.002		0.006
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49
LT3_OS	r	.300*	.429**	0.104	.354*	0.187	.350*	.658**	.290*	0.200	.407**	.392**	0.122	.330*	.600**	.385**	1
	p	0.037	0.002	0.476	0.013	0.197	0.014	0.000	0.043	0.169	0.004	0.005	0.402	0.021	0.000	0.006	
	N	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49	49

Mod/High Rumination (Inhibition Task)

		RF1_OS	RF2_OS	RF3_OS	RF4_OS	RF5_OS	LF1_OS	LF2_OS	LF3_OS	LF4_OS	LF5_OS	RT1_OS	RT2_OS	RT3_OS	LT1_OS	LT2_OS	LT3_OS
RF1_OS	r	1	.582**	.373*	0.114	0.095	.649**	0.178	-0.077	0.142	-0.006	0.118	0.041	.640**	0.107	0.290	.453**
	p		0.000	0.019	0.488	0.567	0.000	0.278	0.639	0.389	0.973	0.476	0.803	0.000	0.518	0.073	0.004
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF2_OS	r	.582**	1	.819**	.530**	.494**	.499**	.654**	.518**	.358*	0.232	.347*	0.310	.651**	0.275	.367*	.397*
	p	0.000		0.000	0.001	0.001	0.001	0.000	0.001	0.025	0.155	0.031	0.055	0.000	0.090	0.021	0.012

	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF3_OS	r	.373*	.819**	1	.743**	.635**	.341*	.586**	.481**	.375*	.477**	.365*	.401*	.362*	0.263	.349*	.338*
	p	0.019	0.000		0.000	0.000	0.033	0.000	0.002	0.019	0.002	0.022	0.012	0.024	0.105	0.030	0.036
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF4_OS	r	0.114	.530**	.743**	1	.747**	0.203	.517**	.382*	.628**	.590**	0.222	.331*	0.047	0.231	0.276	.415**
	p	0.488	0.001	0.000		0.000	0.215	0.001	0.016	0.000	0.000	0.175	0.039	0.776	0.158	0.089	0.009
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RF5_OS	r	0.095	.494**	.635**	.747**	1	0.068	.369*	.407*	.536**	.561**	0.260	.387*	0.077	-0.089	-0.012	0.141
	p	0.567	0.001	0.000	0.000		0.682	0.021	0.010	0.000	0.000	0.109	0.015	0.641	0.590	0.940	0.393
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF1_OS	r	.649**	.499**	.341*	0.203	0.068	1	.445**	0.218	.319*	0.147	0.149	0.054	.434**	0.160	.394*	.727**
	p	0.000	0.001	0.033	0.215	0.682		0.005	0.183	0.048	0.371	0.366	0.743	0.006	0.329	0.013	0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF2_OS	r	0.178	.654**	.586**	.517**	.369*	.445**	1	.773**	0.272	0.172	0.315	0.262	0.175	.360*	.505**	.549**
	p	0.278	0.000	0.000	0.001	0.021	0.005		0.000	0.094	0.296	0.051	0.106	0.287	0.025	0.001	0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF3_OS	r	-0.077	.518**	.481**	.382*	.407*	0.218	.773**	1	.317*	0.135	.355*	0.299	0.188	0.129	0.262	0.259
	p	0.639	0.001	0.002	0.016	0.010	0.183	0.000		0.050	0.413	0.027	0.065	0.251	0.434	0.108	0.111
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF4_OS	r	0.142	.358*	.375*	.628**	.536**	.319*	0.272	.317*	1	.326*	0.238	0.182	0.099	0.017	0.187	.404*
	p	0.389	0.025	0.019	0.000	0.000	0.048	0.094	0.050		0.043	0.144	0.267	0.547	0.917	0.254	0.011
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LF5_OS	r	-0.006	0.232	.477**	.590**	.561**	0.147	0.172	0.135	.326*	1	0.065	0.315	0.005	-0.098	-0.153	0.059
	p	0.973	0.155	0.002	0.000	0.000	0.371	0.296	0.413	0.043		0.692	0.051	0.975	0.552	0.353	0.720
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RT1_OS	r	0.118	.347*	.365*	0.222	0.260	0.149	0.315	.355*	0.238	0.065	1	.821**	0.178	0.267	.356*	0.112
	p	0.476	0.031	0.022	0.175	0.109	0.366	0.051	0.027	0.144	0.692		0.000	0.278	0.100	0.026	0.496
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RT2_OS	r	0.041	0.310	.401*	.331*	.387*	0.054	0.262	0.299	0.182	0.315	.821**	1	0.144	0.228	0.270	0.099

	p	0.803	0.055	0.012	0.039	0.015	0.743	0.106	0.065	0.267	0.051	0.000		0.381	0.163	0.096	0.548
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
RT3_OS	r	.640**	.651**	.362*	0.047	0.077	.434**	0.175	0.188	0.099	0.005	0.178	0.144	1	0.245	.319*	0.229
	p	0.000	0.000	0.024	0.776	0.641	0.006	0.287	0.251	0.547	0.975	0.278	0.381		0.133	0.048	0.160
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LT1_OS	r	0.107	0.275	0.263	0.231	-0.089	0.160	.360*	0.129	0.017	-0.098	0.267	0.228	0.245	1	.814**	0.295
	p	0.518	0.090	0.105	0.158	0.590	0.329	0.025	0.434	0.917	0.552	0.100	0.163	0.133		0.000	0.068
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LT2_OS	r	0.290	.367*	.349*	0.276	-0.012	.394*	.505**	0.262	0.187	-0.153	.356*	0.270	.319*	.814**	1	.635**
	p	0.073	0.021	0.030	0.089	0.940	0.013	0.001	0.108	0.254	0.353	0.026	0.096	0.048	0.000		0.000
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39
LT3_OS	r	.453**	.397*	.338*	.415**	0.141	.727**	.549**	0.259	.404*	0.059	0.112	0.099	0.229	0.295	.635**	1
	p	0.004	0.012	0.036	0.009	0.393	0.000	0.000	0.111	0.011	0.720	0.496	0.548	0.160	0.068	0.000	
	N	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39	39

