Developing caries inequalities risk prediction tools for children under the age of six

By

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"We may define a cause to be an object followed by another ... where, if the first object had not been, the second never had existed."

David Hume (1748, Section VII)

ABSTRACT

Introduction:

Dental caries in deciduous teeth is the 10th-most prevalent condition, affecting 621 million children worldwide (Kassebaum et al., 2015). Although overall prevalence of Early Childhood Caries (ECC) is declining, but there is no improvement in the unequal distribution of caries across social gradients. It is therefore important to investigate and demonstrate the relationships between the factors responsible for caries inequalities in children under the age of six.

Methods

This thesis used systematic review methods and the Cochrane-validated tool (PROGRESS-PLUS) to explore factors implicated in ECC inequalities. Existing conceptual ECC frameworks were then evaluated (mapped) against factors identified from the review. The relationship between identified factors were established using Directed Acyclic Graphs and path analyses to create new conceptual models, and their predictability assessed using traditional and machine learning statistical techniques.

Results

Sixty-seven publications were eligible for this review and the result showed that there are 24 ECC-related risk factors. The mapping of existing caries frameworks also revealed that none of the current caries conceptual model had more than 48% relevant Social Determinants of Health (SDH) in their frameworks. Two separate ECC conceptual

frameworks (temporal and hierarchical) were developed based on systematic review evidence, and the predictability of the models also showed an Area under Receiver Curve (AUC) of 76% and 74% for the child-level and area-level prevalence prediction respectively.

Conclusion

This thesis found that existing conceptual frameworks lacked in-depth consideration of the full wider determinants that are responsible for health inequalities, which may be the cause of the current low ECC prevalence, but widening or stagnant caries inequalities pattern seen globally. The model developed in this thesis included more SDH content (64%) than any existing models and therefore increases the chances that preventive measures, built on the new SDH models, will be able to address both prevalence and inequalities. This model also demonstrated good predictive capabilities.

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ABBREVIATIONS

AIC:	Akaike Information Criteria
AUC:	Area under Receiver Curve
CDC:	Centers for Disease Control and Prevention
CDHS:	Child Dental Health Survey
CI:	Confidence Interval
DAG:	Directed Acyclic Graph
ECC:	Early Childhood Caries
HDI:	Human Development Index
HR:	Hazard Ratio
ICC:	Intra-class Correlation Coefficient
ICDAS:	International Caries Detection and Assessment System
IMD:	Index of Multiple Deprivation
IRR:	Incidence Rate Ratio
ISCO:	International Standard Classification of Occupations
JBI:	Joanna Briggs Institute
LA:	Local Authority
MAE:	Mean Absolute Error
MICE:	Multivariate Imputation by Chained Equations
NHS:	National Health Service (United Kingdom)
NICE:	The National Institute for Health and Care Excellence

NOS: Newcastle Ottawa Scale

OHS: Oral Health Survey

ONS: Office for National Statistics

OR: Odds Ratio

PCA: Principal Component Analysis

PHE: Public Health England

PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses

PROGRESS: Place of residence, Race, Occupation, Gender, Religion, Education, SES, and Social capital

RMSE: Root Mean Squared Error

ROB: Risk of Bias

ROC: Receiver operating characteristic

SDH: Social Determinants of Health

SEM: Structural Equation Modelling

SES: Socioeconomic Status

SVM: Support Vector Machine

UK: United Kingdom

US(A): United States (of America)

VIF: Variable Inflation Factor

WHO: World Health Organisation

GLOSSARY OF TERMS

Health Inequalities

"Health inequalities are the preventable, unfair and unjust differences in health status between groups, populations or individuals that arise from the unequal distribution of social, environmental and economic conditions within societies, which determine the risk of people getting ill, their ability to prevent sickness, or opportunities to take action and access treatment when ill health occurs." (Public Health England Definition)

Social Determinants of Health

Defined by WHO as "the conditions in which people are born, grow, live, work and age". Their definition went even further by emphasising that "these circumstances are shaped by the distribution of money, power and resources at global, national and local levels" (WHO definition).

Neighbourhood Context

"Refers to a local area which is defined in some way physically or by people's perceptions of what constitutes their local area" (WHO definition).

Caries Prevalence

Defined as "the percentage of the population affected by dental caries following the WHO criteria (1997)" (WHO definition).

Health Policy

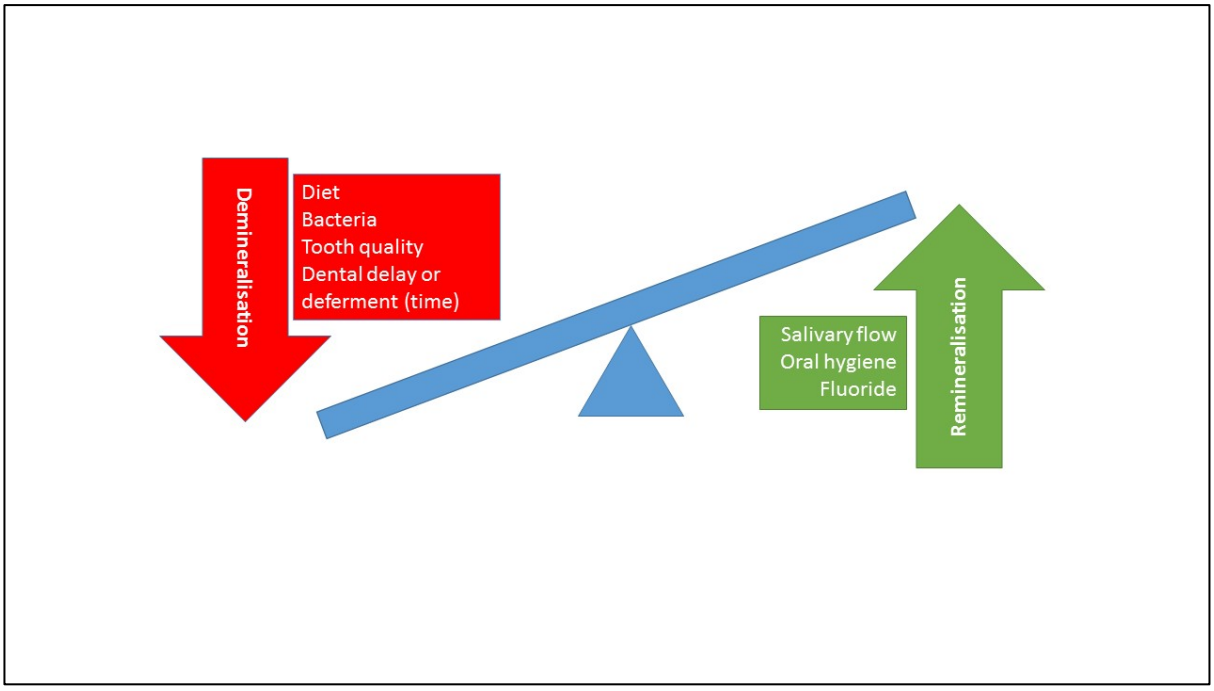
"Health policy refers to decisions, plans, and actions that are undertaken to achieve specific health care goals within a society" (WHO definition).

CHAPTER ONE: INTRODUCTION

1.1 Background of study

In epidemiology, early childhood caries (ECC) is defined as “the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months of age or younger” (American Academy of Pediatric Dentistry, 2008). Although caries is regarded as the most preventable childhood disease worldwide, it still poses a major Public Health concern in children globally (Selwitz et al., 2007, Smith and Riedford, 2013). ECC is mainly caused by three essential factors, which are: dental plaque (which can contain acid-loving bacteria), fermentable carbohydrates from diet (which is able to diffuse into the teeth and dissolve their mineral contents), and a susceptible tooth (Featherstone, 2008), which are collectively known as the Keyes Triad (Eriksen and Bjertness, 1991). It is also understood that every tooth goes through a never-ending process of demineralisation (destruction) and remineralisation (repair), known as the Levine “see-saw” mechanism, and tooth decay occurs when the rate of destruction surpasses that of repair (Levine, 1977) (Figure 1)

Figure 1: Factors influencing remineralisation and demineralisation of teeth modified from Levine's see-saw ionic theory (Levine, 1977)



The implication of several remineralisation and demineralisation processes in deciduous teeth, which are known to have thinner enamel, is that it makes children's teeth to become highly susceptible to caries, because of the little resistance against bacteria (Vos et al., 2012, Selwitz et al., 2007, Lynch, 2013). Children who experienced caries in their primary teeth are more likely to develop further caries in their permanent teeth (Çolak et al., 2013, Leroy et al., 2005). Previous history of caries is also the most accurate single predictor of tooth decay in all age groups, and once tooth decay in permanent teeth is established, its consequences last for a lifetime (Mejare et al., 2014, Schwendicke, 2013, World Health Organisation, 2003).

1.2 The size of the problem in population studies

The two main public health challenges of ECC are related to its prevalence and inequalities within populations (Levin et al., 2009, Aida et al., 2008). Prevalence is defined as the proportion of a population who have a disease, health condition or specific characteristic in a given time period, whereas, inequalities is defined as differences in the health of individuals or groups (Arcaya et al., 2015) . Caries has a relatively high prevalence globally, varying between 60 - 90% in children (World Health Organisation, 2012) with an age-standardised incidence rate of 15,205 cases caries per 100,000 person-years in 2010 (Kassebaum et al., 2015). Table 1 demonstrates caries prevalence in children for those countries for which this data is available. This table shows that prevalence of preschool caries varies between 23.3% in the United Kingdom to 97.1% in the Philippines. The Global Burden of Disease 2010 study also showed that untreated caries in deciduous teeth was the 10th-most prevalent condition worldwide that is affecting 9% of the global population, or 621 million children worldwide (Kassebaum et al., 2015).

Table 1: Dental caries prevalence by country in children
(Bagramian et al., 2009, Razmiene et al., 2011, Du et al., 2016)

Country	**Year**	**Age**	**Sample size**	**Prevalence (%)**
UK	2017	5 years	96,005	23
Ohio, USA	2004	5 years	2,555	38
Brazil	2007	0 to 5 years	1,487	40
Taiwan	2006	1 to 6 years	981	53
India	2015	5 years	-	62
China	2002	5 years	140,712	76
Philippines	2006	6 years	4,050	97

1.2.1 Inequalities in early childhood caries

Evidence also suggest that there are socio-economic related inequalities in caries prevalence between and within groups of children, which have either remained unchanged for decades (Davies et al., 2002, Levin et al., 2009) or are actually widening in some places (Do et al., 2010, Kramer et al., 2015). For instance, in the United States, children above the poverty level saw a 48% decrease in their caries prevalence from 1971–1974 to 1988–1994, whereas those children below poverty level only experienced a 23% decrease (Brown et al., 2000). This pattern is also seen in other countries like Australia (Do et al., 2010) and Canada (Baghdadi, 2016), where children from socially deprived backgrounds have a higher caries prevalence rate than children from affluent backgrounds (Slade et al., 1996).

In the United Kingdom, around a quarter (23.3%) of British 5-year olds suffer from tooth decay, according to the latest figures from the most recent oral health survey of 5-year olds published by Public Health England (PHE) (Public Health England, 2018b). The report of this survey highlighted that in some deprived areas of England, for example, Pendle and Rochdale Local Authorities, prevalence of caries in 5-year-old children is over 49.4% and 47% respectively, which means that almost one in every two children from those areas has caries (Public Health England, 2018b). However, children from affluent areas, like Horsham and Waverley Local Authorities, have a prevalence of 4.4% and 5.1% respectively (i.e., approximately one in every twenty children has caries) (Public Health England, 2018b). Further analysis of the survey of five-year-old children showed that caries prevalence consistently remained higher in local authority areas where mean deprivation scores are higher (Public Health England, 2018b), and there is an almost 20-fold difference in severity

between Waverley Local Authority in Surrey (0.1) and Pendle Local Authority in Lancashire (2.3) (Thornton, 2018, Public Health England, 2018b).

Prevalence of caries was also shown to vary regionally in the PHE report, with the highest rates of tooth decay found among children living in the North West of England (33.9%) and the lowest in the South East of England (16.4%)(Public Health England, 2018b). These figures were 33.4% and 20.1% respectively in the previous survey in 2015, which is an increase of 0.5% in the North West prevalence and a decrease of 3.7% in the South East. Some of the explanations for the regional variations seen in UK include socio-economic factors, the availability of water fluoridation and access to NHS dental care (Faculty of Dental Surgery, 2015b, Buahin, 2015). Children from Eastern European countries and those from Chinese and Eastern European backgrounds have also shown a far higher prevalence, severity and extent of caries compared to other ethnic groups in the 2016-17 Oral Health Survey (Public Health England, 2018b).

1.2.2. Consequences of preschool caries

Caries is known to have substantial impacts on quality of life in children, causing severe distressing experiences such as pain, impaired speaking, eating and sleeping disruptions (Filstrup et al., 2003). Children who present with late symptoms of caries, especially when the decay has affected a large proportion of the tooth morphology, sometimes develop systemic complications like dental abscess, fever, and life-threatening infections, such as, bacteremia and cellulitis (Li et al., 2000, Robertson et al., 2015). These cases will often require a referral to a specialist for complex restorations or multiple extractions under General Anaesthesia (Faculty of Dental Surgery, 2015b). Caries is the main reason why

children aged five to nine years are admitted to hospital in England, according to the UK Hospital Episodes Statistics data (Public Health England, 2013). Children with caries are also three times more likely to miss school as a result of dental pain, than those without caries (Jackson et al., 2011), which ultimately undermines the ability of these children to perform well academically (Jackson et al., 2011, Rebelo et al., 2019). Similarly, untreated caries is one of the causes of failure-to-thrive in otherwise healthy children (Sheiham, 2006). This is mainly because toothache is known to lead to a reduction in food intake (malnourishment), disturbed sleeping habits, poorer oral health-related quality-of-life (OHRQoL), and ultimately chronic anaemia in children (Casamassimo et al., 2009).

Finally, caries studies show that caries in preschool children has a far reaching economic impact on the society, where children admitted mainly for caries-related diseases cost the NHS in England £50.5 million between 2015 and 2016 (Public Health England, 2017). A recent Public Health England publication revealed that there were 7,926 caries-related admissions in under the age of five-years costing approximately £7.8 million per annum (Public Health England, 2017). Parents have also reported loss of income and employment owing to days of work missed from taking their children for multiple dental appointments (Casamassimo, 2009).

In summary, this chapter showed that there is a consistent complex relationship between vulnerable groups of children, such as, minority children and those from families with low incomes, and prevalence of early childhood caries. These inequalities pathways need to be further explored to wholly understand how caries inequalities develop and the factors

responsible for them, which will ultimately help in the identification of areas of opportunities needed to reduce the societal burden of caries in children.

1.3 Structure of the thesis

In capturing the various aspects of the research, an outline of what each chapter of the thesis covers is presented as follows: Chapter one discusses the problem by providing the background to the study and outlining the prevalence of dental caries in children, associated inequalities in prevalence across countries and especially within the UK and the overall impact of the disease. Chapter two highlights the cause of the problem, also known as "cause of the causes". It further discusses the upstream social determinants of health (SDH) factors and the underpinning theories of how these factors influence health behaviour and ultimately health inequalities generally. Chapter three extends the knowledge gained from the previous chapters by identifying those important "cause of the causes" risk factors influencing caries inequalities in preschool caries. These factors were identified by systematically reviewing longitudinal studies and case-control studies. Longitudinal studies and case-control studies were preferred because of its characteristics to demonstrate temporality, which is arguably the most important factor in causal pathways. The overall objective of chapter four was to develop a conceptual framework for ECC built on evidence from the systematic review. Conceptual frameworks have been found useful in disease epidemiology because they visually aid the understanding of disease progression, as well as, highlight potential areas of intervention that offers the greatest benefits. In chapter five, a caries inequalities risk model was developed that is able to predict caries risk at individual level. This risk model was developed using real world epidemiologic dataset from Child

Dental Health Survey dataset (2013). Similarly, in chapter six, a caries inequalities risk model which is able to predict caries prevalence at local authority level was developed. The final chapter contains a discussion of the findings of the entire study in the context of the overall aim and objectives outlined in Section 2.3. This chapter was divided into three main sections: the first section is an outline of the synthesis of the findings from the four previous chapters (chapters 3-6) with discussions on the relationship of the key findings to existing literature; the second section underscores the overall strengths and weaknesses of the thesis while the third section highlights the potential policy implications of this study and recommendations for future research.

CHAPTER TWO: SOCIAL DETERMINANTS OF HEALTH (SDH) IMPACTING EARLY CHILDHOOD CARIES

2.1 Introduction

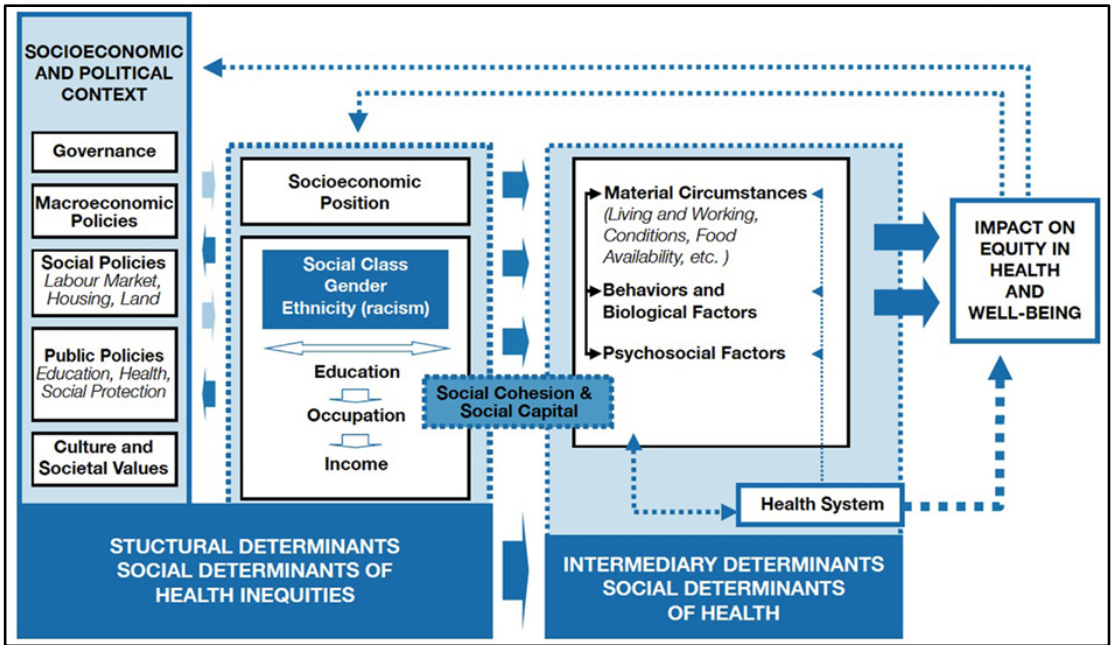
Contemporary epidemiological studies have highlighted that caries development is more complex than the demineralisation and remineralisation theory suggested by Levine and which was described in chapter 1 (Qiu et al., 2014, Duijster et al., 2014, Fisher-Owens et al., 2007). These studies emphasised that caries develops from a complex relationship between microbial, social, genetic, biochemical and physical environmental, and health-influencing behavioural factors (Fisher-Owens et al., 2007, Martins et al., 2014, Duijster et al., 2014, Cypriano et al., 2011). Petersen et al., (2005) study equally showed that the current pattern of caries and oral diseases risk factors is far more related to living conditions, lifestyles and environmental factors (Petersen, 2005). Social structures and social environments, also known as upstream factors, exert direct and indirect influences on both oral health behaviours and caries causal pathways in early life (Watt, 2007). These upstream factors indirectly tilts the “see-saw” balance (Figure 1) towards the negative (demineralisation) direction (Murray et al., 1991, Sgan-Cohen et al., 2014, Chi and Masterson, 2013). These upstream risk factors are collectively known as social (wider) determinants of health (SDH) (Watt, 2007, Williams et al., 2008), and the World Health Organisation (WHO) defined SDH as "the conditions in which people are born, grow, live, work and age" and "the fundamental drivers of these conditions" (World Health Organisation, 2010). They are known to shape individual health behaviours (Glanz and Bishop, 2010), and social epidemiologists summarised them collectively as the 'causes of the causes' of diseases and illnesses (Braveman and Gottlieb, 2014, Greenwood and de Leeuw, 2012, Wilkinson, 2003, World Health Organisation, 2013).

The theory that SDH influences health behaviour has long been widely accepted by majority of social epidemiologists but its pathways, including how to measure it, is not yet fully understood (Yen and Syme, 1999, Bonnefoy et al., 2007, Braveman et al., 2011). However, the development of effective social policies and health interventions that are robust enough to tackle health inequalities is largely dependent on the understanding of the complex causal pathways between SDH and disease (Braveman et al., 2011, Walker et al., 2014).

In an effort to explain the possible pathways through which SDH influences health behaviour, the WHO made an attempt to propose three main theoretical perspectives, which include: the social selection perspective, the social causative perspective and the life course perspective (World Health Organisation, 2010). The social selection perspective believes that it is health that determines our socioeconomic positions, instead of vice-versa, such that unhealthy people tend towards the lowest rung of the social gradient and healthy individuals move up the gradient (World Health Organisation, 2010). The social causative perspective believes that social position determines health through intermediary factors, such as, behavioural, psychosocial factors and negative life events (World Health Organisation, 2010). According to WHO, this perspective is the main explanation for health inequalities. Finally, the life course perspective emphasises the importance of time and timings when making causal links between exposures and outcomes. This perspective helps to understand how SDH influences disease during the main life phases: early childhood, childhood, adolescence and adulthood. It is also important to stress that these three perspectives need not be treated as being mutually exclusive, but as complementing one another (World Health Organisation, 2010).

Furthermore, the WHO developed a framework to assist in tackling SDH risk factors that are influencing health behaviour negatively Figure 2. This framework was organised into structural determinants and intermediary determinants, highlighting that the upstream factors influencing health inequalities operate structurally at social, economic and political levels, and at an intermediary level via health systems, health behaviours, material circumstances, psychosocial, social cohesion and biological factors (Marmot et al., 2008). These risk factors come into existence as a result of a combination of poor political decisions, inequitable economic plans and poor social policies and poor health-related programmes (Marmot et al., 2008, Marmot and Bell, 2012).

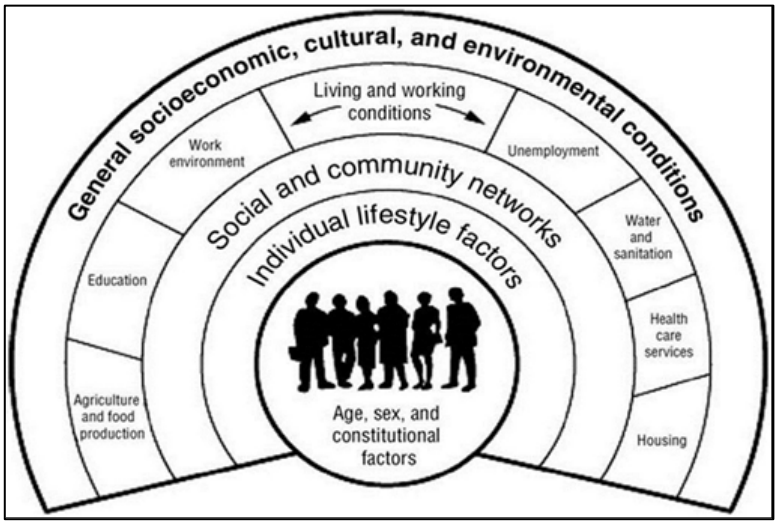
Figure 2: WHO Conceptual Framework on Social Determinants of Health (World Health Organisation, 2010)



Another important and unarguably the most widely used SDH framework is the Dahlgreen and Whitehead SDH Framework (Smith-Merry et al., 2009). The Dahlgreen and Whitehead SDH Framework emphasised the importance of environmental factors driving health-related behaviours (Dahlgren and Whitehead, 1991). This framework is mainly divided mainly into four (outer) modifiable 'policy levels' layers and an innermost (core) layer (Figure 3). This core layer is made up of non-modifiable risk factors, such as, age, sex and the genetic make-up (Söderbäck and Udén, 2009). The first modifiable layer (lying immediately above the core layer) in the Dahlgreen and Whitehead framework consists mainly of individual lifestyle (behavioural) factors. Some relevant examples of factors within this layer include diet, oral health behaviour, routine oral hygiene. The second modifiable layer involves the support an individual receives from family, friends and the local community. The third modifiable layer comprises water, education, occupation and health care and according to Dalhgreen and Whitehead, this level can only be influenced through laws, policies and strategies (Dahlgren and Whitehead, 1991). The final modifiable layer is an

overarching major structural environment that cuts across all the other layers and influencing this would require a long-term structural interventions like tax policies, environmental and trade agreements between countries etc (Dahlgren and Whitehead, 1991).

Figure 3: Wider determinants of health according to Dahlgren and Whitehead
Source: (Dahlgren and Whitehead, 1991).



2.2 Evaluation of existing caries risk frameworks for SDH factors

Disease frameworks are known to improve the understanding of disease progression and they can help highlight the potential areas for targeted interventions (Edelstein, 2006). Comprehensive health frameworks should incorporate both the structural and the intermediary social determinants factors in order to be fit for the purpose of reducing health inequalities (World Health Organisation, 2010). It is therefore important to explore existing caries risk frameworks, which are known to be the building blocks of caries prevention (Ten Cate, 1994), against established SDH frameworks like the WHO and Dahlgreen and Whitehead SDH frameworks. Existing caries frameworks were explored and

fourteen were identified, with the earliest developed in 1969 and the most recent in 2016. (Chi, 2013, de Silva et al., 2016, Duijster, 2014, Fierro Monti et al., 2014, Fisher-Owens et al., 2007, Holst et al., 2001, Keyes, 1969, Kim Seow, 2012, Patrick et al., 2006, Pine et al., 2004, Qiu, 2014, Roncalli et al., 2016, Selwitz, 2007, Watt and Sheiham, 2012). The SDH characteristics of these frameworks are summarised in Table 2 and an assessment of these frameworks against standardised social determinants showed that there were variations in the level of details presented, especially when it comes to the components that make up the frameworks (Table 2). For example, the earliest and still the most widely promoted framework in caries prevention had no SDH component included in the framework (Keyes, 1962), and the other frameworks were not consistent in their SDH components.

Table 2: Current caries frameworks mapped against WHO tool

Authors	Year	Age Group	Prior Review of Risk factors	Method	Policies	Socio- Economic Position	Gender	Race	Education	Occupation	Income	Social capital	Health System
Keyes	1969	undefined	No	Conceptual									
Holst et al	2001	undefined	No	Conceptual	Policies, Economic conditions						Material factors	Social Environment	
Cynthia Pine et al	2004	3-6years	No	Conceptual	access to fluoride	SES		Ethnicity			Poverty		Preventive Health Services
Patrick et al	2006	undefined	No	Conceptual	Fluoride, policies, access to care								Delivery of Services
Selewitz et al	2007	undefined	No	Conceptual					Education		Income		
Fisher-Owens et al	2008	0-18 years	No	Conceptual	Physical and Social environment	SES	Gender	Ethnicity	Education			Safety, Social capital/Support	Health Systems
Watt and Sheiham	2012	undefined	No	Conceptual	Policies, Economic conditions	Social class	Gender	Ethnicity		Occupation	income	Social capital	
Kim Seow	2012	<6years	No	Conceptual		SES, Social Disadvantage		Ethnicity	Maternal education				
Donald L. Chi	2013	<18 years	No	Conceptual	Fluoride, policies							Social capital, Social Support	
Duijster D et al	2014	6 years	No	Statistical Modelling	Neighbourhood quality			Ethnicity	Maternal education			Social support	
Brunotto et al	2014	3-5years	No	Statistical Modelling		SES	Gender		Maternal education				
Qiu et al	2014	5years	No	Statistical Modelling	dental insurance	SES							
De Silva et al	2016	undefined	No	Conceptual	Distance to clinic, fluoridation			Ethnicity	Education				
Roncalli et al	2016	12 year olds	No	Conceptual	HDI, Infant Mortality, Gini, primary care expenditure						Poverty		Dentist population ratio

Some of the weaknesses in existing conceptual caries frameworks explored are highlighted as follows: Firstly, the exercise showed a lack of agreement or standardisation on the wider determinants risk factors needed to be included in a caries framework, which I have termed in my thesis as the "which SDH determinants" gap. It is advised that identification of components of a framework should follow a systematic evidence-based process, in order to identify those parameters that are "relevant" during the process of framework development (Tappenden, 2012). The second weakness identified in these frameworks is the "methodological factor" gap. Majority of the frameworks (11 frameworks) were developed based on authors' conceptions without describing how the risk factors in the framework were selected. A conceptual framework is defined by Holzemer et al as a mental image that is for the purpose of describing, relating and predicting a desired situation (Holzemer et al., 2007), but ultimately, quantitative description of the relationship of the factors involved is essential for burden of disease studies and analyses of interventions (Kruijshaar et al., 2002).

These weaknesses described therefore highlighted that none of the existing caries frameworks was developed with equity in mind, i.e. they were not developed to address inequalities. Equity is defined by WHO as "the absence of unfair and avoidable or remediable differences in health among population groups defined socially, economically, demographically or geographically"(World Health Organisation, 2010). Lack of well-constructed equity-focused caries frameworks is possibly a major omission, given that the WHO recommended that disease frameworks need to incorporate both structural and the intermediary social determinants factors (World Health Organisation, 2010).

The implication of these limitations is that current frameworks are inadequate and may not be fit for the purpose of reducing inequalities because of the gaps identified in existing frameworks. Therefore, implementing any of the existing caries frameworks might therefore misguide policy developers that have the mandate to plan caries prevention programmes in children and narrow inequalities gap. Hence, there is a need to develop evidence-based caries inequalities framework, using systematic methods that would address both the "which SDH factors" and the "methodological factor" gaps, and will be able to support policy development.

2.3 Thesis aims and objectives

Given these issues and the consequences of ECC enumerated above in section 1.2, the rationale for conducting this research is mainly because ECC has become a global public health challenge, especially since prevalence has remained unchanged or worsening among some groups of children with similar characteristics. ECC also impacts on the child, family and the health system at large and therefore the need to understand the underlying mechanisms of both caries inequalities.

The overall aim of this research was to develop a caries inequalities risk prediction model that can inform policy and decision makers about caries prevention, and in fulfilling this aim, the following objectives were addressed:

I. To identify all possible SDH factors influencing the development of caries in preschool children, aged 71 months of age or younger, i.e., "which SDH factors".
II. To identify theoretical pathways by which SDH influences tooth decay and use these to develop a conceptual caries inequalities framework, i.e., "methodological factor"

III. To develop conceptual frameworks that describe causal pathways between SDH and caries development.

CHAPTER THREE: SOCIAL DETERMINANTS OF HEALTH INFLUENCING THE DEVELOPMENT OF EARLY CHILDHOOD CARIES: A SYSTEMATIC REVIEW

3.1 Introduction

The overall purpose of this systematic review chapter was to identify SDH-related risk factors influencing caries development in primary teeth of children 71 months of age or younger, i.e., "which SDH determinants". This review is essential owing to the two main observations in the evaluation of existing caries risk frameworks, carried out in chapter two. The first observation showed that there is no collective agreement (inconsistency) in the wider determinants influencing the development of caries in children under the age of six, and the second observation was that none of the SDH-related risk factors within the frameworks were identified from a prior systematic review, as recommended by Tappenden (Tappenden, 2012). This review therefore aims to address this gap in the literature, and to the best of the research student's knowledge, there is no systematic review at the time of undertaking this review that has explored SDH related influences associated with development of Early Childhood Caries (ECC). Also considering the vastness of SDH factors, none has employed the use of a validated, evidence-based framework to guide the search strategy and retrieval of relevant publications in SDH impacting on the development of ECC.

3.2 Developing research question

The first step in this review was the development of an appropriate research question, using the Population, Concept and Context approach as recommended by Joanna Briggs Institute (Peters et al., 2015):

Population (Participants)

The study population in this review was defined in advance, as children (boys and girls) of any race, religion, or nationality who are under the age of six years.

Concept

This is also known as “exposure” (explanatory variable), which is defined in this study as any SDH risk factor(s) that may be associated with the development of early childhood caries. One of the key challenges in social epidemiology is how to identify all possible SDH that affect health (Oliver et al., 2008). Therefore, some researchers came up with an equity-focussed framework called PROGRESS-PLUS that helps to ensure all possible SDH are explored (O'Neill et al., 2014). PROGRESS-PLUS is an acronym used to identify all possible socially stratifying factors that cause variations in health outcomes. Cochrane and Campbell Equity Methods Groups have validated and advocated using the framework, when reporting equity-focused systematic reviews (O'Neill et al., 2014). PROGRESS stands for: Place of residence (rural/urban/inner city, low, medium income countries), Race/ethnicity/ culture/language, Occupation, Gender/sex, Religion, Education, SES, and Social capital (Evans and Brown, 2003). Then, “PLUS” covers other context-specific SDH factors that also enable disadvantage (Nasser et al., 2013). Cochrane classified PLUS factors into three groups, which are: personal characteristics associated with discrimination (e.g. age, disability), features of a relationship (e.g., smoking parents, excluded from school), and finally, time-dependent relationships and other circumstances that may indicate disadvantage.

3.2.2.1 PROGRESS-PLUS Definitions in this thesis

Definitions for the components of PROGRESS-PLUS in this thesis were based on evidence provided from the two separate studies conducted by Oliver et al., (2008) and O'Neil et al., (2014), where they acknowledged the difficulties of classifying and assessing SDH risk factors and recommended the use of an evidence- based framework that ensures SDH factors are explored (Oliver et al., 2008, O'Neill et al., 2014). The description of the components of the PROGRESS-PLUS framework are described below:

1. **Place of Residence** – this includes the physical, geographical, social or economic characteristics of the area of residence of a child or carer. It also includes the Human Development Index (HDI) of the area, which is a statistical tool used to rank (assess) countries by their level of the socioeconomic developments.
2. **Race/Ethnicity** – this is the racial or ethnic characteristics of a child, which is sometimes determined or implied in papers by either primary language spoken at home, or country of birth of parents or child.
3. **Occupation** –Occupation is defined in this review as whether the primary care giver is employed or not. This also includes studies that explored the number of hours the main primary care givers spend at work, that is, full time or part time (Ismail, 2009, Tanaka, 2013, Schroth et al., 2014). Similarly, some studies explored the impact of the type of job the primary giver does, categorised into white collar, blue collar, professional, technical or manual job types on caries development in children (Mattila et al., 2000, Meurman and Pienihakkinen, 2011, Tanaka, 2013). The European Union defined white collar jobs as job categories within International Standard Classification of Occupations

(ISCO) codes 1-5 (including legislators, senior officials and managers, professionals and technicians and associate professionals, clerks and service workers and shop and market sales workers). Similarly, blue collar is defined as jobs that fall within ISCO codes 6-9 (skilled agricultural and fishery workers and craft and related trades workers; plant and machine operators and assemblers and elementary occupations) (EUROFUND, 2010).

4. **Gender** – characteristics relating to gender of the child (boy of girl), or gender of the main primary caregiver (male/man or female/woman).
5. **Religion** – defined in this thesis as the religious background of the child or parent/primary carer.
6. **Education** – Educational attainment or number of years in school of parents.
7. **Socioeconomic Status** – Encompasses all income or socioeconomic related measures (deprivation status) at individual level.
8. **Social Capital** – Social capital is defined as social connections and all the benefits they generate (Harper and Kelly, 2003). Researchers widely accept that social capital is difficult to measure (Illingworth, 2012, Pichler and Wallace, 2007), and have therefore recommended the use of proxies or indicators to measure it (Illingworth, 2012). This PhD used the five-dimensional classification that was developed by The Organisation for Economic Co-operation and Development (OECD) and validated by The Office for National Statistics (ONS) to define and measure Social Capital based risk factors in the retrieved studies (Harper and Kelly, 2003). These five dimensions are: social participation; civic participation; social networks and social support; reciprocity and trust; and finally, views of the local area (Harper and Kelly, 2003).

9. **PLUS** – Definition of PLUS is retrospective, that is, the research student was only able to define PLUS risk factors after data extraction. Therefore, PLUS factor is defined in this thesis as all SDH-related caries influencing risk factors identified that are not captured by (or does not fit into) PROGRESS. Inclusion of PLUS factors allowed for the assessment of additional context-specific risk factors that could not fit under PROGRESS.

3.2.3 Context

The context is the outcome, which is defined as caries in primary teeth. This is usually assessed by the dmft (number of decayed, missing and filled teeth), dmfs (decayed, missing and filled surface) indices, which are the two most commonly used oral epidemiological indices for assessing dental caries experience (Broadbent and Thomson, 2005, Banava et al., 2012), and validated by WHO (World Health Organisation, 2013, Braga et al., 2009). The other widely used epidemiological index accepted for inclusion in this study is the International Caries Detection and Assessment System (ICDAS), based on visual inspection. This was introduced because of its ability to include non-cavitated caries in epidemiological surveys (Braga et al., 2009). Oral epidemiologists have acknowledged that the ICDAS can generate results comparable to the WHO validated dmft/s index (Braga et al., 2009).

With all the three criteria (Population, Concept and Context) required for developing a research question defined for this review, a simple research question was developed as follows:
"Which social determinants of health are responsible for caries inequalities in children under the age of six?"

3.3 Methods

Systematic reviews developed from studies that are able to account for temporality (the time between exposure and outcome) are still the best strategy for appraising evidence (Balshem et al., 2011). Therefore, in order to improve the quality and level of evidence of this review and to avoid bias, studies included were those with the ability to control for confounders that may have arisen from time-related variables and population-group baseline differences, such as cohort and case control studies (Schünemann et al., 2011). Temporal precedence is known as the most important factor needed to validate that the effect occurred after exposure (Centers for Disease Control and Prevention, 2004). Cross-sectional studies were purposely excluded, because they only offer single-point outcome assessment (Mann, 2003), and therefore fail one of the main criteria in Bradford Hill's criteria for causation, known as temporal precedence (Schünemann et al., 2011).

3.3.1 Selection criteria

Inclusion criteria

Included in this review were longitudinal studies such as prospective or retrospective cohort studies and case-control studies, where the primary outcome is caries in children under the age of six (< 71 months), assessed either by clinical examination or self-reported (by primary caregiver). This age group was chosen in line with the age-group definition for early childhood caries (American Academy of Pediatric Dentistry, 2008), however, also included were studies

where participants' ages were greater than 6 years old, but which have a separate analysis of participants aged 0 to 6 years old.

Exclusion criteria

Initial selection criteria included both cohort and case-control studies, however, case-control studies were subsequently excluded in the review when there were more than five cohort studies in the SDH category explored. Wherever there was a gap in any evidence, the study would include lower quality evidence (case-control studies) to see what it can contribute to the review. Studies were also excluded if they were cross-sectional studies or reviews, as were studies where participants are a cohort of children with underlying illness or systemic disease or articles written in languages other than English due to constraint of time and resources. Also excluded were non-peer reviewed journals, conference abstracts and studies submitted for a PhD.

3.3.2 Search Strategy

Searches used a combination of specific controlled vocabulary for each database, such as MeSH in MEDLINE and EMTREE in EMBASE and natural language search techniques to identify relevant materials and maximise retrieval across a range of databases (Centre for Reviews Dissemination, 2009). Natural language searches were truncated where relevant, to allow for plurals and to utilise word stems to maximise retrieval. Separate searches were then developed under each of the three main categories mentioned earlier, *Population, Context and Concept*. Within the relevant categories, "Children", "Social Determinants of Health" and "Caries" were

identified using keywords and subject headings, and the Boolean operator 'OR' used to connect them. Finally, the three category sets of searches developed were linked with the Boolean operator "AND". Searches were restricted to peer-reviewed studies reported in English language (due to resource constraints) and in humans. There was no restriction on study design or on publication year.

3.3.2.1 Databases

The Centre for Research Dissemination recommends conducting a comprehensive search in a minimum of two databases, in order to avoid unintentionally introducing bias into the review (Centre for Reviews Dissemination, 2009). They recommended searching at least MEDLINE and EMBASE (Centre for Reviews Dissemination, 2009). Seven different databases were searched in this review, which included MEDLINE, EMBASE, and CINAHL because these are the largest and commonly used databases that cover a vast majority of dental journals (Lamurias et al., 2017, Hamric et al., 2014), whilst Dentistry and Oral Sciences database was also included because they focus mainly on dental journals and have extensive selection of dental journal literature (Swogger and Samsky, 2014). Additionally, due to the complexity and broadness of the topic (social determinants of health), this search was then widened to include databases which are specialist in indexing sociology or social medicine papers, the largest databases being PsycINFO, SocIndex and Social Science and Abstracts (University of Nevada, 2017). All the databases were searched on EBSCO platform, except EMBASE that was searched on OVID. This search was last updated on the 30th of December 2018. The search strategies used on EBSCO (for MEDLINE,

Dentistry & Oral Sci, CINAHL, PsycINFO, Soc INDEX, Social Sci) and OVID (for EMBASE) platforms are in Appendix 1 and Appendix 2.

3.3.2.2 Reference Management in Endnote

All articles identified were exported from their sources to the bibliographic management program EndNote version X9 (Clarivate Analytics, New York, United States). Citations from all the seven databases used in this study were all exported into their separate database groups and the reference manager used to combine the citations from all the databases. Endnote was also employed to remove duplicates and a separate folder created, where all citations that meet inclusion criteria were saved. All excluded citations (and reasons for exclusion) at second screening phase were also documented in a separate folder on EndNote and reasons for exclusion was documented in Appendix 7.

3.3.3 Eligibility Assessment

3.3.3.1 Initial screening

PhD research student and Dental Research Assistant screened titles and abstracts against the inclusion/exclusion criteria in order to identify eligible articles for full review and to minimise bias from a single reviewer. The opinion of a third reviewer was sought (Director of Studies), where there was disagreement on inclusion of any article, abstracts were categorised as either 'potentially relevant' or 'not relevant' studies.

3.3.3.2 Second Screening

Full texts of potentially eligible studies were retrieved and screened by the research student, using a tailored data extraction form (Appendix 3 and Appendix 4) as recommended by Cochrane Collaboration (Deeks et al., 2011). Where full papers were not obtained, the research student used interlibrary loan and at last resort, wrote to the corresponding author to request it. Where full text articles were unavailable, the studies were not included in the review. Multiple publications and reports were linked as advised by Centre for Reviews and Dissemination, in order to reduce double counting, and prevent inadvertently overestimating significant treatment effects when these findings are combined (Centre for Reviews Dissemination, 2009). Similarly, in situations where more than one report was retrieved for the same study and the outcome measure was similar, preference was given to the peer-reviewed paper with the largest sample, or, if the sample size was the same, the most recent publication (Henry et al., 2016). However, in situations where outcomes differed for same study, all outcomes were extracted for the review (Beckers et al., 2017). Finally, the research student searched the reference list of all identified studies, for possible eligible studies missed through the electronic search.

3.3.4 Data extraction

Data extraction is a process that locates and synthesizes relevant information about study characteristics and findings from the included studies (Centre for Reviews Dissemination, 2009), and is a pivotal step in all reviews (Munn et al., 2014). The development of a data extraction form is an important step in a systematic review because it ensures that there is consistency in

the extraction of data from the articles, thereby reducing bias (Centre for Reviews Dissemination, 2009). A data extraction form, modified from the Joanna Briggs Institute manual for scoping reviews, was piloted and used as a template for documenting significant study characteristics of the included articles in the review (Peters et al., 2015).

The following information was extracted from each included study:

1. **Publication-level data**: Authors, Publication Year, Journal, Title, Identification Method (i.e. Electronic or Hand Search).
2. **Study-level data**: Study Aims/Purpose, Study Design, Methodology, Country of Study, Study Population and Sample Size (if applicable).
3. **Population-level data:** Number of Participants, Age range/mean age, Gender, Ethnicity, Population Source (e.g., “Hospital” or “Community”).
4. **Measurement:** Caries assessed by dmft, dmfs, ICDAS or self-reported (by primary caregiver).
5. **Exposure(s) investigated:** Social Determinants of Health explored, which was then organised using PROGRESS-PLUS framework (O'Neill et al., 2014). Exposures that did not fit within PROGRESS-PLUS framework were discussed under “Other Factors”.
6. **Reported measure of association(s):** Odds ratio, risk ratio, difference in means (with their 95% Confidence Interval)
7. **Key findings:** These included results of the analyses carried out on the SDH Risk factors

3.3.5 Methodological Quality Assessment

The quality of the included studies was assessed by the research student using a standardised quality assessment tool, also known as “Risk of Bias” (ROB) tool. Several types of ROB are available and choice is determined by the type of study to assess. In this review, the Newcastle Ottawa Scale (NOS) ROB was used to assess included studies because it was specifically developed to assess quality of longitudinal studies, such as, cohort and case-control studies. The NOS tool is known for its high level of reliability and validity and easy to use (Li et al., 2008, Savović et al., 2014, Wells et al., 2011), and recommended by Cochrane Collaboration for use in systematic reviews of non-randomised studies (Deeks, 2011). There are three main domains assessed in the NOS tool, which are (a) representativeness of study groups (4 points); (b) comparability of study, that is, study controlled for demographic factors such as age and sex (1 point); and (c) outcome assessment (4 points), therefore the total NOS scores range from 0 to 9. Studies were concluded to be of low quality if their NOS score is ≤ 4, moderate quality if NOS is between 5 and 7, and high quality if their NOS is ≥ 8 (Schwendicke, 2015). Copies of the Risk of Bias tool used in this systematic review are found in Appendix 5 and Appendix 6.

3.3.6 Approach to analysis

Meta-analysis is the use of statistical methods, like statistical pooling using forest and funnel plots, to summarise the results from a systematic review (Crombie and Davies, 2009). However, this is not always possible or appropriate when there is high level of diversity in population characteristics, and a narrative synthesis of the results is recommended under such circumstances (Arai et al., 2007). Therefore, a narrative synthesis of results was adopted in this

review, given the high level of heterogeneity of the study designs (Rodgers et al., 2009), the population characteristics, as well as, high level of heterogeneity in the definitions used by authors for each of the SDH assessed in included studies (Deeks et al., 2011). The synthesis and reporting was undertaken according to recommendations from the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Deeks, 2011).

3.3.7 Presentation of Results

The results of this review were presented in two parts as recommended by Murray et al., (Murray et al., 2016):

1. A numerical analysis: This process mapped the data in tabular and diagrammatic form, highlighting the distribution of studies by themes as follows: Authors and Date of Publication; Study Design; Age of Participants; Sample Size; Country of Study; Human Development Index (HDI); Risk Definitions and Outcomes; and Risk of Bias Score (Appendix 8).

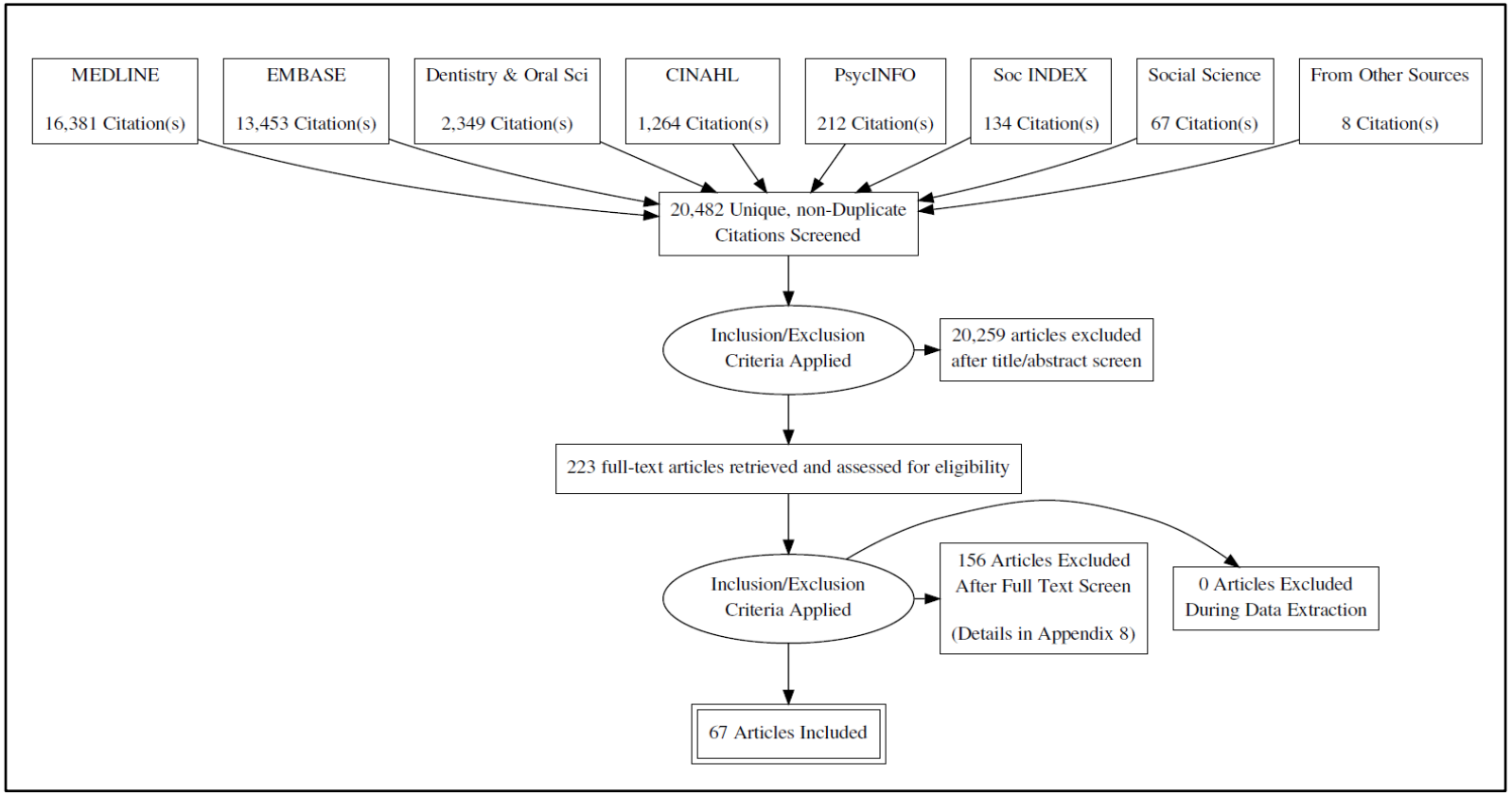
2. A thematic summary: This phase provided a narrative analysis detailing how the studies identified relates to the research question and the main findings from these, organised by themes. Identification of these determinants will help to further understand characteristics of children who are at risk of developing caries, as well as map the gaps in available literature.