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

a. RUMb_Binary Low_Mod+ = Mod/High

Waist Circumference Resting State Functional Connectivity Correlation Functional Connectivity Correlation

Waist_Cat_MF			RF1_ORS_Av	RF2_ORS_Av	RF3_ORS_Av	RF4_ORS_Av	RF5_ORS_Av	LF1_ORS_Av	LF2_ORS_Av	LF3_ORS_Av	LF4_ORS_Av	LF5_ORS_Av	RT1_ORS_Av	RT2_ORS_Av	RT3_ORS_Av	LT1_ORS_Av	LT2_ORS_Av	LT3_ORS_Av
Low	RF1_ORS_Av	r	1	.447**	.478**	.356*	0.237	.653**	0.106	0.056	0.034	-0.046	0.222	0.215	.436**	-0.160	-0.036	.325*
		p		0.003	0.002	0.022	0.136	0.000	0.509	0.727	0.831	0.773	0.163	0.178	0.004	0.316	0.825	0.038
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RF2_ORS_Av	r	.447**	1	.949**	.757**	.743**	0.199	-0.071	0.067	0.233	-0.123	.622**	.517**	.767**	-0.204	0.117	0.295
		p	0.003		0.000	0.000	0.000	0.213	0.657	0.679	0.144	0.443	0.000	0.001	0.000	0.200	0.467	0.061
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RF3_ORS_Av	r	.478**	.949**	1	.876**	.819**	0.157	-0.048	0.098	0.302	-0.016	.636**	.520**	.802**	-0.222	0.119	.382*

		p	0.002	0.000		0.000	0.000	0.328	0.764	0.543	0.055	0.922	0.000	0.000	0.000	0.162	0.457	0.014
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RF4_ORS_Av	r	.356*	.757**	.876**	1	.823**	0.083	-0.149	0.009	0.149	-0.062	.414**	.311*	.753**	-0.307	0.000	0.229
		p	0.022	0.000	0.000		0.000	0.604	0.352	0.954	0.353	0.699	0.007	0.047	0.000	0.051	0.999	0.150
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RF5_ORS_Av	r	0.237	.743**	.819**	.823**	1	0.035	-0.003	0.153	.363*	0.122	.493**	.397*	.745**	-0.145	0.120	.364*
		p	0.136	0.000	0.000	0.000		0.828	0.988	0.338	0.020	0.448	0.001	0.010	0.000	0.366	0.454	0.019
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LF1_ORS_Av	r	.653**	0.199	0.157	0.083	0.035	1	.336*	0.224	0.199	0.196	0.280	0.256	0.274	0.290	0.148	0.301
		p	0.000	0.213	0.328	0.604	0.828		0.032	0.158	0.213	0.219	0.076	0.107	0.083	0.066	0.355	0.056
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LF2_ORS_Av	r	0.106	-0.071	-0.048	-0.149	-0.003	.336*	1	.927**	.675**	.678**	0.292	.330*	0.057	.586**	.478**	.489**
		p	0.509	0.657	0.764	0.352	0.988	0.032		0.000	0.000	0.000	0.064	0.035	0.722	0.000	0.002	0.001
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LF3_ORS_Av	r	0.056	0.067	0.098	0.009	0.153	0.224	.927**	1	.703**	.614**	.311*	.315*	0.087	.563**	.490**	.421**
		p	0.727	0.679	0.543	0.954	0.338	0.158	0.000		0.000	0.000	0.048	0.045	0.589	0.000	0.001	0.006
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LF4_ORS_Av	r	0.034	0.233	0.302	0.149	.363*	0.199	.675**	.703**	1	.775**	.569**	.564**	0.256	.597**	.510**	.603**
		p	0.831	0.144	0.055	0.353	0.020	0.213	0.000	0.000		0.000	0.000	0.000	0.107	0.000	0.001	0.000
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LF5_ORS_Av	r	-0.046	-0.123	-0.016	-0.062	0.122	0.196	.678**	.614**	.775**	1	.521**	.599**	0.164	.650**	.537**	.674**
		p	0.773	0.443	0.922	0.699	0.448	0.219	0.000	0.000	0.000		0.000	0.000	0.307	0.000	0.000	0.000
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RT1_ORS_Av	r	0.222	.622**	.636**	.414**	.493**	0.280	0.292	.311*	.569**	.521**	1	.951**	.705**	.328*	.579**	.693**
		p	0.163	0.000	0.000	0.007	0.001	0.076	0.064	0.048	0.000	0.000		0.000	0.000	0.036	0.000	0.000
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RT2_ORS_Av	r	0.215	.517**	.520**	.311*	.397*	0.256	.330*	.315*	.564**	.599**	.951**	1	.651**	.365*	.622**	.737**
		p	0.178	0.001	0.000	0.047	0.010	0.107	0.035	0.045	0.000	0.000	0.000		0.000	0.019	0.000	0.000