3.4 RESULTS

3.4.1 Findings of the search strategy

There were a total of 20,482 abstracts identified and 223 titles and abstracts were considered to be potentially eligible on the first screen (title and abstract) (Figure 4). Full texts of these studies were retrieved for full text screen review, and a further 156 papers were excluded, and a total of 67 publications from 59 studies were considered eligible for this review. A PRISMA flow chart of the screening and selection of studies is shown in (Figure 4), and the three major reasons for exclusion were: Social Determinants of Health were not assessed (n=46), study design was cross sectional (n=43), and participants' ages were not between 0-6 years (n=39). Other reasons for exclusion are detailed in Appendix 7.

Figure 4: PRISMA Flow Diagram



3.4.2 Characteristics of eligible studies

Sixty-seven publications were retrieved from 59 studies, which included 43 cohort studies and 16 case-control publications. Sample sizes ranged from 60 children (Smith et al., 2002) to 132,462 retrospective birth cohort in the United States (Flaherman et al., 2018), with an overall median of 543 children (median preferred to mean because data was skewed). There were 466,433 participants in total from 19 different countries: nine studies were undertaken in the United States, eight in Brazil, six in Australia, five in India and Japan, four in Sweden, three in Canada and Thailand, two in China, Finland, Norway, Singapore and United Kingdom and one each in Belgium, Croatia, Denmark, Germany, Netherlands and New Zealand. Forty-one studies (66%) were from developed countries with Human Development Index (HDI) >0.8, thirteen studies (26%) were from developing countries with HDI between 0.6–0.8; and five studies (8%) from under developed countries with HDI < 0.6. The quality of the studies varied from 2 to 9 on the Newcastle Ottawa scale and two publications from two studies were judged to be of low quality (NOS ≤4), thirty-two publications from twenty-nine studies of moderate quality (5–7), and thirty-three publications from twenty-eight studies were of high quality (≥8). The number of papers retrieved is summarised in Appendix 8.

3.4.2.1 Place of Residence

Thirteen papers explored the relationship between dental caries and place of residence, where six explored the difference in risks of dental caries associated with living in areas with and without optimally fluoridated water; three examined geographical place of residence and risk of caries; one study examined area-level deprivation and risk of caries; and the last

three studies explored the relationship between the number of dentist per 1,000 population and the risk of caries. Each of these groups are further discussed below and the characteristics and the findings of the thirteen studies are shown in Appendix 9 to Appendix 12.

Water Fluoridation

Of the six studies that explored the influence of living in an area with fluoridated drinking water, four were cohort studies (Peltzer, 2014, Sanders and Slade, 2010, Hong et al., 2014, Gussy et al., 2016) and two were case-control studies (Mahesh, 2013, Tiberia et al., 2007). Although, all the studies in this category assessed the effect of sub-optimal water fluoridation on early childhood caries, definition of optimal water fluoridation varied between the countries in these studies. Definition of optimal fluoridation varied from greater than 0.3mg/L in the study undertaken in India (Mahesh, 2013), to greater than 0.6mg/L in both studies from Australia and United States (Sanders and Slade, 2010, Koh et al., 2015, Hong et al., 2014). In four of the six studies, the source of the drinking water was pipe-borne water in both the suboptimal and optimal group. However, Peltzer et al., used source of water as a proxy to determining a child's access to water fluoridation, such that fluoridation was considered to be optimal if the source of drinking water was either pipe or bottled water, and suboptimal, if the main source of drinking water was either from rain or well water (Peltzer, 2014).

Based on the results from the studies, there were four papers that concluded that children who lived in areas with suboptimal compared to optimal water fluoridation had significantly higher risk of developing caries (Mahesh, 2013, Peltzer, 2014, Sanders and Slade, 2010,

Hong et al., 2014), and the last two (one cohort, and one case-control) did not find any statistically significant difference in children with or without access to water fluoridation (Tiberia et al., 2007, Gussy et al., 2016). The characteristics and the findings of the six studies are shown in Appendix 9.

Geographical area of residence

Three studies explored the geographical location of a child's residence and caries (Ju et al., 2016, Mattila et al., 2000, Ohsuka et al., 2009). Two of these studies defined the geographical area of residence of a child as either rural or urban (Mattila et al., 2000, Ohsuka et al., 2009). Urban regions were defined as built up cities or major towns, usually with more population, while rural areas were defined as remote areas that are outside of major cities, with less population (Mattila et al., 2000, Ohsuka et al., 2009). The outcome in the two studies using urban-rural definition showed that children living in rural areas were at higher risk of developing caries than children living in urban areas. Mattila et al., study demonstrated an almost 2.5 times risk of developing caries in children living in rural areas. Oshuka et al. study did not present odds or risk ratio in their study but presented prevalence and average caries experience per population (i.e. 3-year olds). Their study found that the prevalence of caries in 3-year olds was 25.7% in children living in urban areas and 47.6% in children living in rural areas [$p<0.05$]. Similarly, average dmft in urban area was 0.88 ± 1.97 and in rural areas was 1.73 ± 2.69 [$p<0.05$].

The final study in this category was undertaken in Australia and it used more refined definitions of geographical area of residence instead, and the authors classified a child's usual place of residence into four major groups: 'major city', 'inner regional', 'outer regional' and 'remote and very remote' (Ju et al., 2016). The outcome measured in this study was

self-reported caries experience, provided by the primary caregiver. The authors found children residing in outer regional areas had the greatest risks of developing caries compared with children residing in major cities [risk ratio 1.30 (1.02–1.66)], after adjusting for confounders such as mother's education level, sweet food intake, maternal age and brushing frequency. The characteristics and the findings of the three studies are shown in Appendix 10.

Community-Level Deprivation

This section explored the impact of social (area-level) deprivation on developing caries in children and there was only one cohort study in this category, carried out in the United States (Ismail, 2009). The authors in this study used a computed neighbourhood disadvantage index (made up of number of grocery stores, dentists and churches per geocoded area), to determine deprivation status of the place of residence of the children participating in the study (Ismail, 2009). Participants' area-based deprivation status were categorised into four major groups, ranging from most disadvantaged community (category 1) to least disadvantaged community (category 4), depending on where the children lived. This study showed that children living in most disadvantaged low-income neighbourhood had a significantly higher incidence rate ratio [IRR = 1.43; p=0.03] than their counterparts in least disadvantaged neighbourhoods. The characteristics and the findings of the study are shown in Appendix 11.

Access to care

There were three cohort studies in this category, and only two found significant relationships between access to dentistry and ECC (Brickhouse, 2008, Rocha et al., 2017, Wigen and Wang, 2011b). Wigen and Wang study concluded that, at bivariate level, children

of parents with poor access to dental care have higher risk of developing caries with an odds ratio of 2.0 [1.2–3.3]. Similarly, Brickhouse et al., defined access as the ratio of dental providers providing care to children enrolled in Medicaid or State Children's Health Insurance Program (SCHIP) and this was categorised into three major groups as follows:

i. < 5 dentists per 1000 population
ii. 5-10 dentists per 1000 population
iii. > 10 dentists per 1000 population

The study concluded that the lifetime caries risk of a child living in areas with low dentist to population ratio were at higher than those living in areas with higher dentist to population ratio (Brickhouse, 2008). This was found to be significant in children living in the highest dentist to population neighbourhoods [Incidence Rate Ratio = -0.16; p≤0.001]. The characteristics and the findings of the three studies are shown in Appendix 12.

3.4.2.2 Race/Ethnicity/Culture/Language

Twenty-eight separate publications explored the relationship between dental caries and a child's or their parents' race, ethnicity, language, immigrant status or country of birth, with some studies exploring more than one factor in their analysis. The characteristics and the findings of these studies are shown in Appendix 13 - Appendix 15 and are further described below.

Race

The fifteen papers retrieved in this category explored the influence of the five major racial groups (Whites, Blacks, Asians, Arabs and Mixed) that was defined in the UK by the Office for National Statistics (ONS) (Office for National Statistics, 2012), and caries. These studies

were ten cohort studies and five case-control. The results of cohort will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. The results from all ten cohort studies demonstrated that a child's race is a factor in developing caries (Brickhouse, 2008, Cabral et al., 2017, Gao et al., 2010, Meurman and Pienihakkinen, 2011, Schluter et al., 2007, Schroth et al., 2014, Skafida and Chambers, 2018, Un Lam et al., 2017, van der Tas, 2016, Wigen and Wang, 2014). Nine out of the ten cohort studies demonstrated that non-natives have significantly higher risk of developing early childhood caries than indigenous children, and Brickhouse et al., (2008), study was the only contrary study, where authors concluded that children of White origin had higher risks of developing ECC (Brickhouse, 2008). Two of these studies explored the risk of caries in relation to their ethnicities (Gao et al., 2010, Schluter et al., 2007), however, the definition of ethnicity varied in both studies. For instance, In the study carried out in New Zealand, ethnicity of the children was dichotomised into Pacific or non-Pacific ethnicities, where non-Pacific ethnicity was defined as children outside of Samoan, Tongan, Cook Island Maori, "Other Pacific" origin (Schluter et al., 2007). This study showed that children of Pacific mothers were almost three times [OR= 2.7, CI=1.0, 6.8; p=0.04] as likely as children of non-Pacific mothers to have fillings and/or tooth extractions (due to caries). In the case of the study in Singapore, Gao et al., divided their study population into four groups: Chinese (68.4%), Malays (18.8%), Indians (8.9%), and others (3.9%), and they concluded that Malay children were at greater risk of developing caries than children from other three ethnicities (Gao et al., 2010). The characteristics and the findings of the fifteen studies are shown in Appendix 13.

Country of Birth

There were eleven publications from ten studies that explored the influence of one's country of birth on developing ECC. Two of the papers retrieved used the same population (Wigen and Wang, 2014, Wigen, 2011), and therefore were regarded as one study, and the most recent results were presented and discussed, as per protocol. Six of the studies retrieved were cohort studies and the remaining four were case-control, therefore, only the results of the cohort studies will be reported, as discussed in section 3.3.1

The result showed that four out of the six cohort studies concluded that either of foreign-born children or children of foreign-born parents had a greater risk of developing caries (Grindefjord et al., 1996, Julihn et al., 2018, Wigen and Wang, 2014, Wong et al., 2012). Wigen and Wang study showed that five-year old children with one or both parents of non-Western background had almost five-and-half times odds of developing caries [Odds ratio: 5.4 (1.6–7.3)], when compared to children with parents of western background (Wigen and Wang, 2014). The remaining two cohort studies in this group did not find any significant relationship between country of birth and caries in children. The characteristics and the findings of the eleven studies are shown in Appendix 14.

Language mainly spoken at home

Three studies explored the effect of language mainly spoken at home and developing caries in children (Meurman and Pienihakkinen, 2011, Nunn et al., 2009, Seow, 2009), and all of them showed that there is a significant relationship between the language spoken at home and caries in children. Nunn et al., (2009) demonstrated that US children whose primary language spoken at home was not English were more than three times the odds of developing caries than their counterpart US children whose primary language spoken at

home was English (OR = 3.2, P < 0.001) (Nunn et al., 2009). Seow et al., (2009) also made a similar conclusion, where they determined children's ethnicity by the language spoken at home and whether the child was of Australian aboriginal descent. Their study showed that children who were able to speak languages other than English at home had almost six times the odds [OR: 5.62 (1.43–22.11) p=0.032] of developing caries than Children of Australian descent (Seow et al., 2009).

The final study in this category exploring language spoken at home and caries was a study undertaken in Finland. In this study, Meurman and Pienihäkkinen (2011) defined ethnicity as the language of the child's mother, where children of mothers who did not speak the national official language (Finnish or Swedish) were considered to be of another ethnicity, and assessed in the non-Finnish group (Meurman and Pienihakkinen, 2011). This study demonstrated a significant relationship after univariate analysis, where non-Finnish children showed almost three-and-half times significant risk of developing caries than Finnish children [OR=3.3; CI: 1.1–10.1; p=0.037]. However, this significant relationship ceases to exist in multivariate analysis, after adjusting for occupation of caretaker, microbial colonisation, sugar consumption, night feeding and reported oral health of the parents (Meurman and Pienihakkinen, 2011). The characteristics and the findings of the four studies are shown in Appendix 15.

3.4.2.3 Occupation

There were fifteen studies that explored the effect of the occupation of each parent on the dental caries experience of their children. Seven of these studies defined occupation as employment status of the primary care giver, that is, whether they were employed or not,

whilst three studies considered the number of hours of work done per week, that is, full time vs part time. Finally, there were five studies that explored the relationship between the type of job (White collar vs Blue collar) and ECC.

Employment Status

Seven publications from six studies explored the relationship between primary caregivers' employment status and caries in children. Two of the papers retrieved used the same population (Pinto et al., 2016 and dos Santos Pinto et al., 2016) and therefore were regarded as one study, and the most recent results were presented and discussed, as per protocol.

Five of these studies (six publications) were cohort studies (dos Santos Pinto et al., 2016, Fontana et al., 2011, Pinto et al., 2016, Rocha et al., 2017, Tanaka et al., 2013, Yokomichi et al., 2015), and the last study in this category was a case-control study (Mahesh et al., 2013). The results in this category showed three studies demonstrating that a relationship exists between employment status of the primary caregiver and developing caries (Fontana et al., 2011, Mahesh et al., 2013, Yokomichi et al., 2015). The study carried out in the United States showed that Caucasian children with one or two unemployed parents were significantly at risk of developing caries [OR = 3.64; CI = 1.49-8.85](Fontana et al., 2011), which is similar to the outcome of a study carried out in India where children of unemployed mothers demonstrated a significant higher risk of developing caries, in comparison to children of employed mothers [Risk Ratio: 3.45, CI = 1.70–6.99] (Mahesh, 2013). Yokomichi et al (2015) study also showed that that the prevalence of ECC increased by 11% as the percentage of unemployed parents increased [Relative risk increases (RRI) (%) = 11% (CI, 6–

16, P < 0.001). The characteristics and findings of the seven studies are shown in Appendix 16.

Full-time or Part-time working parents and caries in children

A total of three studies explored the relationship between the total numbers of hours the main primary caregiver spends at work and developing caries in children (Schroth et al., 2014, Ismail, 2009, Tanaka, 2013). The three studies were all cohort studies, and were carried out in two highly developed countries of Japan (Tanaka, 2013) and the United States (Ismail, 2009, Schroth et al., 2014). None of the three studies in this category was able to demonstrate the existence of any significant relationship between the number of hours the primary caregiver spends at work and caries in children. The characteristics and findings of the three studies are shown in Appendix 17

Primary Caregiver's employment type (White collar vs Blue collar)

There were five cohort studies in the category, where the authors explored if there was any relationship between the type of job the primary caregiver does and caries in children (Cabral et al., 2017, Mattila et al., 2000, Meurman and Pienihakkinen, 2011, Skafida and Chambers, 2018, Tanaka et al., 2013). Four of these studies showed that the type of job the primary caregiver does is a risk factor in developing caries in children, but Tanaka et al (2013) did not find any significant relationship between job type of parents and ECC. Two out of the four studies with significant relationships demonstrated that children of "blue collar" job parents were at higher risk of developing caries, and the relationship was significant (Mattila et al., 2000, Meurman and Pienihakkinen, 2011), and Meurman and Pienihäkkinen study went further to demonstrate the children of "blue collar" job parents were almost twice at risk of developing caries, when compared to their counterparts from

"white collar" job parents [Odds Ratio: 1.9; CI = 1.0–3.4; p = 0.034] (Meurman and Pienihakkinen, 2011). The remaining two studies used standardised job classifications such as NS-SEC in the UK and Brazilian classification of occupations and both found that children of parents on the lowest rung of the job classification ladder were at higher risk of developing ECC (Cabral et al., 2017, Skafida and Chambers, 2018). Cabral et al. study showed that this risk of developing ECC can be as high as six times the risk of children whose mothers are higher up the ladder [HR = 6.04 (2.22–16.4)) (Cabral et al., 2017). The characteristics and the findings of the five studies are shown in Appendix 18 below.

3.4.2.4 Gender

There were 27 publications (from 26 studies) that explored the relationship between caries incidence and gender of the child. Sixteen were cohort studies and ten were case-control studies, therefore only the results of the 16 cohort studies were presented, as highlighted in section 3.3.1. There were four publications from three studies that found a relationship between gender and developing ECC, and the two publications from Ismail et al., (2008 & 2009), were considered as one. The results showed that two of the cohort studies found males to significantly have higher risk than females (Guedes et al., 2016, Yokomichi et al., 2015) and only one study (two publications) found females to have higher risks of developing caries (Ismail et al., 2008, Ismail, 2009). The remaining 13 cohort studies concluded that there was no significant relationship between gender and caries in children. The characteristics and the findings of these studies are shown in Appendix 19.

3.4.2.5 Parental Education

Parental level of education was one of the most frequently explored associations in this review. There were 44 publications (from 37 studies) that have explored relationship between parental education level and developing ECC. Majority of the studies explored associations between maternal level of education and caries, whilst nine studies explored relationship between both parents' level of education and caries. Twenty-seven studies (34 publications) were cohort studies and the remaining ten were case control studies, therefore, the findings of the cohort studies will be only reported in this section.

The studies used different indices to define parental level of education, where majority of the studies measured education level using number of years spent in formal education, however, there were eight studies that measured education level of parents using certificate level achieved (Cabral et al., 2017, Hong et al., 2014, Mattila et al., 2000, Peltzer, 2014, Skafida and Chambers, 2018, Thitasomakul et al., 2009, Un Lam et al., 2017, Warren et al., 2016). The minimum number of years observed in the studies that assessed duration of formal education was four years and all of the studies that showed significant relationships concluded that children of mothers with less number of years of formal education were at higher risk of developing caries than their counterparts with more number of years. Similarly, all the eight studies that measured level of education using certificate level achieved demonstrated that children of mothers with lower school certificate achievement had a higher risk of developing caries than those whose mothers had a university or postgraduate degree. The characteristics and findings of the studies are shown in Appendix 20.

3.4.2.6 Socioeconomic Status (SES)

Although, association between area-level deprivation and caries has been discussed above under place of residence, this section explored relationship between individual-level socioeconomic status and caries in children. A total of thirty-two papers were identified, where twenty-two of the studies were cohort studies and ten case-control studies. It is also found that the authors identified SES risk factors using one of two definitions: twenty-eight of these papers defined socioeconomic status as monthly or annual family income, and the remaining four studies defined SES using a predetermined social class index.

Results from studies that used class system

There were six studies that explored social class, two cohort studies and four case-control studies. These studies used different definitions to determine social class in all of them which are highlighted in Table 3. In Riberio et al study, classes of the participants were determined using an SES index that was developed by the Brazilian Association of Research Companies. The main components of the index were the possession of durable goods and the educational level of the head of the household (Ribeiro et al., 2017). Similarly, Wagner et al., (2017) study used the Brandenburg social index, which is mainly based on parents' education and employment (Wagner and Heinrich-Weltzien, 2017).

Table 3: Types of class system used

Author	Study Design	Country	Classes System	Components of the index
Peres et al., 2017	Cohort	Brazil	5 Classes	Family income quintiles (Brazilian real)
Ribeiro et al., 2017	Cohort	Brazil	3 class system	Possession of durable goods, educational level
Wagner et al., 2017	Case-control	Germany	3 class system	Parents' education and employment
Agarwal, 2011	Case-control	India	5 classes	Not discussed
Menon et al., 2013	Case-control	India	5 classes	Not discussed
Sridevi et al., 2018	Case-Control	India	Undefined	Combination of education, income and occupation

Four out of the six studies, three from India and one from Brazil, found a significant association between family social class and caries (Agarwal et al., 2011, Menon et al., 2013, Peres et al., 2017, Sridevi et al., 2018). They showed that children in lower class families were at a higher risk of developing caries than children in higher social class, and the risk could be almost 3 times the risk of children in higher class families [2.6 (1.7 - 4.1)] (Peres et al., 2017). The remaining two studies did not find any relationship between social class and caries in children. The characteristics and findings of the five studies are shown in and further details in Appendix 21 below.

Results from studies that used family income

There were twenty-one publications from 18 cohort studies in this group, therefore, the results of cohort will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. Eight out of the 18 cohort studies showed a significant relationship between monthly or annual family income and caries in children. These studies showed that children living in families with lower income have a higher risk of developing caries than those in higher income families. This risk in lower income families varied between 1.21 (1.06-1.37) in the United States and 1.98 (1.71 to 2.38) in Sweden (Fontana et al., 2011, Julihn et al., 2018). The only study that contradicted the others was the findings of Zhou et al (2012) study from China, which showed that higher family income is a significant predictor of developing caries [2.40 (1.23–4.66)] (Zhou et al., 2012). The remaining papers did not find any significant relationship between family income and caries in children. The characteristics and findings of the studies are shown in and full details in Appendix 22 below.

3.4.2.7 Social Capital

A total of nineteen publications from 17 studies were found to explore the association between social capital and development of caries in children under the age of six. Eleven of these studies were cohort studies (13 publications) and six were case-control studies. These studies are further divided into three categories that fit under two out of the five dimensions from Harper and Kelly's tool mentioned above (section 3.2.2.1). These three categories are:

I. Mothers' marital / Single parenthood status (Social networks and social support class)
II. Personal relationships to the main carer (Social networks and social support class)
III. Community engagement activities of the parents (Social participation class)

Single Parenthood (Maternal Marital Status)

Fourteen publications from 12 studies assessed the relationship between mother's marital status and developing caries in pre-school children. Nine out of the 12 studies were cohort studies and three were case-control studies, therefore, the results of cohort will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. Six out of the 11 publications (from nine cohort studies) concluded that children living in single-parent families had a greater risk of developing caries, compared to children living in two-parent families. The studies that presented odds ratio showed that the odds of developing caries in children living in single-parent families varied between 1.36 (1.02 - 1.85) and 3.3 (1.5-7.6), relative to children living in two-parent families (Mattila et al., 2000, Piva et al., 2017). The characteristics and findings of the fourteen studies are shown in Appendix 23 below.

Personal Relationships

This category explores the relationship between the personal relationships that the child develops and caries, and four studies were identified in total. Two were case-control studies and the other two were cohort studies. Ohsuka et al., found that children who grew up living with grandparents (compared to those children living with their parents) were at a higher risk of developing caries (Ohsuka et al., 2009). Similarly, Seow et al., found that a significant relationship exist, albeit on univariate analysis only, between a child's family status and caries (Seow, 2009). In Seow et al., study, they found that children living in an original family (i.e. both parents living together) were at a lower risk of developing caries, when compared with children living in a "step family" or those living in a sole-carer family ($p<0.001$) (Seow, 2009). Finally, Matilla et al., study demonstrated that children who make lots of social contacts (measured by lots of playmates and spends more times playing with playmates) were at a much lower risk of developing caries, compared to children who don't make lots of social contacts (Mattila et al., 2000). Whereas Ismail et al., did not find any relationship between caregivers' biological relationship and developing caries in children (Ismail et al., 2008). The characteristics and findings of the four studies are shown in Appendix 24 below.

Community Participation

There was only one study in this category that assessed mother's participation in the community and developing caries in children (Tavares et al., 2008). This study is a case-control study that was carried out in Brazil, and it showed that no significant relationship exists between the two. The characteristics and findings of this study are shown in Appendix 25 below.