		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	RT3_ORS_Av	r	.436**	.767**	.802**	.753**	.745**	0.274	0.057	0.087	0.256	0.164	.705**	.651**	1	-0.133	0.247	.565**
		p	0.004	0.000	0.000	0.000	0.000	0.083	0.722	0.589	0.107	0.307	0.000	0.000		0.407	0.119	0.000
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LT1_ORS_Av	r	-0.160	-0.204	-0.222	-0.307	-0.145	0.290	.586**	.563**	.597**	.650**	.328*	.365*	-0.133	1	.659**	.363*
		p	0.316	0.200	0.162	0.051	0.366	0.066	0.000	0.000	0.000	0.000	0.036	0.019	0.407		0.000	0.020
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LT2_ORS_Av	r	-0.036	0.117	0.119	0.000	0.120	0.148	.478**	.490**	.510**	.537**	.579**	.622**	0.247	.659**	1	.652**
		p	0.825	0.467	0.457	0.999	0.454	0.355	0.002	0.001	0.001	0.000	0.000	0.000	0.119	0.000		0.000
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
	LT3_ORS_Av	r	.325*	0.295	.382*	0.229	.364*	0.301	.489**	.421**	.603**	.674**	.693**	.737**	.565**	.363*	.652**	1
		p	0.038	0.061	0.014	0.150	0.019	0.056	0.001	0.006	0.000	0.000	0.000	0.000	0.000	0.020	0.000	
		N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
Increased	RF1_ORS_Av	r	1	0.209	0.116	.312*	0.030	-0.007	0.014	0.077	-0.013	0.098	0.029	.433**	0.223	0.220	0.144	0.177
		p		0.159	0.439	0.033	0.844	0.961	0.925	0.608	0.932	0.514	0.846	0.002	0.131	0.137	0.335	0.235
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RF2_ORS_Av	r	0.209	1	.890**	.439**	0.121	-0.130	.568**	.623**	.485**	0.225	.716**	.813**	.352*	.369*	.307*	0.144
		p	0.159		0.000	0.002	0.416	0.385	0.000	0.000	0.001	0.128	0.000	0.000	0.015	0.011	0.036	0.334
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RF3_ORS_Av	r	0.116	.890**	1	.417**	0.032	-0.098	.567**	.586**	.447**	0.179	.772**	.785**	0.166	.410**	.317*	0.091
		p	0.439	0.000		0.004	0.830	0.511	0.000	0.000	0.002	0.229	0.000	0.000	0.265	0.004	0.030	0.541
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RF4_ORS_Av	r	.312*	.439**	.417**	1	.309*	-0.021	.328*	.527**	.414**	0.131	.418**	.561**	.363*	0.181	0.215	0.182
		p	0.033	0.002	0.004		0.035	0.886	0.024	0.000	0.004	0.380	0.003	0.000	0.012	0.223	0.147	0.221
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RF5_ORS_Av	r	0.030	0.121	0.032	.309*	1	0.105	0.022	.365*	.467**	0.026	0.019	0.062	-0.151	-0.092	-0.140	-0.254
		p	0.844	0.416	0.830	0.035		0.484	0.883	0.012	0.001	0.863	0.901	0.678	0.311	0.538	0.347	0.084
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47

	LF1_ORS_Av	r	-0.007	-0.130	-0.098	-0.021	0.105	1	-0.140	-0.125	0.001	-0.002	-0.116	-0.103	-0.040	-0.107	-0.130	-0.066
		p	0.961	0.385	0.511	0.886	0.484		0.348	0.402	0.992	0.987	0.437	0.490	0.791	0.474	0.382	0.660
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LF2_ORS_Av	r	0.014	.568**	.567**	.328*	0.022	-0.140	1	.738**	.373**	-0.092	.330*	.418**	.405**	.607**	.474**	.293*
		p	0.925	0.000	0.000	0.024	0.883	0.348		0.000	0.010	0.540	0.024	0.003	0.005	0.000	0.001	0.046
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LF3_ORS_Av	r	0.077	.623**	.586**	.527**	.365*	-0.125	.738**	1	.814**	0.210	.394**	.578**	0.258	.397**	0.279	0.044
		p	0.608	0.000	0.000	0.000	0.012	0.402	0.000		0.000	0.156	0.006	0.000	0.080	0.006	0.058	0.771
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LF4_ORS_Av	r	-0.013	.485**	.447**	.414**	.467**	0.001	.373**	.814**	1	.476**	0.258	.442**	-0.162	0.110	-0.040	-.306*
		p	0.932	0.001	0.002	0.004	0.001	0.992	0.010	0.000		0.001	0.079	0.002	0.275	0.461	0.788	0.036
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LF5_ORS_Av	r	0.098	0.225	0.179	0.131	0.026	-0.002	-0.092	0.210	.476**	1	0.053	0.238	-0.027	-0.119	-0.156	-0.163
		p	0.514	0.128	0.229	0.380	0.863	0.987	0.540	0.156	0.001		0.722	0.107	0.858	0.427	0.296	0.273
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RT1_ORS_Av	r	0.029	.716**	.772**	.418**	0.019	-0.116	.330*	.394**	0.258	0.053	1	.828**	0.230	.397**	.511**	0.243
		p	0.846	0.000	0.000	0.003	0.901	0.437	0.024	0.006	0.079	0.722		0.000	0.119	0.006	0.000	0.100
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RT2_ORS_Av	r	.433**	.813**	.785**	.561**	0.062	-0.103	.418**	.578**	.442**	0.238	.828**	1	.362*	.453**	.453**	0.225
		p	0.002	0.000	0.000	0.000	0.678	0.490	0.003	0.000	0.002	0.107	0.000		0.012	0.001	0.001	0.128
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	RT3_ORS_Av	r	0.223	.352*	0.166	.363*	-0.151	-0.040	.405**	0.258	-0.162	-0.027	0.230	.362*	1	.312*	.462**	.620**
		p	0.131	0.015	0.265	0.012	0.311	0.791	0.005	0.080	0.275	0.858	0.119	0.012		0.033	0.001	0.000
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LT1_ORS_Av	r	0.220	.369*	.410**	0.181	-0.092	-0.107	.607**	.397**	0.110	-0.119	.397**	.453**	.312*	1	.879**	0.188
		p	0.137	0.011	0.004	0.223	0.538	0.474	0.000	0.006	0.461	0.427	0.006	0.001	0.033		0.000	0.205
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47