3.4.3 Characteristics of PLUS Factors (i.e. additional risks factors identified)

As highlighted above in section 3.2.2.1 that the definition of PLUS factors was retrospective, that is, research student was only able to define “PLUS” risk factors after data extraction. According to Nasser et al, “PLUS” covers other possible risk factors which may indicate a disadvantage, such as age, sexual orientation, and disability (Nasser et al., 2013). Hence in this thesis, all other (non-behavioural) risk factors identified in retrieved articles, that is, risk factors that are not based directly on the oral health behaviour of the main subject (children under six), and did not fit under any of the PROGRESS categories, were discussed under the PLUS factors. After assessing all the studies in this review, a total of eleven other risk factors that indirectly affect caries outcome in children were identified under PLUS factors, which research student categorised under “Three Ps” as follows (Table 4):

1. Perinatal factors
2. Parental factors
3. Payment-related factors

Table 4: Summary of papers identified that explored Other Factors (PLUS) and caries in children

Perinatal factors				Parent-related factors						Payment-related factors
Birth weight	Type of Delivery	Pre-term birth	Child's birth order	Maternal age at child birth	Maternal Oral Health Behaviour	Age at start of tooth brushing	Family Caries History	Smoking-Families	Parent supports child with OH	Dental Insurance
Kay	Boustedt	Sridevi	Dabawala	Yokomichi	Mattila	Thitasomakul	Limaa	Wigen	Nunes	Brickhouse
Hong	Sridevi	Nirunsittirat	Mahesh	Warren	Grytten	Wong	Plonka	Boustedt	Yokomichi	Fontana
Yokomichi		Seow	Tiberia	Wigen	Werneck	Limaa	Fontana	Ju	Huntington	Huntington
Lima		Limaa	Yokomichi	Mattila	Agarwal	Lulić-Dukić	Grytten	Tanaka	Mahesh	Nunn
Slade		Ju	Huntington	Peres	Lima	Nunes	Ismail	Yokomichi	Werneck	Tiberia
Sridevi		Schroth	Menon	Tavares	Östberg	Dabawala	Boustedt	Plonka	Pinto	Werneck
Seow		Zhou	Ohsuka	Lam	Smith	Wagner	Mattila	Julihn	Nishide	
Sanders et al		Rocha	Werneck	Julihn	Tavares		Nishide	Leroy	Schroth	
Zhou		Yokomichi	Leroy	Ju			Pinto	Peltzer	Dabawala	
Wigen		Wigen	Nishide	Menon			Warren	Schluter	Lima	
Nirunsittirat			Peltzer	Peltzer			Peltzer	Warren	Wagner	
Peltzer			Wigen	Piva			Rocha	Rocha		
Thitasomakul			Sanders et al	Rocha			dos Santos Pinto			
Fontana				Sanders et al						
Rocha				Schluter						
Ju				Skafida						
				Smith						
				Zhou						

3.4.3.1 Perinatal factors

Twenty-seven studies explored perinatal factors and developing caries in children, and these factors are discussed under four sub-groups of SDH exposures: birth weight, type of delivery, pre-term birth and child birth order. Sixteen of these studies assessed the relationship between birthweight and caries, thirteen studies explored a child's birth order and ECC, ten studies explored the effects of premature birth on ECC, and finally three studies on the mode of delivery of child. The findings of the studies in each of these categories are highlighted in more depth below.

Low Birth Weight and Caries

Sixteen studies explored the association between birth weight of the child and development of caries in children under the age of six, twelve were cohort studies and four case-control studies. Therefore, the results of cohort will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. The results showed that there were only three out of the twelve cohort studies that demonstrated significant relationship between birthweight and developing caries (Hong et al., 2014, Kay et al., 2010, Yokomichi et al., 2015). The study conducted by Hong et al. is the only one that demonstrated, by the age of 5 years, low birth weight children are 1.7 times at risk of developing caries [Odds Ratio is 1.70 [p=0 .04]] on their primary second molar, compared to children with normal birth weight (Hong et al., 2014). Kay et al., found that risk of developing caries increased per every 100 g increase 1.08 (95% CI: 1.03, 1.13), and Yokomichi et al., found birthweight less than 2500g to be protective, with a relative risk of 19% (CI, 11–28, P < 0.001). The characteristics and findings of the studies are shown in Appendix 26.

Child's birth order

Thirteen studies were identified under this category. Six of these studies were cohort studies and the remaining seven were case-control studies. Therefore, the results of the cohort studies will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. The exposure of interest in this category is the assessed child's birth order, where five studies concluded that a child's birth order is a significant risk factor for developing caries in children. Two out of the five studies that found an association demonstrated that higher birth order (second born and above) was associated with higher risk of developing caries (Sanders and Slade, 2010, Yokomichi et al., 2015). The remaining three studies did not find any association between birth order and developing caries in children. The characteristics and findings of the thirteen studies are shown in Appendix 27 below.

Prematurity and Caries

Also discussed under perinatal factors is the risk of baby born prematurely and caries, which was defined in majority of the studies as babies born before 37 weeks. Ten papers were identified under this category, which are seven cohort studies and three case-control studies, therefore, only cohort studies are reported in this section. Of the seven cohort studies, there were only one study that found a relationship between prematurity and caries (Nirunsittirat et al., 2016). Their study found that prematurity is a protective factor against developing caries. The characteristics and findings of the ten studies are shown in Appendix 28 below.

Mode of delivery and Caries

Also discussed under perinatal risk factors is the type of delivery of the child, i.e., Normal delivery vs C-section. There were three studies that explored this variable, two of these studies were cohort studies and one case-control studies (Boustedt et al., 2018, Fontana et al., 2011, Sridevi et al., 2018). The results of both cohort studies showed conflicting results; Boustedt et al., study showed children delivered through C-section to be more at risk of developing ECC, whilst Fontana et al., study found children delivered normally (vaginal delivery) to be more at risk of ECC. The only case-control study here did not find any relationship between mode of delivery and ECC. The characteristics and findings of the studies are shown in Appendix 29 below.

3.4.3.2 Parent-related PLUS factors

There were six factors discussed in this category: maternal age at child birth, maternal oral health behaviour, the age that the parent started brushing child's teeth, parental support with routine oral hygiene, family caries history and finally, parental smoking history. The results from these factors are extracted below in relation to the risk of developing childhood caries.

Maternal age at childbirth and risk of caries

This category explores the relationship between maternal age at child birth and risk of caries in children. Eighteen studies were identified in this category: fifteen cohort studies and three case-control studies. Therefore, the results of the cohort studies will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. Seven cohort studies showed that there is a significant relationship between

mother's age and caries in the child (Julihn et al., 2018, Mattila et al., 2000, Peres et al., 2017, Un Lam et al., 2017, Warren et al., 2016, Wigen and Wang, 2011b, Yokomichi et al., 2015). Six of these studies showed that children of younger mothers were at a greater risk of developing caries, with an odds ratio varying between 1.9 (1.2–3.2) and 5.0 (1.3-19.8) in cohort studies carried out in Norway and Finland respectively (Mattila et al., 2000, Wigen and Wang, 2011b). Two studies showed that caries risk was higher in children of older mothers (Julihn et al., 2018, Un Lam et al., 2017). Un Lam et al., found that there is a significant risk of children developing ECC if their mothers are 34 years and older 3.07 (1.15-8.20), however, Julihn et al., study found higher risks in children of mothers that are either younger than 25 years 2.06 (1.89 to 2.24) or older than 34 years 1.26 (1.16 to 1.36) (Julihn et al., 2018, Un Lam et al., 2017).

Finally, it should be noted that Julihn et al., study is the only study that assessed the risk of a child developing ECC in both younger (< 25 years) and older (>34 years) women. Their study showed that children of mothers less than 25 years were more than twice at risk of developing ECC [2.06 (1.89 to 2.24)], and equally, children of mothers greater than 34 years were also at higher risk of developing ECC [1.26 (1.16 to 1.36)]. The characteristics and findings of the studies are shown in Appendix 30.

Parents' Oral Health Behaviour and risk of caries

This category explores the relationship between parental oral health behaviour and risk of caries development in their children. Eight studies were identified in this category, five case-control and three cohort studies. The exposure of interest under this category is parent's

oral health behaviour, which is defined in this thesis as regular tooth brushing or regular preventive dental attendance.

Four studies explored relationship between tooth brushing in parents and caries, and the remaining four studies explored association between preventive dental attendance of the parents and caries in children. Five out of the eight studies demonstrated that there was a significant relationship between parents' oral health behaviour and developing caries in children (Agarwal, 2011, Grytten et al., 1988, Lima et al., 2016, Mattila et al., 2000, Werneck, 2008), three of which showed that children of mothers who rarely or never attend clinics for preventive oral care reasons were more at risk of developing caries themselves than children of parents who attend. Werneck et al. presented an odds ratio, which demonstrated that children of mothers who did not attend dental clinic within the last year were about 4.1 times at risk of developing caries (OR = 4.10 (1.78–9.43) p=0.001) (Werneck, 2008). The remaining two studies were significant on bivariate and univariate analysis only (Grytten et al., 1988, Lima et al., 2016).

Similarly, two studies showed that irregular tooth-brushing pattern of the parents is a risk factor of developing caries in children (Agarwal, 2011, Mattila et al., 2000), and Mattila et al., study showed that children whose mothers rarely brushed their teeth were 2.2 times at risk of developing caries [OR= 2.2 (1.4-3.5), p=0.001]; whereas, children of fathers with irregular brushing pattern were 1.4 times at risk of developing caries [OR = 1.4, 95% CI 1.1-1.7, p = 0.002] (Mattila et al., 2000).

Tavares et al. and Ostberg et al., did not find any relationship between tooth brushing frequency in parents and caries in children (Östberg et al., 2017, Tavares et al., 2008),

meanwhile, smith et al. was the only study that did not find any relationship between parents' dental attendance and caries in children (Smith et al., 2002). The characteristics and findings of the studies are shown in Appendix 31 below.

Age child start tooth brushing

The age when a child under six years starts tooth brushing has been argued as a function of the level of awareness of the parents. Seven studies explored this relationship, three were cohort studies and the remaining four were case-control. The exposure of interest in this category is the age at which tooth brushing was commenced, however, age was assessed differently in all the papers, varying between 6 months and 25 months. Five out of the seven studies concluded that children who started tooth brushing relatively late were at a greater risk of developing caries (Lima et al., 2016, Lulić-Dukić et al., 2001, Thitasomakul et al., 2009, Wagner and Heinrich-Weltzien, 2017, Wong et al., 2012). The characteristics and findings of the seven studies are shown in Appendix 32 below.

Support and supervision of routine oral hygiene and risk of childhood caries

This category explores the relationship between those children who received support for oral hygiene and risk of developing caries in children. Eight studies were identified in this category, which are six case-control studies and two cohort study, and the exposure of interest under this category is defined as children who do not receive regular parental support towards oral hygiene and preventive care. Four out of the eight studies showed that children who were not supervised, by their parents, during tooth brushing sessions were significantly at risk of developing caries, when compared with children who receive support towards their oral hygiene. Likewise, Werneck et al. study found that children whose

parents did not take them to visit a dentist had higher caries risk of developing caries [OR = 2.86 (1.23–6.65); p = 0.013]. The remaining three studies did not find any association between parental support during tooth brushing session and caries in children. The characteristics and findings of the eight studies are shown in Appendix 33.

Family Caries History

The exposure of interest under this category is the history of caries either in primary caregiver or siblings and the outcome measured in all the studies is dental caries in children. Thirteen publications from 12 studies were identified in this category, eleven cohort studies (12 publications) and one case-control. Therefore, the results of cohort will only be presented as there were more than five cohort studies in this category, as discussed in section 3.3.1. Eight out of the eleven cohort studies demonstrated that there was a significant relationship between family's caries history and caries in the child, with majority (seven out of eight) exploring relationship between primary care giver caries history and ECC. Boustedt et al., study explored relationship between caries history in siblings and developing ECC (Boustedt et al., 2018). These studies concluded that a child's risk increases significantly, if any member of the family had a history of caries. Mattila et al. and Ismail et al. both showed that this risk increases significantly as the number of carious teeth increase in either of the parents, and the odds of developing caries in the child ranging between 1.045 [1.018 - 1.072] to 2.1 (1.1–3.5) times that of children without prior family history of caries (Ismail, 2009, Mattila et al., 2000). The characteristics and findings of the studies are shown in Appendix 34.

Parental Smoking History

This category explored the relationship between history of smoking in either of the parents and the child developing caries. Twelve studies were identified in this category, eleven cohort studies and one case-control, therefore, only cohort studies are reported.

Five out of the eleven cohort studies demonstrated that there was a significant relationship between being exposed to cigarette smoking via one or both parents smoking and higher risk of developing ECC, with an odds ratio varying between 1.14 (1.00 to 1.30) and 3.0 (1.7 to 4.9) in Tanaka et al., and Boustedt et al., respectively (Boustedt et al., 2018, Ju et al., 2016, Tanaka et al., 2015, Wigen and Wang, 2011b, Yokomichi et al., 2015). The characteristics and findings of the studies are shown in Appendix 35.

3.4.3.3 Payment-related factors

Studies in this category explored relationship between risk defined as "receipt of financial support towards oral health care" and the development of ECC. Six studies were identified in this category, which are four case-control and two cohort studies. These studies were all carried out in North America, which are four from the United States and two from Canada. The authors also explored two separate, health-insurance based risks under this category, which are:

1. Total lack of access to dental health insurance and
2. Type of insurance (government versus private health insurance.

All the six studies concluded that children who do not have access dental health insurance are significantly at higher risk of developing caries, when compared to those who have dental insurance (Brickhouse, 2008, Fontana et al., 2011, Huntington et al., 2002, Nunn et

al., 2009, Tiberia et al., 2007, Werneck et al., 2008). These studies also highlighted that children on government insurance were more at risk of developing caries, when compared with children who have private insurance. The characteristics and findings of the five studies are shown in Appendix 36 below.

3.5 Discussion

The evaluation of existing frameworks carried out and highlighted in chapter 2 showed a lack of a systematic approach among oral epidemiologists to identify all possible wider determinants influencing the development of caries in children under the age of six. This review therefore set out to identify SDH risk factors responsible for developing caries in children under the age of six years, which is based on evidence from Cochrane-validated PROGRESS-PLUS. The findings of this review showed that there are so many upstream factors at population level that influence oral health behaviour and ultimately the risks of developing caries in some groups of children. For example, living in sub-optimal fluoridated and relatively rural, disadvantaged low-income neighbourhood areas were associated with higher risks of caries. Those who also live in areas with poor access to a dentist or living in relatively lower dentist to population area also tended to have a higher risk of developing caries within the first six years of life. For more than half a century, water fluoridation has been considered the most cost-effective measure for reducing caries at the community level (Jamieson et al., 2010) because of its ability to regularly promote tooth remineralisation and thus strengthen the enamel of the teeth. More recent systematic reviews conducted in 10 countries have also confirmed that water fluoridation significantly reduces prevalence and incidence of caries in children by an average of between 30% and 59% (Rugg-Gunn and Do, 2012)**.** Other studies have also documented that fluoride has the ability to slow or reverse the progression of an existing caries lesion (Aoun et al., 2018).

In spite of these benefits, there are equally public health concerns about the ingestion of excessive amounts of fluoride, which has consistently blocked the public roll out of water

fluoridation. More than 80% of the fluoride toxicity recorded are in children below the age of six (Martínez-Mier, 2011), which often manifests as dental fluorosis. However, Cochrane review on water fluoridation concluded that the benefits of community water fluoridation outweighs the possible adverse effects, where they estimated that the overall percentage of the population with dental fluorosis of aesthetic concern is about 12% (95% CI 8% to 17%) against a 35% caries reduction in children due to water fluoridation (confidence interval was not provided) (Iheozor-Ejiofor et al., 2015).

The other environmental risk factors that increases susceptibility to caries, such as, area-level deprivation, poorer access to dental care and living in rural area which ultimately lead to lower preventive dentistry uptake (Maserejian et al., 2008). Several studies on other health conditions have highlighted that area-level deprivation status of the neighbourhoods in which the child or parents live, such as number of grocery stores per geocoded area (Tellez et al., 2006), deprivation quintiles (Office for National Statistics, 2017), number of schools in area (Ghosn et al., 2017), average time to Health Centre (Ghosn et al., 2017), are determinants of health. Gorbatova et al., study also expressed that there is specifically lower or absence of caries-prevention investments in rural areas, fewer dentists per population, and lower levels of good oral health behaviour in parents and children living in rural areas (Gorbatova et al., 2012). Similarly, those living in rural areas have limited access to healthy food options (Ju et al., 2016), preventive dentistry infrastructures (Ohsuka et al., 2009), toothbrushes and fluoridated toothpaste, as well as limited job opportunities that lead to low socioeconomic status and ultimately poor health (Ju et al., 2016). Teeth were also brushed less frequently in rural children, and the rural populations valued dental health less or consider them less important, than in urban population (Mattila et al., 2000).

This review showed that child's race or ethnicity can be determined using one of these four factors: race, ethnicity, country of birth and the main language spoken at home, and ethnicity was found to be an important risk factor in caries, where caries experience in preschool children were higher in children from minority background, children whose parents were born abroad, and those children living in families where mother tongue was different from the official national language. The findings from the studies reviewed also showed that ethnic minority children are more susceptible of developing ECC because they tended to adopt dentally unhealthy oral health behaviours, such as frequent intake of sweet desserts (Gao et al., 2010, Nunn et al., 2009). Cultural differences influence dietary choices, and other studies have reported variations in the amount of refined sugar content in the diet in ethnic minority children (Church et al., 2006). Church et al., study highlighted that immigrant children eats lots of high-sugar content foods, particularly cakes and sweets, which they thought is because immigrant children are unaccustomed to the indigenous food (Church et al., 2006). Another plausible explanation is that non-indigenous families are known to be, more often than not, unemployed or employed in low pay-low skill jobs and are therefore poorer than indigenous families (Marcenes et al., 2013). The lack of financial resources has a negative influence on their oral health behaviour and diet, which then leads to the inability of the child to attend preventive dental visit or to afford healthy diet (Kim et al., 2012). There is also abundance of evidence that suggest that ethnic minority families have lower education attainment. Education, which will be discussed later, is a known risk factor of caries, where children of parents with lower educational attainment are at higher risk of developing caries. Parents' education attainment also influences annual family income and socioeconomic status (Pastore and Zylberstajn, 1996), as well as, impact on the capacity of the parents to understand printed preventive leaflets (Church et al., 2006).

Immigrant parents also experience financial difficulties, language barriers and they have fatalistic beliefs and attitudes towards their children's oral health (Gao et al., 2010, Wigen, 2011) (Werneck, 2008).

Similarly, children of unemployed parents or those whose parents have lower employment status, such as, blue-collar workers demonstrated higher caries risk than children of highly skilled parents. Type of occupation is associated with level of educational attainment (Sobal, 2001), and parents with low-status occupations are more inclined to adopting unhealthy lifestyles (Wardle et al., 2002, Ball et al., 2002). Being unemployed also leads to lack of empowerment and worsens social integration (Grundy and Holt, 2001, Ansari et al., 2003). Meurman et al. study, as well, demonstrated there is a higher *Streptococcus mutans* colonisation rate in children of blue-collar parents (Meurman et al., 2010). The plausible pathway for this observation is that unemployed parents or parents with low-status occupations are also more likely to be in stressful circumstances (Janlert and Hammarström, 2009), and Tang et al. showed that the link stress influences the ability of parents to regularly supervise routine (daily) preventive care (Tang et al., 2005). It is also reported that majority of low-status job parents are sometimes ineligible to take paid leave from work, in order to take their children to visit a dentist for preventive care, and in countries where health insurance is linked to occupation status (United States and Canada), this group of parents mostly have to pay out-of-pocket to access preventive dental care. Children of unemployed parents are also known to have limited access to healthcare information (Prakash et al., 2012). In some countries like North America, employment status is a strong predictor of care affordability index (Macdonald and Friendly, 2014), such that unemployed parents are unable to afford preventive oral health behaviours.

Gender differences in caries risk seen in some of the studies reviewed is likely due to relatively poor oral health behaviour in boys than girls (Lulić-Dukić et al., 2001), and boys are also more likely to have sweet beverages at night and less likely to begin toothbrushing early (Lulić-Dukić et al., 2001, Kuusela et al., 1999). A study carried out in 20 European countries corroborated this pattern of sugar consumption in boys by demonstrating that in all the countries, boys had more between-meal sugar products more frequently than girls did (Kuusela et al., 1999). Other researchers have also observed that in some developing countries, such as in India, a male child is the preferred and therefore more likely to be rewarded with sugary food (Gupta et al., 2015). However, one study in this review observed a higher risk of ECC in girls (Ismail, 2009), and there is a consensus among oral epidemiologists that this is possible due to the earlier eruption of teeth in preschool girls, thereby making teeth to become exposed to caries risks for longer periods of time (Al-Hosani and Rugg-Gunn, 1998, Al-Darwish et al., 2014, FDI World Dental Federation, 1987).

Parental level of education was another very highly researched SDH variable in this review, and parental educational level was generally assessed by either the number of years spent in formal education, or the highest certificate level achieved. Children of lowly educated parents have higher risks of developing ECC. Studies have shown that poorly educated parents are less likely to be interested in their health, which thus influences their oral hygiene behaviour (Tanaka, 2013), as well as their attitudes towards oral health (Saldūnaitė et al., 2014). They are also relatively less concerned about their children's oral hygiene status than highly-educated parents (Rajab et al., 2002). Parents with low education lacked awareness and knowledge of oral health, and are unable to access relevant information with regards to feeding, diet or dental care (Agarwal, 2011, Zhou et al., 2012). Low educated

parents are also known to initiate oral hygiene practices relatively late in their preschool children (Wigen and Wang, 2011b), and are less likely to choose a better nutrition path than well-educated parents (Parikka et al., 2015). However, well-educated parents are more likely to be aware of the importance of oral health (Narang et al., 2013), and also possess better cognitive abilities to understand prevention-based health information (Marcus, 2014). The other plausible mechanism of action is that educational attainment is the strongest predictor of employment and family income (Gao et al., 2010, Lima et al., 2016, Mechanic et al., 2002, Tollefson, 1989). Parents with a higher level of education also tended to have insurance from work for preventive dental care, as well as, a much better access to healthy food options (Tiberia et al., 2007).

Another important upstream factor is family socioeconomic status (SES), which is usually a function of three other closely related indices: number of years of parental education, parental occupation and parental income (Burt, 2005). This review found that children with low SES showed a higher caries risk than children from high SES families. This is likely because children from lower socio-economic group tended to have a much higher uptake of caries risk behaviours such as poor diet and lack of uptake of preventive dentistry (Vargas and Ronzio, 2006, Bae and Obounou, 2018). Equally, they have limited access to dental care because of their inability to afford preventive dental care services (Plutzer and Spencer, 2008). Other possible mechanism highlighted in other studies is that SES exerts its effects through lack of knowledge of healthy food choices and limited ability to purchase them (Marshall et al., 2007).

Single-parenthood is one of the biggest influences on childhood outcomes statistics in general and studies have shown statistically significant differences in child outcomes, that is entirely due to marital factors, start to develop as early as the age of three (Goodman and Greaves, 2010, Fisher, 2010, Nikolaou, 2012). Goodman and Greaves' study found that children born to married parents have better social and emotional development and stronger cognitive developments and the reason for these differences is because of the greater cooperative behaviour between parents such that their combined resources give them a greater bargaining power and it reduces parental stress (Goodman and Greaves, 2010). Likewise, the findings of this review showed that children living in single-parent families have a greater risk of developing caries, compared to children living in two-parent families. This can also be explained, using Goodman and Greaves argument, that marriage provides the opportunities to combine financial and time resources and also reduces stress (Goodman and Greaves, 2010). Married families possess relatively greater financial power to afford healthy foods and preventive treatments. Both parents can alternate the supervision of the child during routine tooth brushing and can also alternate taking the children for their routine preventive dental appointment.

PLUS-Factors

One of the strengths of using the PROGRESS tool is that it acknowledges that other relevant risk factors, which do not fit under any of the eight main equity-based PROGRESS factors, may exist. In this review, a total of eleven other upstream factors were identified and sub-categorised into three groups: perinatal, parental and payment-related factors.

Findings from earlier studies have highlighted that low birth weight (birth weight less than 2,500grams) and pre-term birth (children born before 37 weeks) are the two known proxy markers of deprivation during the perinatal phase (Kogan, 1995, Dolatian et al., 2014). However, majority of the publications (13 out of 16 studies) reviewed did not find any relationship between birthweight or pre-term birth and ECC. There are three possible explanations for the lack of significant association with ECC. Firstly, the consequences of perinatal events on tooth integrity most likely go unnoticed until later in permanent teeth, i.e., after the age of 6 years (Shulman, 2005, Kay et al., 2010). Secondly, retrospective studies have also shown that there is usually delayed tooth eruption in preterm and low-birth weight babies (Aktoren et al., 2010, Corrêa-Faria et al., 2013), therefore reducing the total amount of time the teeth is exposed to caries-related insults (Zemaitiene et al., 2016). Lastly, this lack of significant association could be because low-birth weight children are more likely to receive more medical and dental follow-ups than normal children do (Tanaka and Miyake, 2014).

Also, under perinatal group are two linked factors: birth order and family size. The impact of a higher birth order is on the overall family size, therefore, both are grouped and discussed together. Larger family size operate by negatively impacting on family resources, and some studies have demonstrated that larger families are more likely to be poorer than smaller families (Cinar et al., 2008, Libois and Somville, 2018, Osei-Amponsah et al., 2010). Parents of larger households are also more likely to be stressed and unable to adequately monitor their children's oral health behaviours (Downey, 1995).

Parent-related factors identified under PLUS-factors exert their effects indirectly on the child, and the main risk factors identified under this category include: maternal age at birth of child, maternal oral health behaviour, age at which child started tooth-brushing, family caries history, smoking families, and whether parents support child during routine oral hygiene. Caregivers' preventive oral health behaviour is regarded as the most significant indicator of oral health behaviour in children (Folayan et al., 2014). There are studies that have also shown that the precursor microorganisms for caries (Streptococcus Mutans) found in mothers also have the ability to colonise pre-dentate infants via vertical (mother-to-child) transmission (Proença et al., 2015, Niji et al., 2010). This colonisation can occur as early as the age of two months (Tankkunnasombut et al., 2009), and this evidence supports the argument that family caries history, especially that of the mother, is a strong predictor of ECC. Family caries history is another important upstream factor in this review, and it is a good proxy for parents' oral health behaviour and healthy eating (Shearer et al., 2011). ECC risk in children with family caries history can be as high as twice that of children without prior family history of caries [2.1 (1.1–3.5)].

Similarly, this review found children who did not receive support for routine oral care or preventive visits to be more at risk of developing caries. Parental supervision of tooth brushing of preschool children has a direct effect on the teeth, because it ensures that the tooth cleaning is properly carried out and teeth are free of cariogenic substances (Prakash et al., 2012). Supervision also models good oral behaviour and empowers the child to continue the good practice. Late start of tooth brushing is also a function of primary carer's oral health awareness (Proença et al., 2015). There is a strong association between mothers'

dietary intake and their children's dietary intake and habits, which can then influence caries risk in children (Wigen and Wang, 2011a, Feldens et al., 2010, Zhong, 2009).

This review demonstrated that there is an association between exposure to either environmental or parental smoking and developing ECC. Some of the links that have been considered are that smoking in the family is a proxy for poor oral hygiene habits and unhealthy diets in children (Majorana et al., 2014). Tanaka et al. study has also suggested the likelihood of nicotine having epigenetic effects, such as modulating immune function, during the development of teeth in children, which therefore lowers deciduous teeth resistance to oral bacteria (Tanaka et al., 2009).

The result of the review also found that there is a relationship between mother's age at child's birth and developing ECC, and children of younger mothers were found to be more at risk of developing caries (Mattila et al., 2000, Peres et al., 2017, Warren et al., 2016, Wigen and Wang, 2011b, Yokomichi et al., 2015). The explanation for this is that younger mothers are inexperienced in instilling good oral health behaviour in their children and are less likely to supervise their tooth brushing (Hallett and O'Rourke, 2006, Niji et al., 2010). Age is also directly related to educational attainment and occupational status, where youngest mothers in a study carried out by Niji et al. study achieved no higher than a junior high or high school education (Niji et al., 2010). Their study concluded that a young maternal age most likely indicate a lower education level and poor knowledge of oral hygiene (Niji et al., 2010).

Payment-related risk factor, which is the last category under PLUS factors, explores the impact of lack of either financial support on caries. Dental insurance is one of the common

ways of financing dental treatments, especially in North American countries and it is most times linked to parents' occupation (Flaer et al., 2011, Darmawikarta et al., 2014), where higher-income groups possess better insurance package (Srivastava et al., 2017). There is ample evidence showing that possession of a dental insurance package is directly associated with a higher probability of visiting the dentist for preventive care, which eventually reduces risk of developing caries (Srivastava et al., 2017, Shi et al., 2010), whereas uninsured children are mostly from low income families with less education (Srivastava et al., 2014), and they experience more barriers to preventive oral healthcare (Shi et al., 2010, Srivastava et al., 2017). This observation therefore demonstrates that affordability is a significant barrier to preventive oral care in children (Srivastava et al., 2014).

3.6 Strengths and weaknesses of the review

The strengths of this systematic review study are discussed in three parts: The first is that the definition of the inclusion and exclusion criteria was structured after the validated Joanna Briggs Institute recommendations. Joanna Briggs Institute approach is a rigorous and transparent method and it is a preferred method of choice when conducting systematic reviews on causality or aetiology of diseases (Moola et al., 2015). Using the Population, Concept and Context systematic approach ensured that a robust search strategy was in place and that all possible search terms relevant to the review were explored across the seven different databases that are relevant to dentistry and social sciences (Peters et al., 2015). These databases selected contain peer reviewed publications in the subjects related to early childhood caries and social determinants of health. Dental databases were included

in order to retrieve relevant papers in early childhood caries, and social science databases in order to retrieve relevant papers in social determinants.

The second strength is that the search strategy retrieved very large numbers of papers that have used different methodologies in their studies. It was initially planned to include both cohort and case-control study designs, and without time limit to my search. However, having put together the search strategy for the study, there were 15,980 potential papers identified. The first trawl through the papers showed that there were a considerable number of high-quality cohort studies that would demonstrate causation, according to Bradford Hill's theory of causality (i.e., studies that assessed children at the beginning and at the end of the study) (Schünemann et al., 2011). For this reason, the decision was made to purposely include only cohort studies (except when there were few evidence in that category) because of their ability to demonstrate temporal precedence, which is the most important criteria for evidencing causality (Mann, 2003, Schünemann et al., 2011).
The final strength of this study is that it is the first systematic review to explore caries inequalities in children under six, using a validated inequalities tool (PROGRESS-PLUS) (O'Neill et al., 2014). This PROGRESS-PLUS tool ensured that all relevant determinants of caries inequalities in children under the age of six are researched.

The main weakness is that some stages of the review were carried out by a single reviewer. Both Cochrane and JBI recommended that at least two independent reviewers should undertake eligibility screening, data extraction and quality assessment, in order to reach consensus (Cochrane, 2011, Joanna Briggs Institute, 2014). Although retrieved abstracts were screened by PhD Research student and Research Assistant, however, data extraction

and assessment of quality were carried out entirely by research student because this is part of the requirements to fulfil for the award of a PhD and there was also constraints of time and resources. This therefore has the potential of introducing selection bias in the data extraction and risk of bias assessment in the reviewed studies.

3.7 Conclusion

Synthesising evidence is an important step in knowledge translation and this is the first systematic review to explore the SDH factors that impact ECC using PROGRESS-PLUS, which is a validated equity-based framework (O'Neill et al., 2014). The results of this review have assisted in uncovering the upstream distal risk factors that impact the development of caries inequalities within the first six years of life. The findings showed, consistent with literature, an association between upstream risk factors and ECC, and this in-depth understanding of wider determinants of caries in preschool children can provide the foundation for developing ground-breaking preventive caries research, health promotion strategies and evidence-backed policies, in order to improve caries inequalities outcomes locally and across the globe. The identification of the upstream risk factors is also important in the development of conceptual caries inequalities framework which is discussed in the next chapter.

CHAPTER FOUR: DEVELOPING A THEORY-DRIVEN CONCEPTUAL FRAMEWORK FOR EARLY CHILDHOOD CARIES

4.1 Introduction

The systematic review in the previous chapter revealed the Social Determinants of Health (SDH) factors associated with caries inequalities in children. These SDH risk factors were categorised under one of the nine core SDH categories influencing the development of ECC, which are, place, race/ethnicity, occupation, education, socioeconomic status, social capital, perinatal, parental and payment-related risk factors. It is not only important to identify the risk factors when intending to halt disease progression, but also vital to conceptualise the interrelationships that these risk factors have with one another, and social epidemiologists argued that conceptualisation of the issue is the first step in tackling social issue and initiating a social change (Martin, 1968, Rosas and Kane, 2012). In disease epidemiology, conceptual frameworks are epidemiological tools that have been used to comprehend and demonstrate the interrelationships between disease-causing risk factors (Jeffery et al., 2006, Burke et al., 2005), and they are consequently useful because they provide visual representations that help to explain the mechanism of the disease process (Smith et al., 1992, Kane and Trochim, 2007).

The terms conceptual framework and conceptual model are used interchangeably in this chapter (Green, 2014, Kitson et al., 2008), and a conceptual framework is defined as a visual or narrative work that explains key factors, concepts, or variables that can be utilised to support and inform research framework (Shokouh et al., 2017). Developing a theory-based conceptual framework is important in Public Health because conceptual frameworks help health planners to understand the complex, real-life interrelationships between all the factors responsible for developing diseases (Burke et al., 2005). They also help to highlight

areas within the disease process that can be modified or halted (World Health Organisation, 2010, Newton and Bower, 2005, Williams et al., 1998), or specific entry points for intervention , in order to prevent the disease from happening and policy development (World Health Organization, 2010, Control and Prevention, 2011).

In spite of the enumerated benefits of developing and using conceptual frameworks, Newton and Bower (2005) have shown that there is a lack of an oral disease framework that reflects the complexities of social processes in oral epidemiology (Newton and Bower, 2005). They also argued that the lack of understanding of the interrelationships between SDH factors, and of their causal pathways have consequently limited research into the social determinants of oral health (Newton and Bower, 2005). The lack of effective oral health frameworks still remain an issue and more researchers have emphasised the need to develop conceptual frameworks, in order to explicate the underlying causal mechanisms in oral health (Masood et al., 2019).

Therefore, the aim of this chapter is to develop a conceptual caries inequalities framework, which demonstrates the interrelationships between SDH factors responsible for caries inequalities in preschool children. This framework will also provide the template for the development of the statistical caries risk prediction model in the next chapter, as proposed by Squires (Squires, 2014).

Chapter Plan

This chapter is divided into four sections, as follows:

1. The first section of this chapter explores existing causal theories, in order to identify all the essential attributes of causal relationships in epidemiology.
2. The second section explores and discusses common epidemiological disease modelling techniques. This will help to determine the most appropriate modelling technique available for preschool caries, with the consideration that the available source of evidence (SDH) is from the systematic review in chapter 3.
3. The third section demonstrates all the possible direct causal interrelationships, based on the á priori knowledge developed from systematic review chapter. This section provides the template for the fourth section below.
4. The final section in this chapter uses evidence from the systematic review and that on the interrelationships between these SDH factors; as well as, utilises the appropriate epidemiological disease modelling technique identified in section 2, in order to develop a conceptual caries inequalities framework that demonstrates the putative real-life relationships that exist between SDH factors and preschool caries inequalities.

4.2 PART 1: Epidemiological Theories of Causality

4.2.1 Historical account of causal thinking

Causality has been explored as far back as the ancient Greek era, and several causal theories have been documented since then (Kim, 2013). While the aims of these causal theories were similar, that is, to demonstrate the link between an exposure and an outcome, the concepts and methodology used to arrive at the conclusion were different. This is outlined in more detail below.

4.2.1.1 Ancient Greek philosophers on causation

The approach of the earliest of the three popular Greek Philosophers to causality was summarised in the Phaedo (96a–100b)(Kelsey, 2004). Socrates (470 – 399 BC) was quoted as saying "taking as my hypothesis in each case the theory that seemed to me the most compelling, I would consider as true, about causes and everything else, whatever agreed with this, and as untrue whatever did not so agree" (Kelsey, 2004, Schultz, 2015). Meanwhile, Plato (428/427 or 424/423 – 348/347 BC), a student of Socrates, was known as the first Greek philosopher to formally define causality with the profound statement, in *Timaeus* 28a, that " all that becomes must needs become by the agency of some cause; for without a cause nothing can come to be" (Owens, 1955). Subsequently, Aristotle (384-322 BCE) shed more light into the theory of causation stating, in his books Physics and Metaphysics that there are four causes (or explanations) needed to highlight causal pathways (Falcon, 2008). These four causes are known as Aristotle's theory of four causes, which includes: the material cause, the efficient cause, the final cause and the formal cause (Physics II.3, 194b23-195a3) (Allmark, 2017, Kim, 2013).

4.2.1.2 Deductive Reasoning Era and Causation

Aristotle's view on causality introduced the use of logic processes that consist of more than one statement to arrive at a conclusion. He is also considered the father of deductive reasoning (Maidanskaya et al., 2017). Deductive Reasoning is a logical process reasoning that is based on one or more statements (premises), such that if the premises are true, then the reasoning will be regarded as valid (Woodcock, 2014). This type of reasoning is a top-down logic reasoning that goes from general principles that have already been confirmed to be true, to a true and specific conclusion (Zhang, 2010). History has it that Aristotle started documenting deductive reasoning as far back as the 4th century BC (Evans et al., 1993), and he provided this classic example below to explain deductive reasoning:

> *"All men are mortal.*
>
> *Socrates is a man.*
>
> *Therefore, Socrates is mortal" (Russell, 1913).*

In his example above, often referred to as syllogism, Aristotle argued that because a general premise already established that "all men are mortal" (major premise), and that "Socrates is a man" (minor premise), consequently, we can confidently conclude that Socrates is mortal (conclusion).

Weaknesses of Deduction

One major weakness of deductive reasoning is that sometimes, the major or minor premises may not be true, and, therefore, all conclusions based on such weak premises will consequently be false. However, this form of reasoning is still employed to solve logic and critical thinking problems globally (Hughes and Lavery, 2015).

4.2.1.3 Inductive Reasoning Era and Causation

Deductive reasoning continued as the methodology for making causal inference until the 13th century when both Roger Bacon (1214 - 1284) and later Francis Bacon (1561 - 1629) introduced another form of reasoning known as Inductive Reasoning (Atkinson, 2016). Although it was Roger Bacon that laid the foundation for inductive reasoning, it was Francis Bacon who advanced the idea and included it as part of the scientific method (Miller, 2015). Francis Bacon is therefore regarded by some as the father of inductive reasoning (Atkinson, 2016), whilst others regard him as the father of empiricism (Rossi, 2013).

Inductive reasoning involves using a logical process, in which multiple premises of existing knowledge or observations, all believed true or found true most of the time, are combined, to reach a specific conclusion (i.e., reasoning moves from specific instances into a more generalised conclusion) (Kalaiselvi, 2016, Elgersma, 2017). Most often, the conclusion arrived at is mainly based on probability (Hacking, 2001), and it is currently assumed that almost all scientific reasoning is based on inductive reasoning (Atkinson, 2016). An example of inductive reasoning provided by Goodman is found below (Goodman, 1965b, Harman, 2002):

1. Every emerald that has ever been observed is green,
2. Therefore, all emeralds are green (Goodman, 1965a)

In summary, the main difference between inductive and deductive reasoning is that deductive reasoning starts with a premise and inductive reasoning starts with a conclusion.

Weaknesses of Induction

Although inductive reasoning is found useful in scientific research, however, there are problems associated with this way of thinking (for reaching conclusion), which were highlighted by the 18th century Scottish philosopher David Hume (1711-1776) (Kim, 2013). Hume's problem with induction was that even after one has established the premises (experience of the operations of cause and effect) of an inductive argument, it still never guarantees that the conclusions made from those premises are true, i.e., (inductive) reasoning could not be justified on the basis that the premise was accurate in the past (Heit, 2000).

One of Hume's famous quotes to support his stance is that "men are not impelled by any reasoning or process of the understanding, but rather from custom or habit. Custom, then, is the great guide of human life" (Stafford, 2010). He went further stating that, "From causes, which appear similar, we expect similar effects" (Holyoak and Morrison, 2005).

For his work, Hume was later credited to be the main representative of the empiricist approach to causation. Hume's causal thinking, similar to that of Aristotle's efficient cause, is characterised by three main factors (Hume, 1978):

(1) Contiguity, i.e., "cause and effect must be contiguous (associated) in space and time" (Schliesser, 2007)
(2) Priority, i.e., "the cause must be prior to the effect" (Schliesser, 2007)
(3) A necessary connection (Hume is quoted saying, "There must be a constant union betwixt the cause and effect. 'Tis chiefly this quality, that constitutes the relation." (Schliesser, 2007)).

In his theory, Hume considered the third factor to be by far the most important, only because this factor is that which differentiates causal from non-causal relationships (Hume, 1978). This "necessary connection" is synonymous with "direction", which is discussed later (Susser, 2001). However, the main argument against Hume's theory was that the most important factor needed to demonstrate causality ("necessary connection") is the most difficult to establish (Susser, 2001).

4.2.2 Modern Epidemiology Era (19th century onwards) and causation

The summary of Hume's criticism is that, more often than not, previous observations do not affect the *probability* of future events (Cussens, 2011). Hume's work brought lots of scepticism into science, until the 20th century when Karl Popper (1902-1994) challenged Hume's arguments. Popper's work highlighted that scientific research is not based on inductive reasoning, but it assumes everything as a hypothesis until it has been disproved (Cussens, 2011, Popper, 2005). What Popper's theory was suggesting was that science is a continuous process of proposing falsifiable theories, and newer and more rigorous scientific evidence are needed in order to falsify existing theories (Buskirk and Baradaran, 2009, Gerstman, 2013). Popper's falsification was the first known theory to divide the scientific process into two: (1) the process of developing a theory (hypothesis) and (2) the process of testing the hypothesis (the context of justification). This process is known as scientific falsificationism or Hypothetico-deductive method (Coccia, 2018, Gerstman, 2013).

It should, however, be mentioned that those theories that survive rigorous attempts at falsification are not proved, they are only being accepted pending further research (Calder et al., 1981). Theories that eventually survive "testing" should be treated as "corroborated",

and can only be held tentatively until it is disproved (Calder et al., 1981). A practical example of this is, 'if all of the swans previously observed are white, therefore, there is an expectation that all swans are white' (Popper, 1959). However, Karl Popper's falsification theory suggested that this concept remains valid until it is disproved (e.g., by a new sighting of black swan) (Gerstman, 2013).

4.2.2.1 Koch's Postulate

Meanwhile, shortly before Karl Popper's falsification theory, Robert Koch propounded what is now known as Koch's Postulates (1890). His four postulates were unarguably the first to demonstrate important causal factors when trying to establish causative relationship between a micro-organism and developing a disease (Higdon, 2017). The summary of Koch's postulate is that a causal exposure is necessary and sufficient to cause a specific outcome (Mackay and Rose, 2013). However, many critics were able to point out that Koch's postulate is weak in explaining multi-causality, especially in chronic diseases (Presidential AIDS Advisory Panel, 2001, Chaitow, 1987).

4.2.2.2 Bradford Hill's Criteria

Koch's causal theory was later improved on by more robust theories that allowed for the possibility of multi-causality of diseases. One of these is Bradford Hill's criteria that was developed in 1965. Bradford Hill's criteria consist of a set of nine criteria that can be applied to assess whether a causal relationship is likely to exist between a cause and effect (Hill, 2015). Although Hill's criteria are extensively cited in epidemiological papers exploring causal link between an exposure and an outcome (Edelstein, 2010, Thygesen et al., 2005),

researchers have exposed some of the fundamental weaknesses in Bradford Hill's concepts (Rothman, 2012, Thygesen et al., 2005). One of the cited weaknesses is that Bradford-Hill's 'strength of association' did not put into consideration that not every important component cause would exhibit a strong association with the outcome (Edelstein, 2010, MacKillop et al., 2017, Ward, 2009). Researchers were able to subsequently demonstrate that the 'strength of association' can actually be dependent on the prevalence of other competing factors in the model (Rothman, 2012, Thygesen et al., 2005). Similarly, contrary to Bradford-Hill's third criterion ("specificity"), it is now widely accepted that a single exposure has the ability to produce more than one effect (Rothman, 2012, Gerstman, 2013, Young, 2005). Finally, the presence of a dose-response relationship (fifth criterion) is not a true marker for causality, only because confounders and other biases have similar ability to produce dose-response relationships (Thygesen et al., 2005).

These weaknesses highlighted in Bradford-Hill's criteria, as well as, the failure of Koch's postulates in addressing the issue of multifactorial causal factors, especially in non-infectious diseases, like cancers and cardiovascular diseases, led into the development of Rothman's causal model in 1976. This causal model, also known as "sufficient component complexes" or "Rothman's Causal Pie", states that "most causes that are of interest in the health field are components of sufficient causes, but are not sufficient in themselves" (Higdon, 2017). Invariably, Rothman's theory demonstrated that disease does not occur, until after all the pieces ("slices" or individual factors or component causes) of the pie (causal pathway or sufficient cause) fall into place, as shown below in figure 4.1 below. Rothman's model is further discussed later in section 4.3.1.

4.2.2.3 Susser's Criteria

Finally, Mervyn Susser was able to summarise previously existing causal theories, discussed above, into what is now regarded as Susser's criteria (Susser, 1991). His theory highlighted that only three important criteria are needed to be satisfied, in order to establish causal relationships (Susser, 1991). His first criterion to fulfil in causality is to ascertain whether exposure and outcome are statistically associated, i.e., causal factor always occurs together with the outcome, and that this association is real not spurious (Susser, 1991). Having demonstrated that this is not a spurious relationship, the second criterion is that the association is time-ordered, such that exposure can be demonstrated to precede the outcome (Susser, 1991). Susser's third criterion is known as direction, which is defined as a demonstration that a change in the outcome is a consequence of change in the exposure, that is, a change in "B" is due to a change in "A" (Susser, 1991). Susser's causal theory suggested that hypothesis should be tested using these three criteria, and researchers can confidently reject an hypothesis if the three criteria are not met (Susser, 1991).

Summary of causal theories

In summary of this section, in spite of the development of several causal theories, only three causality criteria have remained consistent over time, as summarised by Susser's criteria, which are, association, temporality and direction. However, since the studies included in the systematic review chapter are all observational studies, and not experimental in design, only two of the three main criteria (association and temporality) are further explored below.

4.2.3 Association in causality

The term “association” is defined in epidemiology as the presence of a quantifiable statistical relationship between two or more factors and a disease (Fos, 2010). Several of the causal theories discussed earlier, such as, Aristotle’s efficient cause, Hume’s theory (contiguity), Koch’s postulate, Braford Hill’s and Susser’s criteria (Susser, 1991, Cohen, 2017, Hill, 2015, Schliesser, 2007, Peter, 2001), all agreed that this criterion is of major importance in causal research.

Parascandola and Weed’s study showed that two types of statistical association exist, which are, causal association and non-Causal association, depending on whether it is real or spurious relationship (Parascandola and Weed, 2001). Causal association is demonstrated when there is “a true time-ordered relationship between one event (exposure) and another event (outcome), which is the consequence of the first” (Kocabas, 2008, Vineis and Kriebel, 2006).

Similarly, in non-causal association, the relationship observed between a cause and an outcome is actually untrue or unreal (also known as spurious/false associations). These relationships are either due to chance, bias, or there is a presence of an uncontrolled confounding factor in the pathway (DiCenso et al., 2005, Glasser, 2014). Having made this important distinction between causal and non-causal association, it is therefore vital to emphasise that the term “association” should not be used to imply “causation”, as often found in several academic papers (Skupski, 2016, Hernán, 2004, Levi, 1979).

4.2.4 Types of variable relationships

It is imperative to further explore the possible types of causal relationships, in order to understand the explanations for non-causal associations. There are four possible relationships that could occur between risk factors present in a disease. These are summarised below:

I. Direct relationships - one variable 'directly' affects the other

II. Indirect relationship- one variable 'indirectly' affects the other via another variable called a “mediator” (Sung, 2012)

III. Common Cause relationship – this is also known as Reichenbach’s Common Cause Principle (1956), which states that correlation of two separate events can only occur, if and only if, there is a causal relationship between both correlated events, or there exist a third event (factor) that brings about the correlation. This third factor is known as a confounder (Reichenbachian common cause) (Rédei, 2002).

IV. Moderating relationship: These are variables (moderators) that have innate ability to affect the strength or magnitude of a relationship between two other variables, and ultimately produces an interaction effect (Cox, 2015).

The first two relationships are found in causal relationships, and the third relationship is the possible explanations for spurious association.

4.2.5 Temporality

Temporality (or temporal precedence) is defined as the validation that the effect occurred after exposure (Centers for Disease Control and Prevention, 2004, Schünemann et al., 2011). According to Rothman, temporality is the only true criterion necessary to determine

causality (Rothman, 2012). Similarly in Hume's theory, Bradford Hill and Susser's criteria for causation, temporality is found to be very important when trying to ascertain causation between exposure and outcome (Schünemann et al., 2011, Schliesser, 2007). Other researchers have equally agreed that temporality is central when making an inference from cause to effect (Thygesen et al., 2005, Fisher et al., 2008). The implication of time in causal pathways is further discussed below.