	LT2_ORS_Av	r	0.144	.307*	.317*	0.215	-0.140	-0.130	.474**	0.279	-0.040	-0.156	.511**	.453**	.462**	.879**	1	.433**
		p	0.335	0.036	0.030	0.147	0.347	0.382	0.001	0.058	0.788	0.296	0.000	0.001	0.001	0.000		0.002
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
	LT3_ORS_Av	r	0.177	0.144	0.091	0.182	-0.254	-0.066	.293*	0.044	-.306*	-0.163	0.243	0.225	.620**	0.188	.433**	1
		p	0.235	0.334	0.541	0.221	0.084	0.660	0.046	0.771	0.036	0.273	0.100	0.128	0.000	0.205	0.002	
		N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47

**. Correlation is significant at the 0.01 level (2-tailed
*. Correlation is significant at the 0.05 level (2-tailed
a. Cannot be computed because at least one of the variables is constant

Waist Circumference (Continuous Performance Task Attention) Functional Connectivity Correlation

Low		RF1_OC	RF2_OC	RF3_OC	RF4_OC	RF5_OC	LF1_OC	LF2_OC	LF3_OC	LF4_OC	LF5_OC	RT1_OC	RT2_OC	RT3_OC	LT1_OC	LT2_OC	LT3_OC
RF1_OC	r	1	.562**	0.284	.490**	.392*	.916**	.754**	.653**	.514**	0.013	0.108	.573**	.671**	0.258	.342*	.890**
	p		0.000	0.072	0.001	0.011	0.000	0.000	0.000	0.001	0.935	0.503	0.000	0.000	0.103	0.028	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF2_OC	r	.562**	1	.844**	.378*	.324*	.616**	.613**	.484**	.363*	0.084	.764**	.684**	.694**	0.154	0.291	.623**
	p	0.000		0.000	0.015	0.039	0.000	0.000	0.001	0.020	0.601	0.000	0.000	0.000	0.337	0.065	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF3_OC	r	0.284	.844**	1	.331*	0.139	.329*	0.284	0.300	0.175	-0.025	.910**	.685**	.518**	0.118	0.089	.338*
	p	0.072	0.000		0.034	0.388	0.036	0.072	0.057	0.273	0.879	0.000	0.000	0.001	0.461	0.579	0.031
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF4_OC	r	.490**	.378*	.331*	1	.568**	.464**	.368*	.359*	.422**	0.259	0.111	.620**	.447**	0.080	0.138	.430**
	p	0.001	0.015	0.034		0.000	0.002	0.018	0.021	0.006	0.102	0.488	0.000	0.003	0.619	0.390	0.005

	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF5_OC	r	.392*	.324*	0.139	.568**	1	.443**	.399**	.473**	.484**	0.292	-0.015	0.269	0.278	0.018	0.100	.390*
	p	0.011	0.039	0.388	0.000		0.004	0.010	0.002	0.001	0.064	0.927	0.089	0.078	0.912	0.535	0.012
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF1_OC	r	.916**	.616**	.329*	.464**	.443**	1	.767**	.631**	.621**	0.101	0.182	.525**	.663**	0.308	0.291	.915**
	p	0.000	0.000	0.036	0.002	0.004		0.000	0.000	0.000	0.529	0.255	0.000	0.000	0.050	0.065	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF2_OC	r	.754**	.613**	0.284	.368*	.399**	.767**	1	.795**	.396*	0.088	0.187	.430**	.671**	0.251	.592**	.781**
	p	0.000	0.000	0.072	0.018	0.010	0.000		0.000	0.010	0.584	0.242	0.005	0.000	0.114	0.000	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF3_OC	r	.653**	.484**	0.300	.359*	.473**	.631**	.795**	1	.533**	0.119	0.093	.413**	.622**	.408**	0.240	.676**
	p	0.000	0.001	0.057	0.021	0.002	0.000	0.000		0.000	0.458	0.565	0.007	0.000	0.008	0.130	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF4_OC	r	.514**	.363*	0.175	.422**	.484**	.621**	.396*	.533**	1	.568**	0.059	.429**	.375*	0.245	-0.244	.597**
	p	0.001	0.020	0.273	0.006	0.001	0.000	0.010	0.000		0.000	0.713	0.005	0.016	0.122	0.125	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF5_OC	r	0.013	0.084	-0.025	0.259	0.292	0.101	0.088	0.119	.568**	1	-0.019	0.153	-0.026	-0.157	-0.047	0.053
	p	0.935	0.601	0.879	0.102	0.064	0.529	0.584	0.458	0.000		0.907	0.339	0.870	0.326	0.769	0.744
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT1_OC	r	0.108	.764**	.910**	0.111	-0.015	0.182	0.187	0.093	0.059	-0.019	1	.549**	.448**	-0.049	0.118	0.173
	p	0.503	0.000	0.000	0.488	0.927	0.255	0.242	0.565	0.713	0.907		0.000	0.003	0.759	0.462	0.281