Direct implication of time on causal variables

The relative "time" that a variable crystallises (develops/manifests/shows) is equally important in causal thinking. In causal pathways, exposure variables are categorised according to the time these variables crystallised, into two main groups:

1. Ancestor variables: These are "upstream" variables that are directly or indirectly initiating (triggering) the existence of another variable (Morgan, 2013, Staplin et al., 2016).
2. Descendant variables: These are variables that are direct or indirect effects from an ancestor variable.

4.3 Part 2: Disease modelling techniques in epidemiology

This section explores existing disease modelling techniques commonly employed by epidemiologists in order to establish both the relationship and direction between causal variables. Four of these models stand out in epidemiology because they have the potential of establishing valid associations between 'exposures' and health outcomes:

1. Sufficient-Component Cause Model (Rothman's pies)

2. Counterfactual model
3. Structural Equation Modelling
4. Bayesian Networks - Graphical models

It should be mentioned that three of the four techniques only require evidence-based information for their construction; however, SEM is a quantitative modelling technique, and requires data for its construction (Hox and Bechger, 1998).

4.3.1 Sufficient-Component Cause technique

This causal model, proposed by Rothman in 1976, was developed in an attempt to account for the multifactorial nature of causation, and it uses causal pies to help readers to conceptualise causality (Wensink et al., 2014). The basis of Rothman's model suggested that a cause (disease) is made up of a several components and none of these components is sufficient, on their own to cause a disease, i.e., diseases only occur when all component causes of the sufficient causal pie are present (Parascandola and Weed, 2001). Likewise, the absence of at least one of the component causes will result in the outcome not happening (Centers for Disease Control Prevention, 2006).

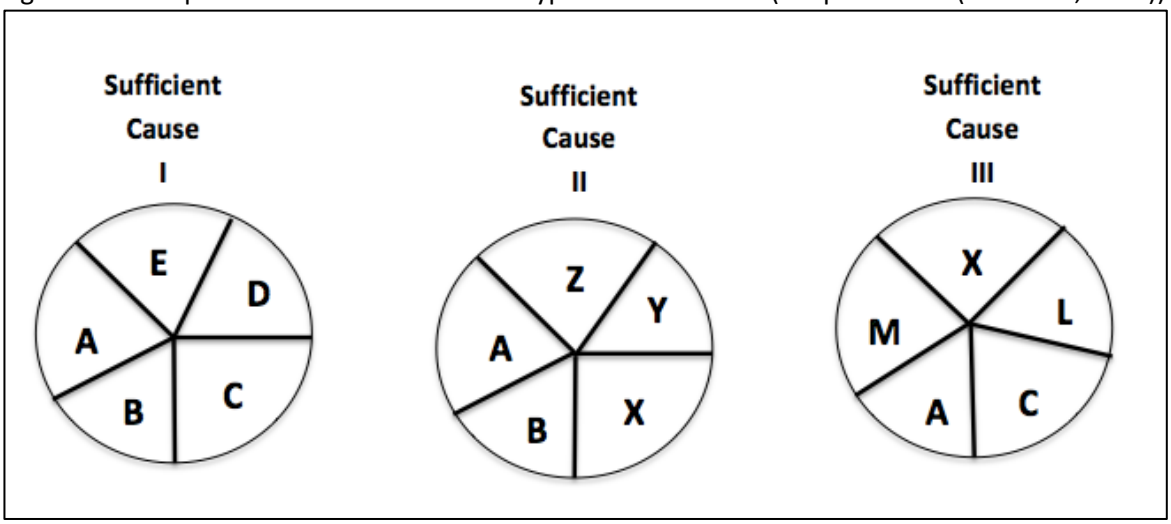
These three definitions below are essential in order to understand Sufficient-Component Cause theory. These include:

1. Component causes are "one member" or "one slice" of a set of causes that creates a sufficient cause (Wensink et al., 2014).
2. Sufficient causes ("complete pie"), which are made up of component cause ("pie slice"), are the minimal set of conditions and events that are sufficient enough for

outcome to occur. They are inevitably responsible for initiating the effect/outcome (Wensink et al., 2014).

3. Necessary cause is the "component cause" variable that appears in every pie or pathway, without which, disease does not occur (Rothman, 1976). An example is shown in Figure 5 below, where component cause "A" can be regarded as a "necessary cause" because it is found all of the sufficient causes (I, II and III). It is good to also state that the outcome (disease) caused need not be the sole result of the "necessary cause".

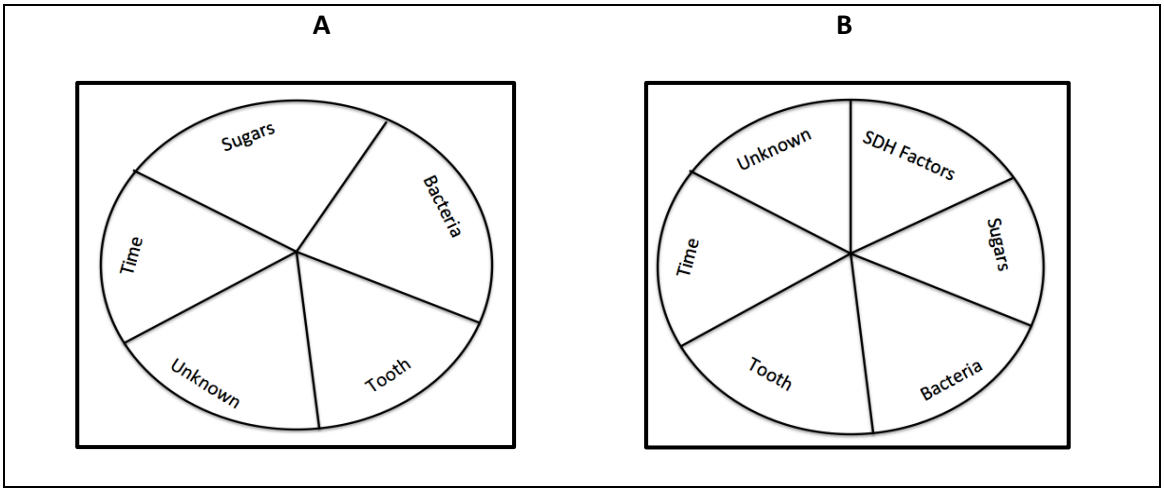
Figure 5: Conceptual scheme of causes of a hypothetical disease (adapted from (Rothman, 1976))



Translating Sufficient-Component Cause knowledge into caries research demonstrates that four necessary-cause variables are needed in caries development (Newbrun, 1983). These four are: poor diet, cariogenic bacteria, tooth and time (Newbrun, 1983, Bokhout et al., 2000), however, none of them is sufficient enough to cause caries in its entirety (Figure 6A). Similarly, in health inequalities research (such as caries inequalities), there is ample evidence that SDH factors are important players in determining general population health inequalities (Bambra, 2009, Marmot, 2005). Several caries studies equally demonstrated

that SDH is a necessary factor in caries inequalities studies (Watt, 2012, Watt et al., 2015, Aida et al., 2008), therefore, the addition of SDH modifies the model diagram A, therefore, making SDH a "necessary component cause" in caries inequalities studies (Figure 6B).

Figure 6: Difference between Caries Model (A) and Caries Inequalities Model (B) using sufficient-cause principles



There are two major drawbacks of using Sufficient-Component Cause model; the first is its failure to address temporal sequence in the model (Vineis and Kriebel, 2006). Temporality has been shown to be very important in causality models because it is the only essential factor that demonstrates that the outcome is a result of the exposure and not otherwise (Laubinger, 2013). The other weakness is the inability of Sufficient-Component Cause model to identify biases, therefore limiting its use in studies demonstrating causal relationships (Thomas et al., 2014).

4.3.2 Counterfactual modelling technique

The counterfactual theory, also known as potential outcome model, is also an important theory to consider when trying to establish epidemiological based causal inferences (Höfler, 2005, Morgan and Winship, 2014). This theory was founded on the premise that an event

"A" is the singular cause of event "B", therefore "If "*A"* had not occurred, "*B"* would not have occurred" either. A "causal factor" is a "necessary factor" in this theory, which was central to the development of randomised experiments by Fisher in 1920, and statistical inference in observational studies by Rubin in 1974 (Höfler, 2005). The counterfactual model is useful in epidemiology because of its ability to present plausible explanation for the causal pathway between exposure and outcome. However, its main weakness is that real-life scenarios are usually more complex than counterfactual statements can deal with (Weisberg et al., 2009).

4.3.3 Structural Equation Modelling

Structural Equation Modelling (SEM) was developed by Joereskog and Goldberger (1975) in an attempt to describe causal relationships between variables (Ikediashi et al., 2013). In SEM, all variables that are likely to contribute towards developing the outcome of interest are included. This model also introduces "third variables" (confounding factors), which were defined as variables that cause spurious correlations between causes and outcomes (Field, 2013).

There are two main advantages of using SEM in research. Firstly, they have the ability to categorise inputted variables into measured (or observed) variables, and latent variables (Ikediashi et al., 2013). Latent (hidden) variables are variables that are not directly measurable or observable, however, their values can be implied by the covariance between two or more indicator variables (i.e., latent variables are measured through other related variables) (Escobar, 2016). Secondly, SEM has the unique ability of providing summary

evaluation measures which ascertains the overall (global) fit of a model (Tomarken and Waller, 2005).

In spite of the enumerated advantages of using Structural Equation Modelling technique, SEM was not considered in the development of a conceptual caries inequalities model, because of its inability to describe the interrelationship between the SDH factors, without the use of data. SEM models use quantitative methods to develop their models (Tomarken and Waller, 2005), whereas, the main objective of this chapter is to develop an evidence-based conceptual caries inequalities framework, using information from the systematic review.

4.3.4 Bayesian Networks Modelling technique

Bayesian Networks are a group of probabilistic graphical models that have the ability to describe the probabilistic relationships between causes and their outcomes. This group of graphical models are collectively known as Directed Acyclic Graphs (DAGs), and they were firstly introduced to epidemiologic research in 1999 (Greenland et al., 1999). DAGs are known to be good tools able to translate researchers' à *priori* assumptions (subject-matter knowledge) into statistical models (Boerebach et al., 2013), and they also help to understand statistical relationships by highlighting the direction of association between exposures and outcomes, as well as, demonstrating possible assumptions made during causal thinking.

In addition, causal diagrams are of important benefit because of their ability to specify the minimum set of variables (i.e., mediators and moderators effect) that need to be adjusted for in order to provide an unbiased estimation of causal relationship between an exposure

and an outcome (Birungi et al., 2017). This allows DAGs to provide a quick and simple visual representation of the structural associations, therefore, making it easy for the researcher to identify and choose the covariates that should be included in the statistical model. DAGs are also judged to be better at addressing the issue of confounding, because of their ability to combine both statistical associations (quantitative approach) with *à priori* subject-matter knowledge (Al-Jewair et al., 2017).

In this thesis, Directed Acyclic Graph (DAGs) was the preferred choice due to the reasons highlighted below:

1. DAGs are simple to develop (and interpret) and the advantage they have over other disease modelling techniques is their ability to visually demonstrate temporality-based causal paths (Suttorp et al., 2014).
2. DAGs also have the advantage of addressing the issues of confounding factors (Reichenbach's Common Cause Principle), thereby helping to distinguish between real and spurious associations (Thorniley, 2015).
3. DAGs have the ability to identify which of all the exposures need to be included in the overall model, in order to accurately measure the effect of one variable on the other (Ayyagari et al., 2008).
4. Employing DAGs in modelling helps to differentiate between direct and indirect effect of the variables included in the model (Ayyagari et al., 2008).
5. DAGs provide the structure needed to test the epidemiological assumptions using empirical datasets, in order to be able to accept or reject the proposed causal pathway hypothesis (Brewer et al., 2017).

6. Finally, DAGs are also preferred epidemiological tool because they are able to visually demonstrate both “association” and “temporality” at the same time. The acyclic, uni-directional arrows in DAGs are used to demonstrate that the “exposure” is associated with “outcome”, and that, “exposure” precedes or causes “outcome” (Thygesen et al., 2005).

4.4 Directed Acyclic Graphs (DAGs)

4.4.1 Definition of cause and effects in a DAG

It is also important at this stage to highlight some definitions in DAG diagrams. For instance, a cause is a variable that influences, either directly or indirectly, the value of another variable and are often referred to as ancestors of the other variables, or parent variables (when they exert direct influence on another variable) (Staplin et al., 2016). Similarly, effects (of the cause) are defined as those variables that have been influenced, either directly or indirectly, by another ancestor or parent variable. These variables (effects) are known in DAGs as descendants, or (direct) children (of a variable exerting the immediate effect) (Staplin et al., 2016, Oakes and Kaufman, 2006). Other DAG terminologies are described below.

4.4.2 Causal Paths (i.e., direction of relationship)

It is important to understand the possible kinds of paths that exist between variables in causal diagrams. A path in a graphical model is defined as a series of non-crossing, non-repeating arrows (edges) that connects exposures with the outcomes. Although arrows are allowed to go in any direction, a path is not meant to cross a node more than once.

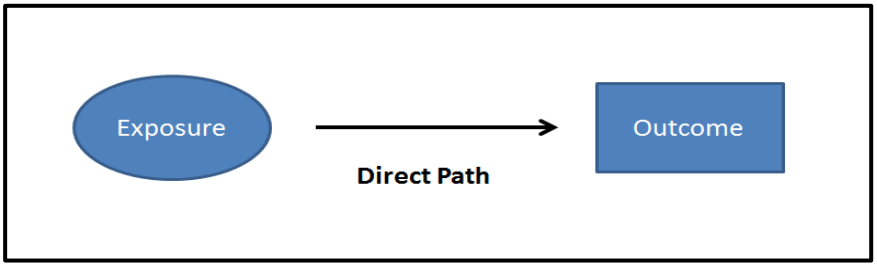
Therefore, the relationship between an exposure and outcome could have more than one path linking them (Akinkugbe et al., 2016). The presence of an arrow between two variables in a DAG diagram implies that a direct relationship exists between the two variables. Otherwise, no direct relationship exists.

4.4.3 Types of causal paths in DAGs

There are four possible types of causal paths within DAGs, which are described below:

a) Direct Causal paths: This is a directed natural path between exposures and outcomes, where no other intermediary variable is known or demonstrated between exposure and outcome (Figure 7).

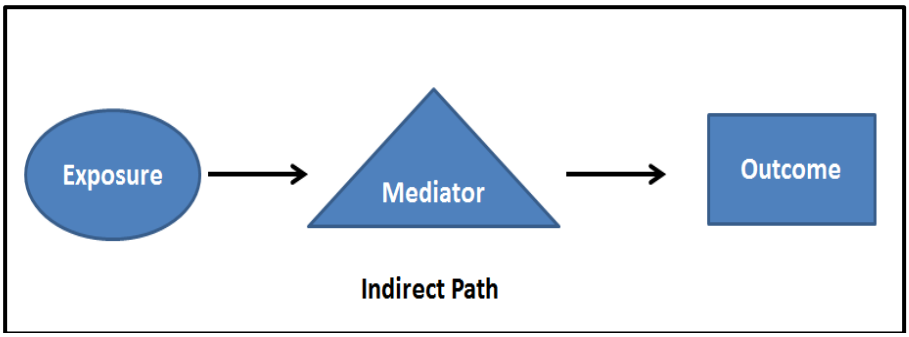
Figure 7: Direct causal path



In dentistry, a common example of direct causal effect is poor oral hygiene and plaque accumulation in the mouth.

b) Indirect Causal paths: In indirect causal paths, there is a variable in between the exposure and outcome (Figure 8). This variable in between exposure and outcome is known as a mediator, that is, a descendant of the exposure and an ancestor of the outcome (Textor et al., 2016).

Figure 8: Indirect path

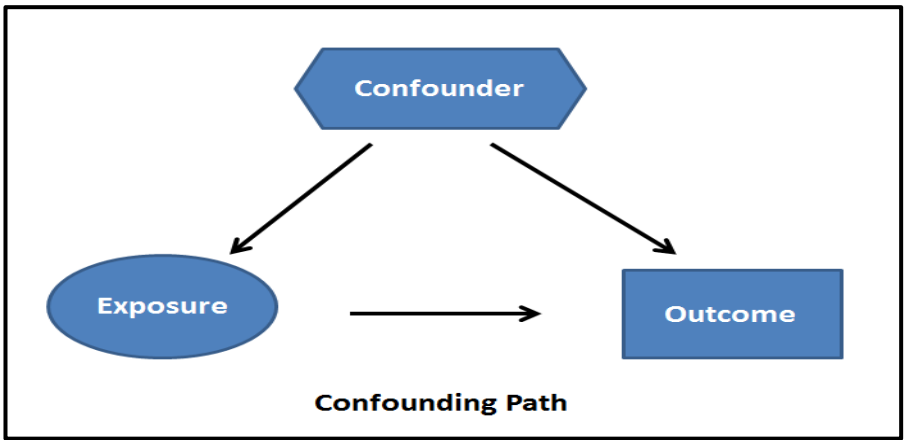


Similarly, the causal link between sugar and caries is via an indirect causal path. Existing evidence suggest that sugar is firstly converted to acid when acted upon by bacteria, and the acid developed leads to demineralisation and ultimately caries.

Sugar → Acids → Caries

c) Confounding path occurs when the natural path between an exposure and outcome contains a shared (common) cause known as a confounder (Oakes and Kaufman, 2006) (Figure 9).

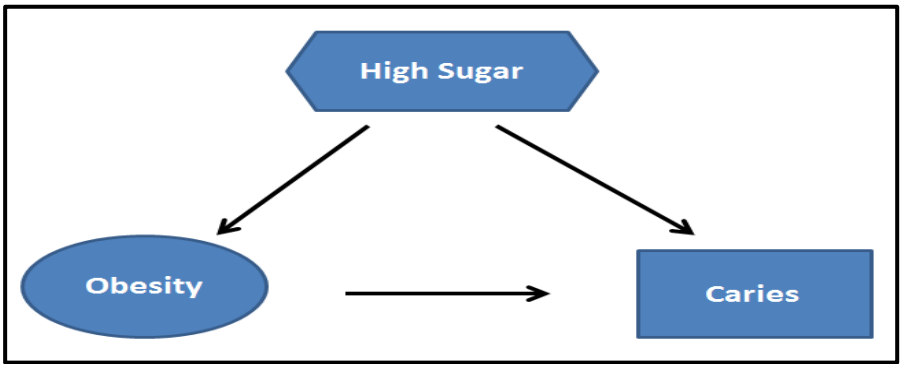
Figure 9: Confounding path



An example of a confounding path common in dentistry is the path between obesity and caries. A few studies have demonstrated that obese people are more susceptible to developing caries (Elger et al., 2019, Hayden et al., 2013, Willershausen et al.,

2004). However, further analysis of the causal pathway identified that there is a common cause of both exposure (obesity) and outcome (caries), which is poor diet. This common cause is also known as a confounder (McNamee, 2003, Oakes and Kaufman, 2006) (Figure 10)

Figure 10: Example of a confounding path in the field of dentistry



d) Colliding path is a natural path between variable ("Collider") with at least two parents (variables$_1$ and variable$_2$), that is, at least two arrowheads are "colliding" on a variable (Figure 11).

Figure 11: Colliding path

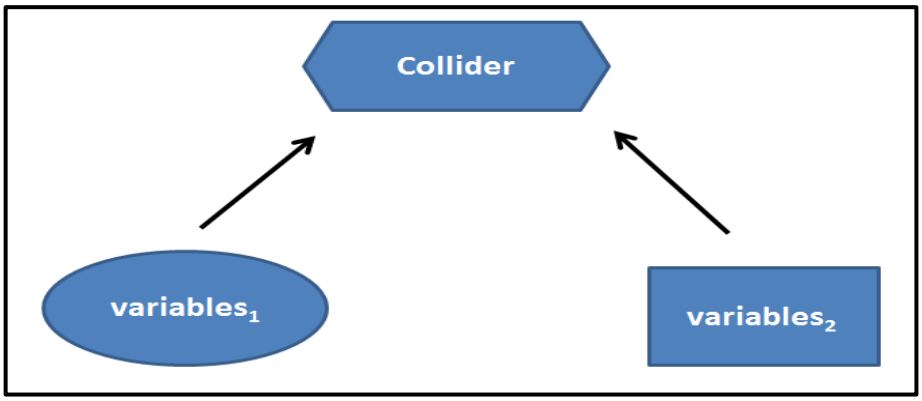
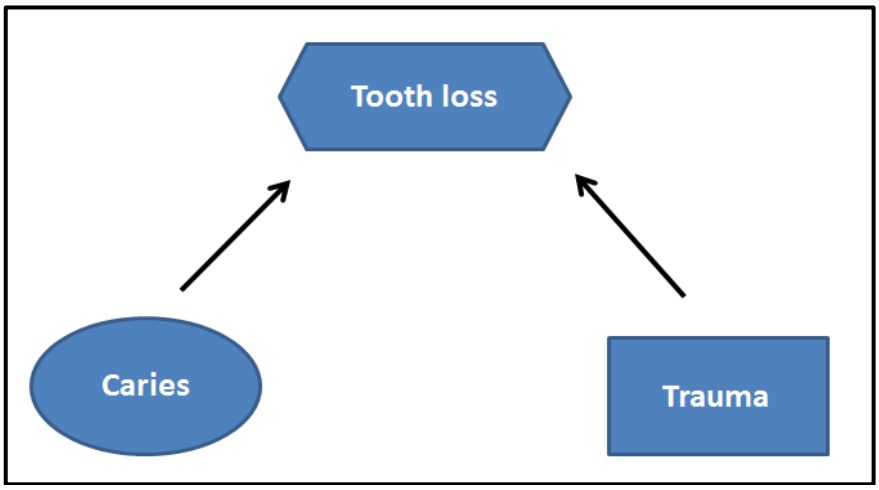


Figure 11 shows and example of a colliding path in dentistry. It is obvious from the diagram that caries is a main cause of tooth loss, but trauma is also a cause of loss. In this example, “tooth loss” is acting as a collider because two separate variables are causing (colliding on) it (Figure 12).

Figure 12: Example of a colliding path in dentistry



In causal diagrams, it is very important to identify colliding paths because colliders block the association between the variables that causes it. Therefore, adjusting, conditioning or stratifying on a collider opens up the blocked path (i.e., collider conditioning bias) and ultimately introduces a selection (or collider-stratification) bias (Merchant and Pitiphat, 2002, Cole et al., 2010). In the example described above, in which “Tooth Loss” is the collider, this factor must not be adjusted for in multivariable regression analyses, otherwise leads to collider-stratification bias (Ananth and Lavery, 2016, Siegerink et al., 2014).

4.4.4 Structure of DAGs

DAGs are made up of two main components, known as nodes and edges (Sauer and VanderWeele, 2013). Nodes are all the explanatory variables, including confounders, previously identified in the disease model, and edges are unidirectional arrows that connect two immediate variables together (Sauer and VanderWeele, 2013). These edges (arrows) are unidirectional in order to demonstrate that it is impossible for a variable cannot cause itself.

Similarly, the direction of the arrows establishes three important points; firstly, they establish that an association exists between the two variables at the other ends of the arrow, such that a change in one variable will cause a change in value of the other. Secondly, the arrows move from left to right in order to demonstrate temporality between the two variables (i.e., the future cannot cause the past), thus establishing the parent variable and descendant variable (Al-Jewair et al., 2017). Thirdly, the direction of the arrows ensures that parent variables are always evaluated first, before the descendant variables (Harel, 1999).

Furthermore, it should also be mentioned that in DAGs, the absence of an arrow between any two variable implies either of two things:

i) The first interpretation of a missing arrow is that of certainty that there is no known causal relationship between the two variables, that is, a change in one variable is not expected to cause a change in value of the other (Sauer and VanderWeele, 2013, Cole et al., 2010). For example, we know as a fact that a boy can never become a girl.

ii) The second interpretation is that there is no evidence to support that a causal relationship between the two variables exists. For example, there was absence of evidence to support (or disprove) the relationship between religion and developing caries in chapter 3.

4.5 Part 3: Evidence-Based Relationships between SDH and caries

One main criteria stated for fitting a DAG is that the relationships between the factors identified need to be based on à *priori* evidence (Merchant and Pitiphat, 2002). Whilst the systematic review conducted in chapter 3 identified the SDH factors influencing development of caries, it did not establish the potential interrelationships between these factors. Therefore, the aim of this section is to demonstrate all the putative direct and indirect causal interrelationships between SDH variables responsible for caries inequalities, as illustrated in section 4.4.3. This step of establishing à *priori* relationships between risk factors is a necessary step when attempting to develop causal diagrams (Merchant and Pitiphat, 2002) that needs to be founded on sound prior knowledge and subject matter expertise of the exposure-outcome relationship, in order for it to be considered plausible (Akinkugbe et al., 2016). This section therefore explored the rationale provided in the 67 papers included in the systematic review in chapter 3, in order to use their rationale to establish all possible direct-effect paths between all the risk factors identified. The rationale provided by the researchers were summarised and presented as a table below (Table 5).

Table 5: Table showing the direct SDH interrelationships highlighted by papers included in the systematic review

SDH Factors	**Sources of evidence**
Rural/Urban → High Sugar Intake	(Thitasomakul et al., 2009)
Rural/Urban→Local Health System	(Ohsuka et al., 2009)
Rural/Urban → Poor Oral Hygiene	(Ohsuka et al., 2009, Mattila et al., 2000)
Rural/Urban→Social Capital	(Ohsuka et al., 2009)
Access to care → Diet/Sugar Consumption	(Nunes, 2014)
Place →Water Fluoridation	(Sanders and Slade, 2010, Hong et al., 2014, Tiberia et al., 2007)
Race →diet/sugar consumption	(Mattila et al., 2000, Gao et al., 2010)
Race → Education of parents	(Wigen, 2011)
Race → Poor Oral Hygiene	(Mattila et al., 2000, Gao et al., 2010, Östberg et al., 2017)
Race → Preventive Dental Visits	(Mattila et al., 2000, Gao et al., 2010, Seow, 2009, Werneck, 2008)
Race → SES [income, Prestige, or Status]	(Gao et al., 2010, Östberg et al., 2017, Werneck, 2008)
Employment status → Insurance	(Brickhouse, 2008)
Blue Collar Job → Poor Oral Hygiene	(Slade, 2006)
Gender →diet/sugar consumption	(Lulić-Dukić et al., 2001)
Gender → Poor Oral Hygiene	(Lulić-Dukić et al., 2001)
Education →diet/sugar consumption	(Wigen, 2011, Agarwal, 2011, Kinnby et al., 1995, Dabawala et al., 2017, Menon
Education →Insurance	(Tiberia et al., 2007)
Education →Occupation	(Tiberia et al., 2007, Peltzer, 2014)
Education →Poor Oral Hygiene	(Kinnby et al., 1995, Agarwal, 2011, Dabawala et al., 2017, Menon et al., 2013,
Education →Preventive Dental Visits	(Agarwal, 2011, Werneck, 2008, Heima et al., 2015, Dabawala et al., 2017,
Education →SES	(Tiberia et al., 2007)
Education →Social Capital	(Kinnby et al., 1995)
SES →diet/sugar consumption	(Mattila et al., 2000, Fontana, 2011, Meurman and Pienihakkinen, 2011, Nunes,
SES →Insurance	(Brickhouse, 2008)
SES →Poor Oral Hygiene	(Mattila et al., 2000, Mahesh, 2013, Menon et al., 2013, Meurman and
SES →Preventive Dental Visits	(Mattila et al., 2000, Fontana, 2011, Gao et al., 2010, Menon et al., 2013,
Caregiver's support →Diet/Sugar Consumption	(Wagner and Heinrich-Weltzien, 2017)
Caregiver's support →Poor Oral Hygiene	(Wagner and Heinrich-Weltzien, 2017, Mahesh, 2013)

Caregiver's support →Preventive Dental Visits	(Wagner and Heinrich-Weltzien, 2017)
Single Parent →Diet/Sugar Consumption	(Wigen, 2011)
Single Parent →Poor Oral Hygiene	(Wigen, 2011)
Single Parent →Preventive Dental Visits	(Wigen, 2011)
Single Parent/divorce →Place	(Wigen, 2011)
Child's birth order →Diet/Sugar Consumption	(Huntington et al., 2002, Warren, 2016)
Child's birth order →Poor Oral Hygiene	(Huntington et al., 2002, Dabawala et al., 2017, Mahesh, 2013, Warren, 2016)
Child's birth order →Preventive Dental Visits	(Huntington et al., 2002, Dabawala et al., 2017)
Child's birth order →SES	(Warren, 2016, Dabawala et al., 2017)
Day care attendance →Child Oral hygiene	(Mahesh, 2013)
Maternal age →Diet/Sugar Consumption	(Mattila et al., 2000)
Maternal age →Education	(Mattila et al., 2000, Tavares et al., 2008)
Maternal age →Maternal and Child Health	(dos Santos Pinto et al., 2016)
Maternal age →Poor Oral Hygiene	(Tavares et al., 2008)
Maternal age →Preventive Dental Visits	(dos Santos Pinto et al., 2016, Tavares et al., 2008)
Maternal oral health behaviour →Child Oral diet	(Wigen and Wang, 2011b, dos Santos Pinto et al., 2016)
Maternal oral health behaviour →Child Oral	(Grytten et al., 1988, dos Santos Pinto et al., 2016)
Insurance →Access to care	(Brickhouse, 2008, Nunn et al., 2009)

4.6 PART 4: Development of Early Childhood Caries Framework

4.6.1 Stages in developing caries framework diagram

Factors influencing oral health do not act singly, but they exert their impact in a more complex manner (Lee and Divaris, 2014, Watt et al., 2014). Further evaluation of the interrelationship between the risk factors identified in the systematic review in chapter three revealed two important characteristics. Firstly, was that these variables were hierarchical, that is, the variables exist naturally in three main clusters; which are:

i. Risk factors that affect child directly

ii. Risk factors that affect child indirectly via the parents and primary care givers

iii. Risk factors that affect the entire population or community as a whole. These factors are also known as contextual variables, which include, home, neighbourhood, community and region of residence characteristics (Dahlgren and Whitehead, 1991, Fisher-Owens, 2007).

These risk factors and their categories are summarised in the Table 6 below

Table 6: Hierarchical classification of ECC risk factors

Child-related	**Parent-related**	**Population-related**
Age	Age child start tooth brushing	Water Fluoridation
Race/Ethnicity	Parental Smoking History	Urban-Rural
Gender	Support of routine oral hygiene	Area-Deprivation
Child's birth order	Family Caries History	Region of residence
	Parent's Oral Health Behaviour	Access to care/dental insurance
	Single Parenthood	Community participation
	Household family size	Local Health System
	Child living with family	
	Parental Education	
	Maternal age at child birth	
	Employment Status	
	Employment type	
	Socioeconomic Status	

Similarly, as discussed in section 4.2.5, the second observation was that SDH variables influencing ECC exhibited temporal relationships with one another. Using the evidence provided from the eligible papers in the systematic review, the effect of temporality was ascertained by aggregating all the variables in the Table 5. All the variables without "parents" were identified and categorised as "ancestor group" risk factors, and the remaining variables (risk factors that have been caused by another risk factor) are categorised as "descendant group" risk factors. Ancestor group risk factors are therefore defined in this study as the most distal risk factors in ECC framework that have no evidence of being caused by any other variable, but they have the ability to influence the development of other ECC risk factors, i.e., descendant variables. This categorisation is summarised in Table 7 below.

Table 7: Table showing categorisation of SDH influencing ECC

Ancestor (SDH present at birth)	**SDH Factors - Descendants**
Gender	Access to care
Race/Ethnicity	Access to dental insurance
Child's birth order	Age child start tooth brushing
	Access to Water Fluoridation
	Area-Deprivation
	Birth Weight
	Child living with family
	Education
	Employment Status
	Employment type
	Family Caries History
	Household family size
	Maternal age at child birth
	Mode of delivery
	Parent's Oral Health Behaviour
	Parental Smoking History
	Prematurity
	Single Parenthood
	Socioeconomic Status
	Support of routine oral hygiene
	Urban-Rural

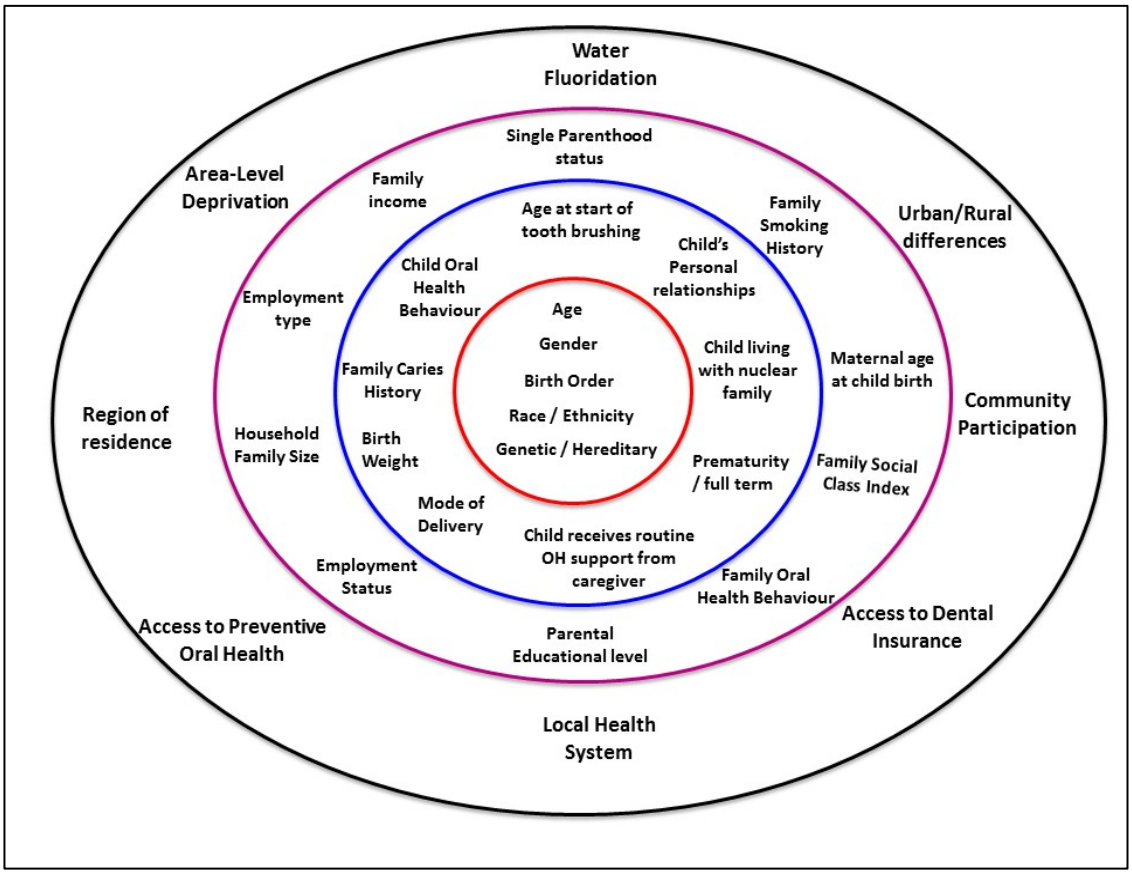
In summary, this section showed that risk factors influencing caries have two main relationship with one another; which are hierarchical and temporal. However, considering the number of the SDH risk factors identified from the systematic review and the possible complexities of the relationships between these SDH, it will be challenging to develop a single framework that demonstrates both temporality and hierarchy at the same time. Multilevel model will be useful to understand the possible areas of policy intervention at population level as emphasised by Dahlgreen and Whitehead (Dahlgren and Whitehead, 1991), and the temporal model will help to highlight the time of crystallisation of the exposure. Therefore, two sets of frameworks were developed, in order to account for both the hierarchical structure of the risk factors, as well as, to highlight the temporal relationship between them (Licaj et al., 2012).

4.6.2 Multi-Level Caries Model

The multilevel model developed in this section, shown in Figure 13 was based on the multi-layered SDH proposed by Dahlgreen and Whitehead (Dahlgren and Whitehead, 1991). The SDH risk factors identified in the systematic review were then mapped to the layers of Dahlgreen and Whitehead model. Risk factors within the red ring are the factors that have already crystallised at birth, and they include age, gender, child's birth order and race/ethnicity. Although genetics and hereditary factors are not SDH, they have been included in this domain in order to account for other non-SDH factors that influences caries development at this level. The risk factors within the blue ring layer are child-related factors, such as: age the child started toothbrushing, child oral health behaviour, whether child regularly receives support with routine oral health care, family caries history, whether child lives with nuclear family and child's personal relationships. This second layer also

include perinatal factors like mode of delivery, birth weight and full-term birth or premature birth. The third layer, the purple ring, includes parent-level risk factors such as: Employment type, family income, family social class index, single parenthood status (especially of the mother), family smoking history, maternal age at the birth of the child, family oral health behaviour, parental level of education, employment status and household family size. Finally, the fourth layer, the black ring, contains information on the living and working conditions that influence ECC and has factors like: water fluoridation, urban and rural differences, community participation, local health system, access to preventive oral health initiatives, region of residence, area-level deprivation and access to dental insurance (relevant to children living in North America countries). The final multi-level diagram is shown in Figure 13.

Figure 13: Conceptual (Multilevel) caries inequalities model for preschool children



Implication of the Multilevel Model

Theory-driven multilevel models are advantageous because they provide useful information on the possible areas where equity-based interventions and policies can be applied in order to reduce caries inequalities in children under the age of six (Griffith et al., 2016). They also enable the identification of possible health barriers at individual, parental and at population level. For instance, implementing prevention at Level 1 (red ring) is reasonably challenging because the majority of the exposures here are quite difficult to modify (Van Dyke and Dave, 2005, Poulter, 2003), whereas, the risk factors in other levels (levels 2 to 4) are best modified using equitable policies and strategies.

4.6.3 Developing a temporal-based framework using DAGs

As highlighted in section 4.4, the methodology for the proposed temporal-based ECC framework diagram was developed using directed acyclic graphs (DAGs). Although the use of DAGs for causal diagrams in dental research has been advocated, evidence suggest they are yet to be widely embraced (Akinkugbe et al., 2016). The process of developing the framework is iterative and the DAG used in this thesis was constructed using the statistical software package available in the public domain called Dagitty (www.dagitty.net).

4.6.3.1 DAG Caries Framework Methods

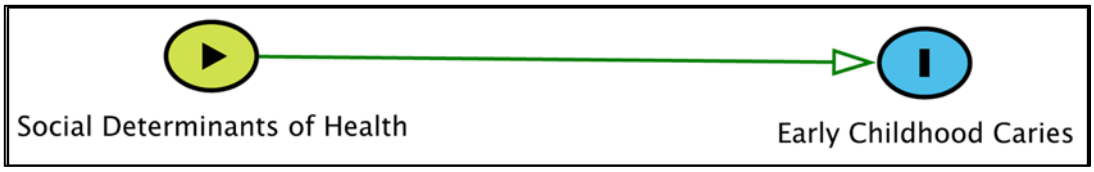
In DAGs, the presence of a unidirectional arrow between two variables suggests that a causal relationship exists between those two variables, and conversely, the absence of an arrow in a DAG means that no causal relationship exists between the two variables, therefore both variables will be treated as independent of each other (Pearce and Lawlor,

2016). Secondly, variables that lie between the exposure (SDH) and the outcome (Early Childhood Caries) were treated as a mediator (blue circles) (MacKinnon, 2012, Sung, 2012).

Model 1: Exposure to Outcome

Janice Moores (2016) highlighted that the simplest cause and effect framework is: **Cause → Outcome** (Morse, 2016), therefore, the first step during this modelling was to demonstrate (draw) the overall research question of this thesis, which is Social Determinants of Health responsible for the development of caries inequalities. The exposure here is SDH and outcome is early childhood caries (Figure 14).

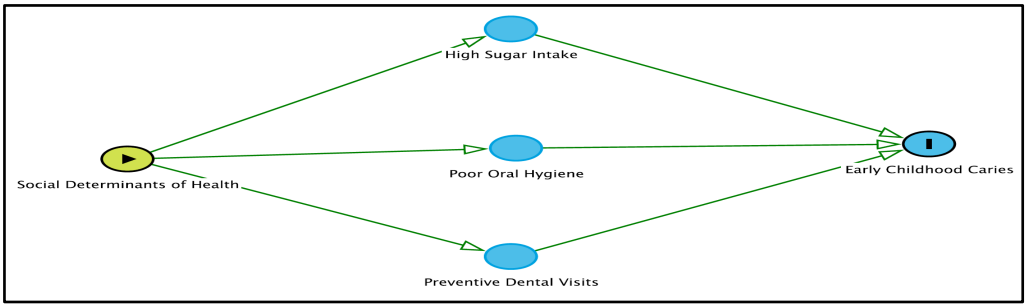
Figure 14: Model 1 demonstrating overall direction of exposure (SDH) and Outcome (Caries)



Model 2: Mediators of dental caries

However, there is vast evidence from literature that caries risk factors exert their effects via three main behavioural pathways (mediators) (Duijster, 2015), which are, poor diet or high sugar intake (Lorber et al., 2017), poor oral hygiene (Lorber et al., 2017) and lack of routine preventive visits (Heima, 2015, Chi et al., 2014). This á priori knowledge was then incorporated into the new framework diagram in Figure 15.

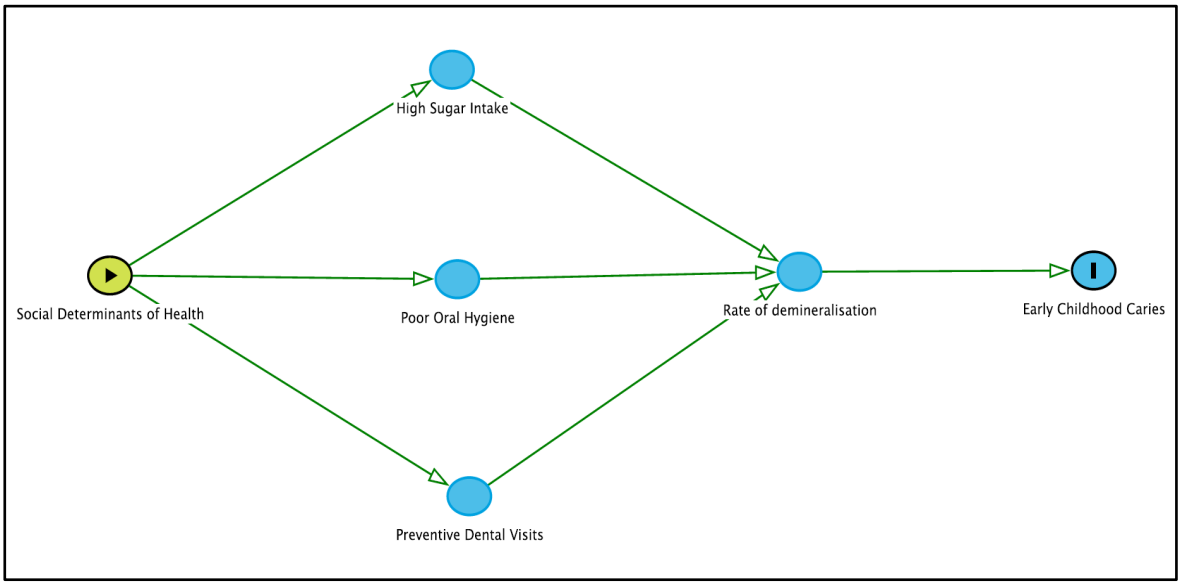
Figure 15: Model 2 highlighting Exposure (SDH), Mediators and Outcome (Caries)



Model 3: Demineralisation and remineralisation (see-saw process)

Levine et al., in their study in 1977 alluded to the fact that the mechanism of dental caries is very complex, and that every tooth goes through a never-ending process of demineralisation (destruction) and remineralisation (repair), known as Levine "see-saw" mechanism (Levine, 1977). However, caries only occurs when rate of demineralisation surpasses repair (Featherstone, 2008, Selwitz, 2007). The diagram below (Figure 16) therefore incorporates this information on demineralisation into the framework.

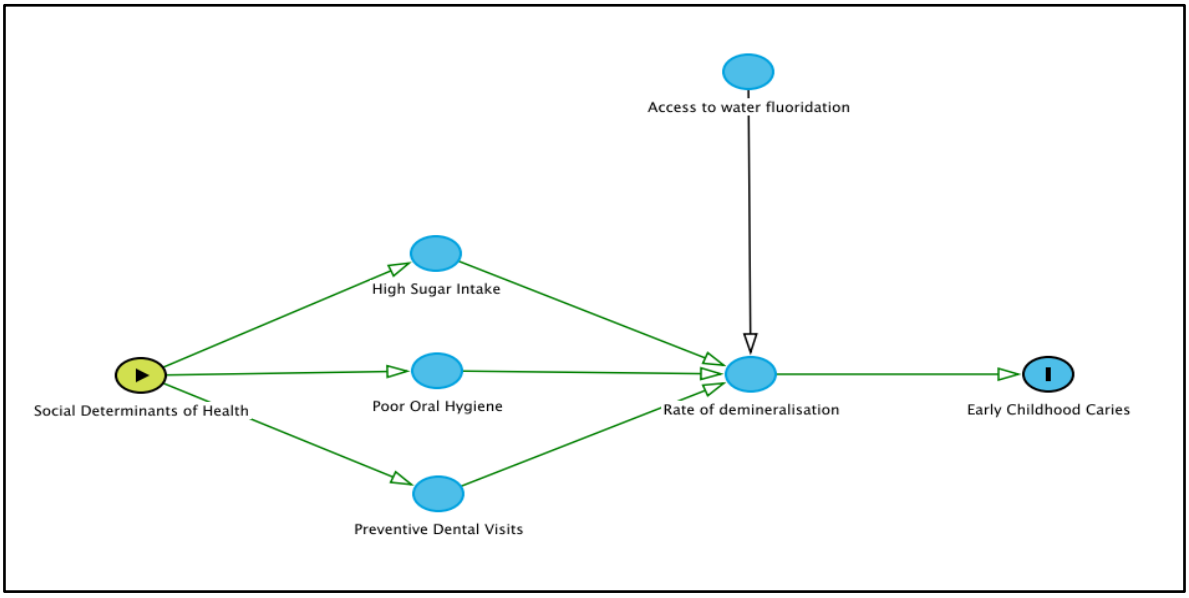
Figure 16: Model 3 showing relationship between Exposure (SDH), Mediators and Outcome (Caries)



Model 4: Exposure to water fluoridation

The addition of water fluoridation further improves the information on the risk factors influencing caries in the previous framework (Figure 17). According to series of systematic reviews, fluoride in water influences caries development by preventing demineralisation and helping with repair, i.e., remineralisation (National Health and Medical Research Council, 2017, Iheozor-Ejiofor et al., 2015, Pizzo et al., 2007, Peckham and Awofeso, 2014).

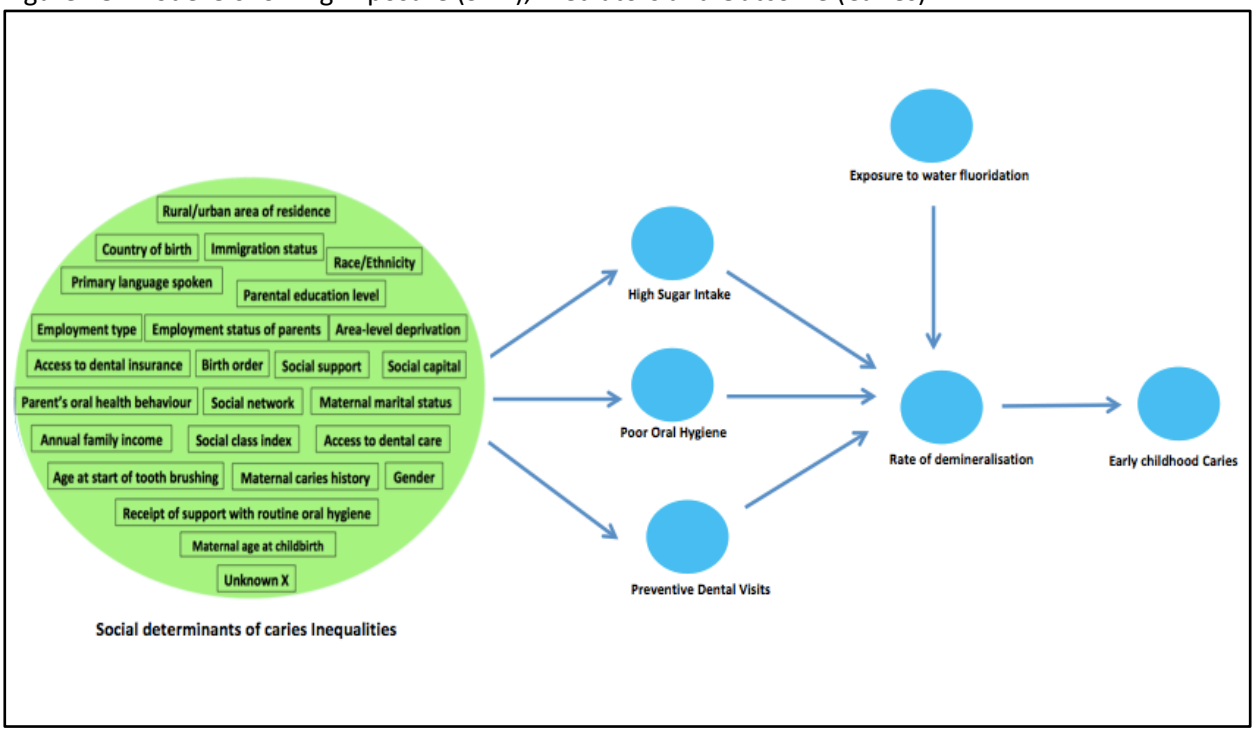
Figure 17: Model 4 showing relationship between Exposure (SDH), Mediators and Outcome (Caries)



Model 5: Precursors of SDH

Prior knowledge from the systematic review chapter showed that SDH is a combination of several risk factors (O'Neill et al., 2014). Therefore, Figure 18 highlights all the important SDH factors that are capable of influencing caries inequalities in children (large green circle), with the assumption that these SDH factors collectively influence caries inequalities through diet, oral hygiene or preventive dental visits.