	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT2_OC	r	.573**	.684**	.685**	.620**	0.269	.525**	.430**	.413**	.429**	0.153	.549**	1	.581**	0.171	0.066	.540**
	p	0.000	0.000	0.000	0.000	0.089	0.000	0.005	0.007	0.005	0.339	0.000		0.000	0.285	0.682	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT3_OC	r	.671**	.694**	.518**	.447**	0.278	.663**	.671**	.622**	.375*	-0.026	.448**	.581**	1	.325*	.310*	.606**
	p	0.000	0.000	0.001	0.003	0.078	0.000	0.000	0.000	0.016	0.870	0.003	0.000		0.038	0.048	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LT1_OC	r	0.258	0.154	0.118	0.080	0.018	0.308	0.251	.408**	0.245	-0.157	-0.049	0.171	.325*	1	-0.224	.344*
	p	0.103	0.337	0.461	0.619	0.912	0.050	0.114	0.008	0.122	0.326	0.759	0.285	0.038		0.159	0.028
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LT2_OC	r	.342*	0.291	0.089	0.138	0.100	0.291	.592**	0.240	-0.244	-0.047	0.118	0.066	.310*	-0.224	1	0.298
	p	0.028	0.065	0.579	0.390	0.535	0.065	0.000	0.130	0.125	0.769	0.462	0.682	0.048	0.159		0.058
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LT3_OC	r	.890**	.623**	.338*	.430**	.390*	.915**	.781**	.676**	.597**	0.053	0.173	.540**	.606**	.344*	0.298	1
	p	0.000	0.000	0.031	0.005	0.012	0.000	0.000	0.000	0.000	0.744	0.281	0.000	0.000	0.028	0.058	
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
Increased																	
RF1_OC	r	1	.314*	0.046	-0.073	0.115	.374*	0.227	0.008	-0.128	0.068	0.169	0.024	.365*	-0.075	-0.105	.322*
	p		0.034	0.760	0.628	0.445	0.011	0.129	0.957	0.398	0.653	0.262	0.874	0.013	0.620	0.487	0.029
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RF2_OC	r	.314*	1	.605**	0.145	0.141	-0.046	0.235	-0.014	.295*	0.017	.443**	0.076	-0.017	-0.174	-0.085	0.032

	p	0.034		0.000	0.338	0.349	0.759	0.115	0.929	0.046	0.910	0.002	0.614	0.909	0.248	0.576	0.833
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RF3_OC	r	0.046	.605**	1	.802**	0.173	-0.081	0.233	0.107	.502**	0.108	.397**	.506**	0.043	-0.118	-0.024	0.246
	p	0.760	0.000		0.000	0.251	0.592	0.120	0.481	0.000	0.474	0.006	0.000	0.778	0.436	0.872	0.099
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RF4_OC	r	-0.073	0.145	.802**	1	0.225	0.055	.302*	0.252	.438**	0.048	0.160	.633**	0.232	0.024	0.025	.462**
	p	0.628	0.338	0.000		0.132	0.718	0.041	0.091	0.002	0.750	0.287	0.000	0.121	0.877	0.869	0.001
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RF5_OC	r	0.115	0.141	0.173	0.225	1	0.034	.301*	0.271	0.040	-0.085	0.106	0.218	0.073	0.033	0.039	.385**
	p	0.445	0.349	0.251	0.132		0.825	0.042	0.068	0.790	0.575	0.484	0.146	0.628	0.829	0.799	0.008
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LF1_OC	r	.374*	-0.046	-0.081	0.055	0.034	1	0.283	.460**	-0.120	0.018	-0.026	0.110	0.019	.518**	.557**	.324*
	p	0.011	0.759	0.592	0.718	0.825		0.057	0.001	0.428	0.906	0.862	0.467	0.899	0.000	0.000	0.028
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LF2_OC	r	0.227	0.235	0.233	.302*	.301*	0.283	1	.769**	0.165	0.033	0.266	.314*	0.064	-0.114	-0.163	.597**
	p	0.129	0.115	0.120	0.041	0.042	0.057		0.000	0.273	0.828	0.074	0.033	0.671	0.449	0.278	0.000
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LF3_OC	r	0.008	-0.014	0.107	0.252	0.271	.460**	.769**	1	0.060	-0.087	0.129	0.253	-0.169	0.188	0.209	.346*
	p	0.957	0.929	0.481	0.091	0.068	0.001	0.000		0.690	0.563	0.392	0.090	0.263	0.212	0.164	0.019
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LF4_OC	r	-0.128	.295*	.502**	.438**	0.040	-0.120	0.165	0.060	1	-0.085	.314*	0.188	-0.014	-0.071	0.041	0.172

	p	0.398	0.046	0.000	0.002	0.790	0.428	0.273	0.690		0.573	0.034	0.211	0.924	0.641	0.785	0.254
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LF5_OC	r	0.068	0.017	0.108	0.048	-0.085	0.018	0.033	-0.087	-0.085	1	0.243	0.154	-0.014	-0.041	0.063	0.268
	p	0.653	0.910	0.474	0.750	0.575	0.906	0.828	0.563	0.573		0.103	0.307	0.924	0.785	0.679	0.071
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RT1_OC	r	0.169	.443**	.397**	0.160	0.106	-0.026	0.266	0.129	.314*	0.243	1	.414**	0.036	0.132	-0.032	.440**
	p	0.262	0.002	0.006	0.287	0.484	0.862	0.074	0.392	0.034	0.103		0.004	0.810	0.380	0.831	0.002
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RT2_OC	r	0.024	0.076	.506**	.633**	0.218	0.110	.314*	0.253	0.188	0.154	.414**	1	0.222	0.040	-0.019	.524**
	p	0.874	0.614	0.000	0.000	0.146	0.467	0.033	0.090	0.211	0.307	0.004		0.138	0.791	0.901	0.000
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
RT3_OC	r	.365*	-0.017	0.043	0.232	0.073	0.019	0.064	-0.169	-0.014	-0.014	0.036	0.222	1	0.267	-0.129	.434**
	p	0.013	0.909	0.778	0.121	0.628	0.899	0.671	0.263	0.924	0.924	0.810	0.138		0.073	0.394	0.003
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LT1_OC	r	-0.075	-0.174	-0.118	0.024	0.033	.518**	-0.114	0.188	-0.071	-0.041	0.132	0.040	0.267	1	.701**	0.223
	p	0.620	0.248	0.436	0.877	0.829	0.000	0.449	0.212	0.641	0.785	0.380	0.791	0.073		0.000	0.136
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LT2_OC	r	-0.105	-0.085	-0.024	0.025	0.039	.557**	-0.163	0.209	0.041	0.063	-0.032	-0.019	-0.129	.701**	1	-0.073
	p	0.487	0.576	0.872	0.869	0.799	0.000	0.278	0.164	0.785	0.679	0.831	0.901	0.394	0.000		0.628
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46
LT3_OC	r	.322*	0.032	0.246	.462**	.385**	.324*	.597**	.346*	0.172	0.268	.440**	.524**	.434**	0.223	-0.073	1

	p	0.029	0.833	0.099	0.001	0.008	0.028	0.000	0.019	0.254	0.071	0.002	0.000	0.003	0.136	0.628	
	N	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46	46

Waist Circumference Inhibition task Functional Connectivity Correlation

Low		RF1_OS_	RF2_OS_	RF3_OS_	RF4_OS_	RF5_OS_	LF1_OS_	LF2_OS_	LF3_OS_	LF4_OS_	LF5_OS_	RT1_OS_	RT2_OS_	RT3_OS_	LT1_OS_	LT2_OS_	LT3_OS_
RF1_OS_	r	1	-0.226	-0.108	.361*	0.025	.451**	0.044	0.012	0.289	-0.152	0.197	.733**	.758**	0.098	0.244	0.131
	p		0.155	0.501	0.020	0.879	0.003	0.786	0.940	0.067	0.344	0.216	0.000	0.000	0.541	0.125	0.416
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF2_OS_	r	-0.226	1	.687**	0.128	.460**	0.202	.443**	0.260	.318*	.605**	0.244	-0.296	-0.034	0.191	0.106	.365*
	p	0.155		0.000	0.426	0.002	0.204	0.004	0.100	0.043	0.000	0.124	0.060	0.832	0.231	0.508	0.019
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF3_OS_	r	-0.108	.687**	1	.354*	.630**	0.264	.406**	0.305	.585**	.413**	.391*	0.014	0.038	0.256	0.223	0.264
	p	0.501	0.000		0.023	0.000	0.095	0.009	0.052	0.000	0.007	0.012	0.933	0.815	0.106	0.162	0.095
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF4_OS_	r	.361*	0.128	.354*	1	.620**	0.267	.338*	0.302	.647**	0.263	0.160	.491**	0.200	0.234	0.196	0.291
	p	0.020	0.426	0.023		0.000	0.091	0.031	0.055	0.000	0.096	0.316	0.001	0.209	0.141	0.219	0.065
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RF5_OS_	r	0.025	.460**	.630**	.620**	1	0.202	0.299	0.233	.630**	.666**	0.226	0.101	-0.049	0.181	0.034	0.281
	p	0.879	0.002	0.000	0.000		0.206	0.057	0.143	0.000	0.000	0.156	0.529	0.760	0.257	0.834	0.075
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF1_OS_	r	.451**	0.202	0.264	0.267	0.202	1	.437**	.567**	.624**	0.164	.363*	0.289	.448**	0.050	.625**	.441**
	p	0.003	0.204	0.095	0.091	0.206		0.004	0.000	0.000	0.306	0.020	0.066	0.003	0.758	0.000	0.004
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41

LF2_OS_	r	0.044	.443**	.406**	.338*	0.299	.437**	1	.705**	.355*	0.210	.573**	0.271	0.051	.654**	.574**	.575**
	p	0.786	0.004	0.009	0.031	0.057	0.004		0.000	0.023	0.188	0.000	0.087	0.752	0.000	0.000	0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF3_OS_	r	0.012	0.260	0.305	0.302	0.233	.567**	.705**	1	.518**	0.149	.452**	0.220	0.107	0.225	.651**	0.230
	p	0.940	0.100	0.052	0.055	0.143	0.000	0.000		0.001	0.352	0.003	0.167	0.505	0.156	0.000	0.149
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF4_OS_	r	0.289	.318*	.585**	.647**	.630**	.624**	.355*	.518**	1	.396*	0.244	0.246	0.250	0.038	.459**	.329*
	p	0.067	0.043	0.000	0.000	0.000	0.000	0.023	0.001		0.010	0.124	0.121	0.115	0.814	0.003	0.035
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LF5_OS_	r	-0.152	.605**	.413**	0.263	.666**	0.164	0.210	0.149	.396*	1	-0.060	-0.301	-0.194	0.014	-0.125	0.250
	p	0.344	0.000	0.007	0.096	0.000	0.306	0.188	0.352	0.010		0.709	0.056	0.225	0.933	0.437	0.114
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT1_OS_	r	0.197	0.244	.391*	0.160	0.226	.363*	.573**	.452**	0.244	-0.060	1	.550**	0.249	.504**	.432**	0.251
	p	0.216	0.124	0.012	0.316	0.156	0.020	0.000	0.003	0.124	0.709		0.000	0.117	0.001	0.005	0.114
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT2_OS_	r	.733**	-0.296	0.014	.491**	0.101	0.289	0.271	0.220	0.246	-0.301	.550**	1	.587**	.323*	0.303	0.052
	p	0.000	0.060	0.933	0.001	0.529	0.066	0.087	0.167	0.121	0.056	0.000		0.000	0.039	0.055	0.747
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
RT3_OS_	r	.758**	-0.034	0.038	0.200	-0.049	.448**	0.051	0.107	0.250	-0.194	0.249	.587**	1	0.184	0.266	0.076
	p	0.000	0.832	0.815	0.209	0.760	0.003	0.752	0.505	0.115	0.225	0.117	0.000		0.250	0.092	0.635
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41

LT1_OS_	r	0.098	0.191	0.256	0.234	0.181	0.050	.654**	0.225	0.038	0.014	.504**	.323*	0.184	1	.328*	.414**
	p	0.541	0.231	0.106	0.141	0.257	0.758	0.000	0.156	0.814	0.933	0.001	0.039	0.250		0.036	0.007
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LT2_OS_	r	0.244	0.106	0.223	0.196	0.034	.625**	.574**	.651**	.459**	-0.125	.432**	0.303	0.266	.328*	1	.645**
	p	0.125	0.508	0.162	0.219	0.834	0.000	0.000	0.000	0.003	0.437	0.005	0.055	0.092	0.036		0.000
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
LT3_OS_	r	0.131	.365*	0.264	0.291	0.281	.441**	.575**	0.230	.329*	0.250	0.251	0.052	0.076	.414**	.645**	1
	p	0.416	0.019	0.095	0.065	0.075	0.004	0.000	0.149	0.035	0.114	0.114	0.747	0.635	0.007	0.000	
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41	41
Increased																	
RF1_OS_	r	1	.781**	.588**	.415**	0.269	.793**	.646**	.463**	.317*	0.138	.376**	0.171	.642**	0.251	.376**	.639**
	p		0.000	0.000	0.004	0.067	0.000	0.000	0.001	0.030	0.356	0.009	0.252	0.000	0.089	0.009	0.000
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RF2_OS_	r	.781**	1	.776**	.493**	.456**	.584**	.701**	.635**	.350*	0.185	.570**	0.232	.780**	.289*	.423**	.468**
	p	0.000		0.000	0.000	0.001	0.000	0.000	0.000	0.016	0.212	0.000	0.117	0.000	0.049	0.003	0.001
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RF3_OS_	r	.588**	.776**	1	.649**	.581**	.353*	.581**	.608**	.292*	.464**	.495**	.334*	.408**	.295*	.368*	0.254
	p	0.000	0.000		0.000	0.000	0.015	0.000	0.000	0.047	0.001	0.000	0.022	0.004	0.044	0.011	0.085
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RF4_OS_	r	.415**	.493**	.649**	1	.659**	.299*	.612**	.601**	.556**	.520**	.493**	.625**	.443**	0.284	.373**	.456**
	p	0.004	0.000	0.000		0.000	0.041	0.000	0.000	0.000	0.000	0.000	0.000	0.002	0.053	0.010	0.001

	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RF5_OS_	r	0.269	.456**	.581**	.659**	1	0.203	.446**	.589**	.598**	.609**	.473**	.494**	.359*	0.024	0.250	0.131
	p	0.067	0.001	0.000	0.000		0.170	0.002	0.000	0.000	0.000	0.001	0.000	0.013	0.873	0.090	0.380
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LF1_OS_	r	.793**	.584**	.353*	.299*	0.203	1	.655**	.493**	.446**	0.181	.311*	0.152	.505**	0.204	.319*	.737**
	p	0.000	0.000	0.015	0.041	0.170		0.000	0.000	0.002	0.223	0.033	0.308	0.000	0.169	0.029	0.000
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LF2_OS_	r	.646**	.701**	.581**	.612**	.446**	.655**	1	.750**	.443**	0.181	.369*	.350*	.583**	.314*	.474**	.612**
	p	0.000	0.000	0.000	0.000	0.002	0.000		0.000	0.002	0.224	0.011	0.016	0.000	0.032	0.001	0.000
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LF3_OS_	r	.463**	.635**	.608**	.601**	.589**	.493**	.750**	1	.628**	.332*	.517**	.390**	.518**	0.114	0.218	.390**
	p	0.001	0.000	0.000	0.000	0.000	0.000	0.000		0.000	0.023	0.000	0.007	0.000	0.445	0.141	0.007
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LF4_OS_	r	.317*	.350*	.292*	.556**	.598**	.446**	.443**	.628**	1	.330*	.491**	.510**	.352*	0.114	0.286	.328*
	p	0.030	0.016	0.047	0.000	0.000	0.002	0.002	0.000		0.023	0.000	0.000	0.015	0.444	0.051	0.025
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LF5_OS_	r	0.138	0.185	.464**	.520**	.609**	0.181	0.181	.332*	.330*	1	.336*	.447**	0.083	-0.070	0.033	0.120
	p	0.356	0.212	0.001	0.000	0.000	0.223	0.224	0.023	0.023		0.021	0.002	0.581	0.639	0.824	0.423
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RT1_OS_	r	.376**	.570**	.495**	.493**	.473**	.311*	.369*	.517**	.491**	.336*	1	.484**	.498**	0.276	.367*	0.262
	p	0.009	0.000	0.000	0.000	0.001	0.033	0.011	0.000	0.000	0.021		0.001	0.000	0.060	0.011	0.075

	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RT2_OS_	r	0.171	0.232	.334*	.625**	.494**	0.152	.350*	.390**	.510**	.447**	.484**	1	.305*	0.276	.401**	0.249
	p	0.252	0.117	0.022	0.000	0.000	0.308	0.016	0.007	0.000	0.002	0.001		0.037	0.060	0.005	0.092
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
RT3_OS_	r	.642**	.780**	.408**	.443**	.359*	.505**	.583**	.518**	.352*	0.083	.498**	.305*	1	0.266	.425**	.521**
	p	0.000	0.000	0.004	0.002	0.013	0.000	0.000	0.000	0.015	0.581	0.000	0.037		0.071	0.003	0.000
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LT1_OS_	r	0.251	.289*	.295*	0.284	0.024	0.204	.314*	0.114	0.114	-0.070	0.276	0.276	0.266	1	.885**	.361*
	p	0.089	0.049	0.044	0.053	0.873	0.169	0.032	0.445	0.444	0.639	0.060	0.060	0.071		0.000	0.013
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LT2_OS_	r	.376**	.423**	.368*	.373**	0.250	.319*	.474**	0.218	0.286	0.033	.367*	.401**	.425**	.885**	1	.464**
	p	0.009	0.003	0.011	0.010	0.090	0.029	0.001	0.141	0.051	0.824	0.011	0.005	0.003	0.000		0.001
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
LT3_OS_	r	.639**	.468**	0.254	.456**	0.131	.737**	.612**	.390**	.328*	0.120	0.262	0.249	.521**	.361*	.464**	1
	p	0.000	0.001	0.085	0.001	0.380	0.000	0.000	0.007	0.025	0.423	0.075	0.092	0.000	0.013	0.001	
	N	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47

Appendix Q Negative BOLD response/fNIRS Signal

haemodynamic activity during each task was negative compared to baseline in all regions indicating that metabolic activity was reducing overall. A small negative dip in OHb followed by a rise and larger fall is a normal pattern of the BOLD response. The initial dip occurs as cells begin to use more oxygen than is supplied, this instigates the neuro-vascular coupling effect to increase oxygenation which is used by the cells (and converted to deoxygenated HHb) creating a fall. Each BOLD response therefore includes both a rise and a fall in OHb levels. Interpretation of the findings in the current study therefore focuses on the average magnitude of change from baseline rather than the direction of change.

Interpretation of a negative signal compared to baseline can indicate increased metabolic demand in nearby brain regions which are thus drawing blood flow away from less active areas. A negative signal can also indicate that the brain is activly inhibiting neural activity a given regions (Wade, 2002).

The action of the BOLD response has been investigated in fMRI studies (Sten et al., 2017). During demanding cognitive tasks the authors found that DMN regions (including the media prefrontal cortices) showed a negative BOLD response. Investigations modelling the effects of glutamate and GABA on calcium ions were able to account for patterns of positive and negative BOLD signal (compared to baseline) during visuo-spatial and working memory tasks; DMN activity was likely inhibited by increased GABA release (creating a negative BOLD response) whereas frontal network activity was increased by glutamate creating a positive BOLD response in this area.

Appendix R Areas for Future Study: Vagal Nerve Tone and Heart Rate Variability

Payne et al. (2015) note that Vagus nerve tone can be affected by both stresses and traumatic circumstances, and moreover that the system can become 'tuned' to stressful circumstances resulting in a person becoming stuck in a 'just in case' hypervigilant state rather than returning to baseline. As part of the parasympathetic nervous system the Vagus nerve runs from the brain to most bodily organs and among other things controls heart rate.

Meta analyses of neurocognitive studies have found a link between Heart Rate Variability (HRV; controlled by Vagus nerve tone) and cognitive impairment (including inhibitory control). This means that autonomic control of the heart (lower resting state HRV) simultaneously affects a person's neurocognitive ability to respond quickly and flexibly to environmental demands (Forte et al., 2019) and therefore produce effective goal-directed tasks/behaviour. As the sympathetic and parasympathetic branches of the autonomic nervous system branch into different cortical brain areas, different cognitive tasks (spatial versus verbal) are more affected by high and low levels of HRV, but in general, research indicates that low HRV is related to worse cognitive function. As well as a link with inhibitory control, high central adiposity is linked to low HRV (Banerjee et al., 2022; Yadav et al., 2017) hence the hypothesis of low HRV fits with the observed relationship between mental health and adiposity and certainly warrants further investigation.

Heart Rate Variability (HRV) is linked to a range of co-morbid health conditions including depression, rumination, decreased frontal activity and reduced inhibitory control (Dell'Acqua et al., 2021). HRV could therefore be a promising

mechanism to explain the link between cognition and health observed in this thesis. Relatedly, the link between heart rate reactivity and cognitive function has also been studied extensively in a body of work by Ginty et al., who, in their studies of stress and cognition, found there was a reliable subgroup of individuals who showed a blunted heart rate response and greater cognitive problems (Ginty et al., 2022) which could be relevant to HRV.

HRV could be a promising physiological measure of physical and mental health vulnerability. This is particularly important as HRV can be monitored with current technology and can be improved with exercise (Routledge et al., 2010; Singh et al., 2018) and caloric restriction (Nicoll & Henein, 2018). Concomitantly, low HRV is observed in relation to obesity, metabolic disorders, weigh gain, visceral fat, and emotional regulation during loss of control eating (Nicoll & Henein, 2018; Spitoni et al., 2017; Struven et al., 2021). Brooding rumination and HRV therefore promising variables to examine in future work related to negative affect, cognition, obesity and cardiometabolic health-risks.