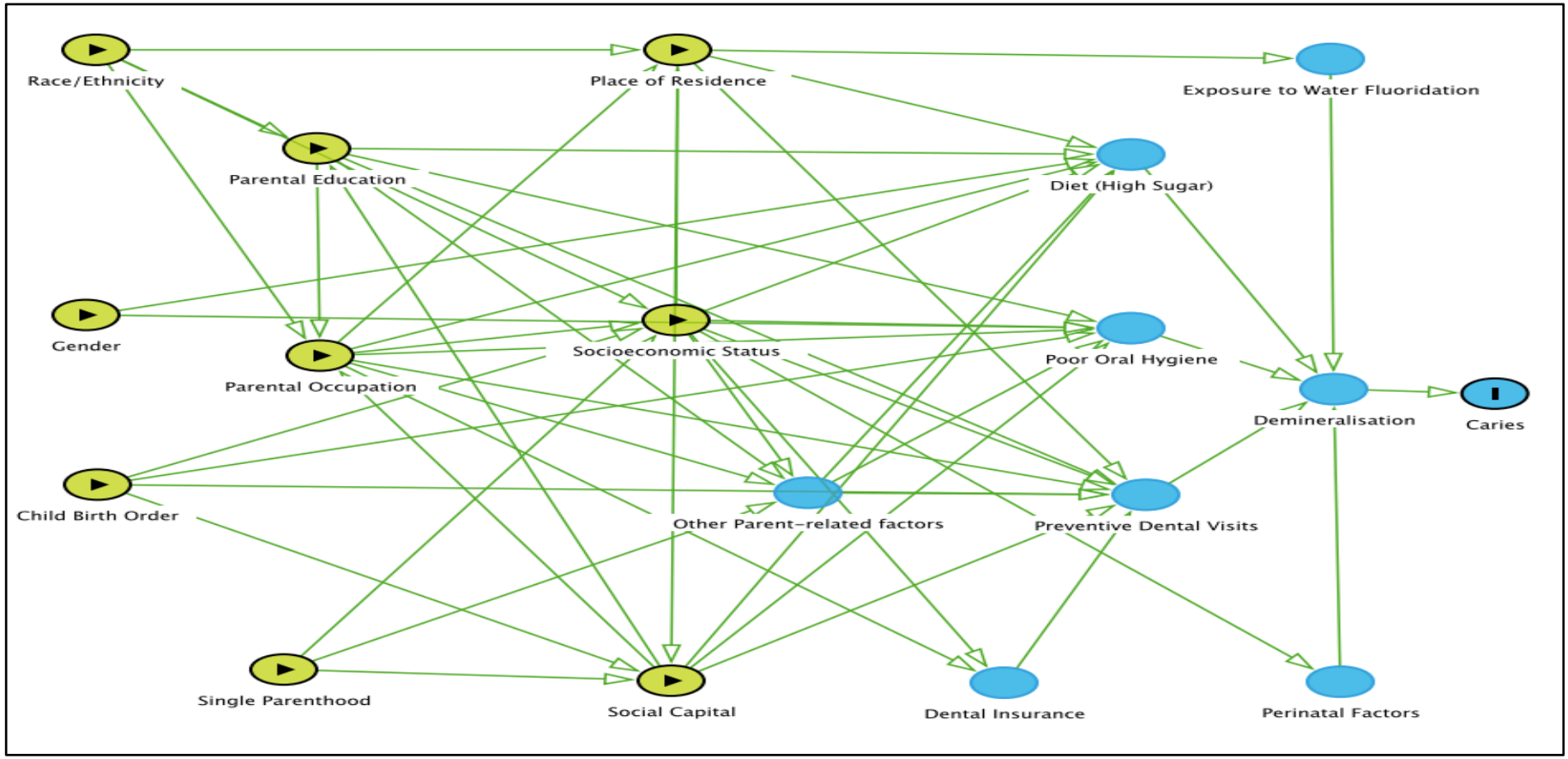
Figure 18: Model 5 showing Exposure (SDH), Mediators and Outcome (Caries)



Model 6: Complex (direct and indirect) relationships between SDH variables

Model 5 above is a simplified way of demonstrating how SDH influences ECC, but in reality, there are several direct and indirect interrelationships (causal links) between these SDH factors (Anne et al., 2017). Therefore, this final framework (Figure 19) uses the prior knowledge of the causal links, summarised in Table 5, between SDH risk factors influencing ECC development in preschool children. The final conceptual caries inequalities framework developed is shown below in Figure 19, and it emphasises that the SDH influencing caries inequalities in preschool children have a very complicated relationship with one another.

Figure 19: Final proposed conceptual framework demonstrating temporal relationship between ECC risk factors



Starting from the most distal (left) SDH factors; for example, there is peer-reviewed evidence that Race/Ethnicity can influence parental education and occupation, as well as, place of residence (Gao et al., 2010, Östberg et al., 2017, Werneck, 2008). Similarly, the DAG also showed that gender influences diet and SES status (Lulić-Dukić et al., 2001), and child's birth order directly influences family income, oral hygiene status, preventive dental visits and social capital (Huntington et al., 2002, Dabawala et al., 2017, Mahesh, 2013, Warren, 2016).

4.6.4 Discussion

Conceptual frameworks are epidemiological tools used to comprehend and demonstrate the interrelationships between disease-causing risk factors. They also provide visual representations that can help to explain the mechanism of the disease process, therefore highlighting specific entry points for interventions and policy development (World Health Organization, 2010, Control and Prevention, 2011). Epidemiologists have also argued that developing a conceptual disease framework is "an inevitable à priori process" in disease modelling, because the framework structure developed guides other stages of risk modelling (Squires et al., 2016). This chapter therefore used SDH information obtained from the systematic review and causal theories to propose a conceptual caries inequalities framework, which helps to improve the understanding of the caries pathway in children under six years. This thesis also demonstrated the two main types of relationships that SDH risk factors have with one another, which are temporal and hierarchical, therefore, leading to the development of two separate conceptual frameworks.

The temporal (DAG) framework developed was able to visually demonstrate the causal pathways between all exposure variables and ECC in a temporal sequence, and equally demonstrated the direct and indirect causal pathways, including the mediators from exposures to the outcome. The four most distal SDH exposures identified were: race, gender, birth order and single parenthood. These are variables with no prior evidence that they have progressed from another SDH risk factor, and the remaining SDH variables are offspring (direct and indirect) of one or more of the ancestor variables. There were also four main mediators

identified in the DAG, which are: diet, oral hygiene, preventive visits, and demineralisation. There is also prior evidence that water fluoridation has direct influence on tooth demineralisation, and therefore acts as one of the mediators through which a child's "place of residence" exerts its caries effects (Chi et al., 2014).

The second framework (i.e., population-based multilevel framework) is the hierarchical conception of how all the risk factors identified in the systematic review influence the development of ECC. This multilevel framework demonstrated three possible (modifiable) policy intervention areas that are important, in order to reduce the inequalities in ECC. This framework showed that factors influencing the development of ECC are expressed at individual, family and societal levels, which is in agreement with Dahlgren and Whitehead's SDH framework and Fisher Owen's caries framework (Dahlgren and Whitehead, 1991, Fisher-Owens, 2007).

The main strength of these conceptual models is in the methodology used to develop the frameworks, where both were based on prior knowledge from a systematic review of longitudinal studies. As previously emphasised, longitudinal studies were purposefully selected because they are the only group of studies that have the ability to demonstrate association, direction and temporality at the same time, which are the three main criteria for determining causality (Susser, 1991). Secondly, the relationship and direction of these risk factors were determined using DAG techniques, which therefore helps to inform the process of developing causal models (Collin et al., 2018). The DAG also enabled this study to visually demonstrate

temporal relationships, including ancestor, descendants, and mediators variables (Collin et al., 2018).

Another strength emphasised is that there were two separate frameworks developed in this study, one based on temporality and the other based on hierarchy of risk factors. Separate conceptual frameworks were developed in order to emphasis the two types of association between ECC risk factors, which is in contrast to all the previous caries frameworks explored. The mapping of existing frameworks carried out showed most of the existing ECC frameworks did not include temporality in their models, except two conceptual frameworks from Fisher-Owens et al and Keyes (Fisher-Owens, 2007, Keyes, 1969). In fact, the original Keyes triad did not include temporality, until after Newbrun modified it later by adding time factor to it (Newbrun, 1978). However, neither of the frameworks from Fisher-Owens and modified Keyes framework was able to adequately demonstrate the temporal relationship, such that risk factors proceed from one ancestor variables to descendant variables (i.e. causal pathway) (Figure 20 and Figure 21). One possible reason for this omission is that it could be quite challenging to conceptualise both hierarchy and temporality in a single framework.

This study therefore emphasised that in order to replicate the real-life ECC pathway, risk factors in ECC conceptual models will need to be represented in the three-dimensional space, that is, the SDH risk factors are, firstly, associated with one another (length), and then each of the risk factors is related to another risk factor both temporally (width) and hierarchically (depth).

Figure 20: Diagram demonstrating multilevel influences involved in the development of caries. Taken from Influences on children's oral health: A conceptual model. *Pediatrics,* 120, e510-e520

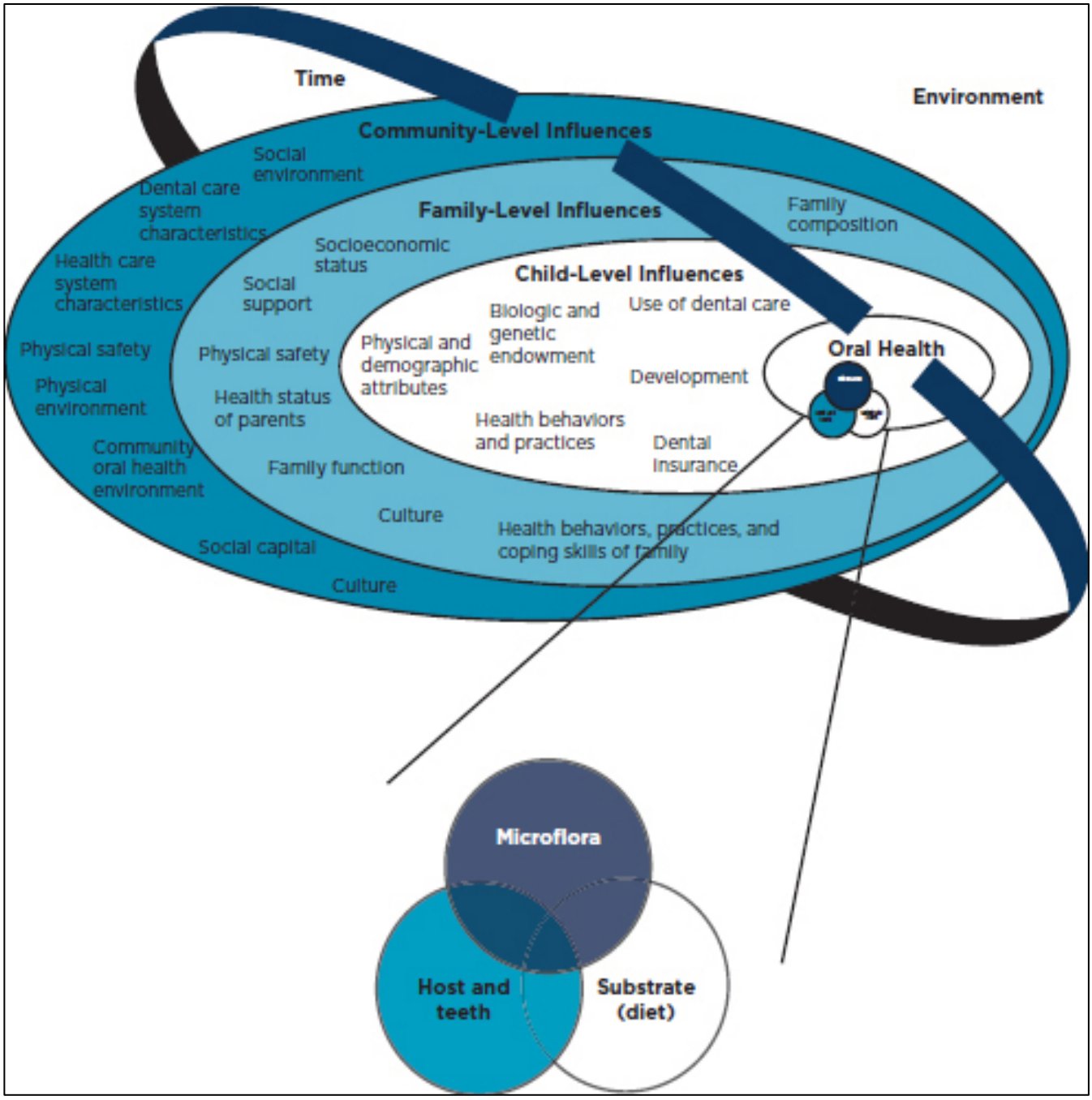
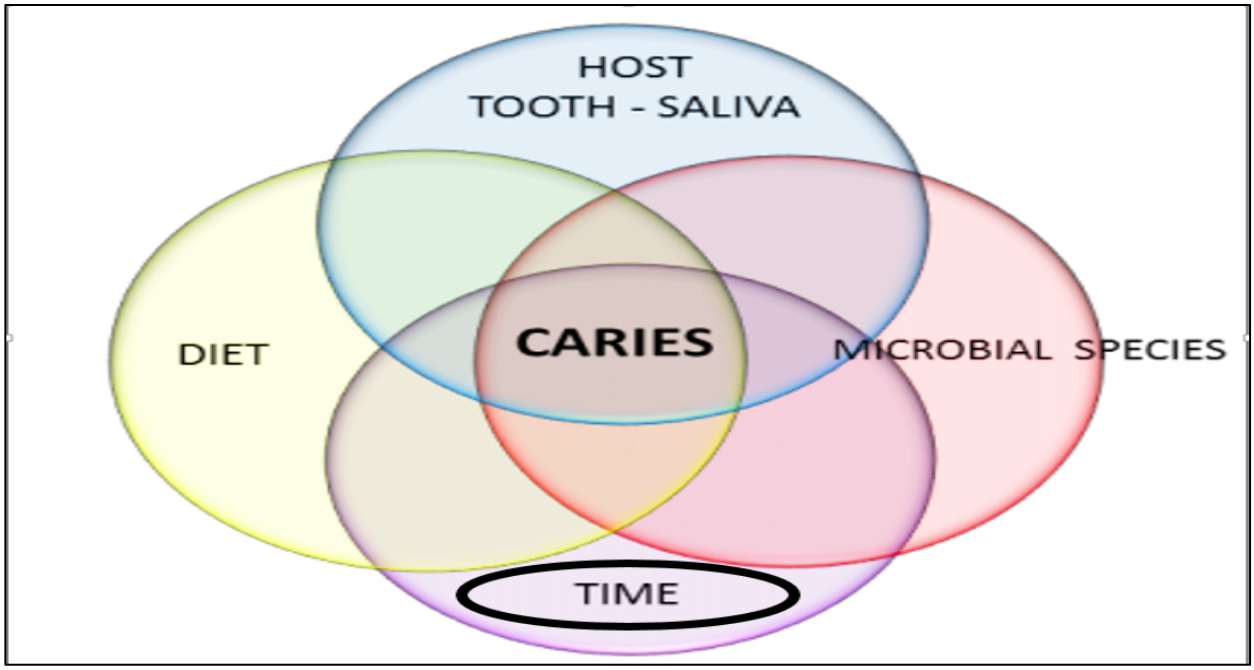


Figure 21: Diagram demonstrating modified Keyes triad.
Taken from Newbrum E. Cariology, 1st edition Baltimore: Williams Wilkins; 1978



One major limitation in this study is that the proposed DAG is very complicated and therefore will be difficult to explain. Although DAGs are employed in order to help simplify the causal pathway and to aid understanding of the disease process, but this becomes challenging in a situation where there were many inter-related risk factors, such as SDH, involved in the disease process. Another limitation is that DAG allows for sound assumptions to be made during development, therefore, the construction and interpretation of DAGs depends largely on sound à priori knowledge, in this case, a sound background understanding of basic science, biological mechanisms and disease epidemiology. Readers will also require similar sound prior knowledge in order to understand the DAG framework being presented (Williams et al., 2018).

Finally, the DAG framework developed is unable to provide information on the magnitude (size) of the association between the SDH risk factors and therefore recommended that data from empirical studies be used to test the conceptual framework after the development of a hypothesised conceptual frameworks (Walker, 2012). Hence, the next chapter will use available caries data obtained from UK dental surveys in order to see if the available data support or reject the hypothesised causal relationships highlighted in the proposed DAG framework.

CHAPTER FIVE: DEVELOPING AN INDIVIDUAL RISK PREDICTION MODEL FOR EARLY CHILDHOOD CARIES

5.1 Introduction

Two separate conceptual frameworks were developed in the previous chapter (chapter four), based on the evidence from systematic review conducted (in chapter 3), and both frameworks demonstrated the proposed pathways connecting social determinants of health (SDH) with caries. The first (multilevel) conceptual model highlighted the individual, parental and societal factors that are able to influence caries outcomes in children under the age of six; and the second (temporal based) describes the SDH risk factors in terms of the time order of manifestation; that is, ancestor variables and descendant variables.

Researchers have mentioned, however, that it is a good practice to validate conceptual models using quantitative methods (Kruijshaar et al., 2002, Walker, 2012), which was one of the gaps identified in other models discussed in section 2.2 and demonstrated in Table 8. Validation of conceptual model is a process of using data from empirical studies to test whether the conceptual model can support what it is intended to be used for, in this case, validation is using data to test the predictability of ECC in children (Liu et al., 2011).

Therefore, the primary aim of this chapter is to provide an empirical test of the conceptual caries inequalities model developed in chapter four. The use of structural equation model (SEM) is recommended, which is a powerful statistical technique that is able to simultaneously test complex interrelationships between the variables within conceptual models (Bender et al., 2017, Baker, 2007).

Objectives

1. To identify and describe the relevant datasets used and map variables in the dataset with risk factors identified in systematic review (chapter 3).
2. To develop a caries risk model that describes the relationship between SDH and caries incidence at child level.
3. To determine the predictability of the risk model for caries outcome, using appropriate statistical methods.

5.2 Methods

5.2.1 Study Design

This study is a secondary analysis of the data from the UK Children's Dental Health Surveys (CDHS) 2013. This data was used because it is the largest oral health epidemiological survey in children that is conducted across three United Kingdom countries: England, Wales and Northern Ireland. The data is also publicly available and can be accessed from the UK Data Service website, which is a national service commissioned to provide data access to the UK's largest collection of social, economic and population data for research purposes (UK Data Service, 2013). The CDHS 2013 is the latest of the series (fifth series) of national children's dental health surveys that are carried out every ten years since 1973 and the survey was based on a representative sample of children aged 5, 8, 12 and 15 years attending government maintained and independent schools in England, Wales and Northern Ireland (Masood et al., 2019). The survey was carried out using a multi-stage stratified sampling technique. This stratification was carried out regionally, in England and Wales, where 81

Local Authorities and 27 Unitary Authorities were selected in England and Wales respectively, and by a simple random sampling of schools in Northern Ireland.

One of the strengths of the 2013 CDHS dataset is that it collects clinical, behavioural, individual/household level socioeconomic indices, area level deprivation scores and attitudinal information of both the children and parents/carers, and therefore is a good resource to explore the impact of social determinants in children as required for the present study. Data collection was carried out in two separate phases, which are clinical examination phase and questionnaire phase. A technical report for this survey that includes information on all the data collected is available and key aspects are outlined below (Health and Social Care Information Centre, 2015).

CDHS 2013: Clinical examination

Clinical examination was carried out in children whose parents have consented (opted in). Seventy-five trained and calibrated dentists carried out dental examination on the children. The examination was carried out in a dental chair, under standardised dental lighting. Radiographic investigations e.g bitewing radiographs were not used, however, the trained examiners had good inter-examiner reliability, with their kappa values ranging between 0.814 and 0.928 (Health and Social Care Information Centre, 2015).

CDHS 2013: Questionnaires

A questionnaire was sent to each of the parents or guardians of each of the 5 year old children who took part in the dental examination (Health and Social Care Information Centre, 2015). This self-completion questionnaire was completed either by the parent or guardian that is most responsible for the dental health of the participating child. The

questionnaire was used to obtain additional lifestyle, behavioural and attitudinal information, such as, oral hygiene practices, parental level of education, occupation, preventive dental history etc.

5.2.2 Variables selection

Generally, there are two classes of variables in the CDHS dataset, as in other datasets: the first is known as the outcome (dependent) variables and the second class of variables are known as independent (predictor/explanatory) variables.

5.2.2.1 Definition of the outcome (dependent) variables in CDHS 2013

Early Childhood Caries is the outcome of interest in this study, and in previous CDHS epidemiological surveys, the definition of caries has been caries in dentine, which is also referred to as "obvious tooth decay" (Vernazza et al., 2016a). Therefore, the caries outcome definition used in this study is any experience of tooth decay in enamel, dentine or pulpal layers of the crown of primary teeth.

5.2.2.2 Definition of independent (predictor/exposure) variables in CDHS 2013

The definition of exposure variables includes all SDH variables mapping on to any of the SDH variables identified from the systematic review in chapter 3. These include: water fluoridation, urban-rural, area-deprivation, access to care, race/ethnicity /country of birth /language, parental employment status, parental employment type, parental education, family socioeconomic status, single parenthood, child living with family, mode of delivery, prematurity, birth weight, child's birth order, child personal relationships, household family size, maternal age at child birth, parent's oral health behaviour, age child start tooth

brushing, support and supervision of routine oral hygiene, family caries history, parental smoking history and access to health insurance - none vs government vs private health insurance. Gender was also included as an exposure, in order to test if it agrees with existing literature.

5.2.3 Methodology for dealing with missingness

It is not uncommon to have some data missing in epidemiologic studies, however if the missingness is not adequately addressed, it can lead to biased assessments or misleading interpretations (Sterne et al., 2009). Inappropriately dealing with missingness is also known to reduce the power of the study (Sterne et al., 2009). There are three main types of missingness in statistics, which are: Missing Completely at Random (MCAR), Missing at Random (MAR) and Missing Not At Random (Pedersen et al., 2017). Understanding the type of missingness in a dataset is an important step, in order to apply the most appropriate treatment technique to the missing data.

Data is said to be Missing Completely at Random when the probability of missing is independent of both observed and unobserved data, i.e., the missingness on the variable is completely unsystematic (Pedersen et al., 2017). Assessing group characteristics of both the missing and the complete cases can help to test the assumptions for MCAR. If the group characteristics are different for both groups, then MCAR assumption cannot be assumed (Garcia and Marder, 2017). The second type of missingness is known as missing at random (MAR), which occurs when the probability of missing data is independent of the missing data but dependent on observed data (Garcia and Marder, 2017). That is, MAR is assumed when it is observed that only a certain group of individuals (e.g. pregnant) have missing

values, therefore, the probability of missing data is linked to group characteristics (e.g. pregnancy). Missing not at random (MNAR) is the final type of missingness, which occurs when the probability of missing data depends on unobserved information. For example, missing data is regarded as MNAR in a situation when data on income are missing because participants with high (or low) income refused to declare their income (Garcia and Marder, 2017).

There are several ways of dealing with missing data, which depends mainly on the type of missingness. These are grouped into: (a) missing indicator method, (b) single value imputation, (c) sensitivity analyses with worst- and best-case scenarios, multiple imputation and lastly, (e) complete case analyses (Pedersen et al., 2017).

Missing Indicator Method

This is a method where missing values are either categorised into a "missing category" or set to a fixed value. The advantage of this method is that it is able to use all the available data values from the entire sample size, however, because this method is not theoretically driven, it has the likelihood of presenting results that are inaccurate (Van der Heijden et al., 2006).

Single Value Imputation

This is a method that replaces missing values with a single value, such as using the average value of the variable, carrying the last value forward, or regression-based predicted mean imputation. It is applicable to data MCAR and also has the advantage of using the entire dataset for analysis, but its disadvantages are that it overestimates the standard error. This

technique also weakens both the correlation and covariance relationships that originally exist in the data (Pedersen et al., 2017).

Sensitivity analyses

This technique replaces missing values with either the lowest or highest value observed in the dataset, and can be used when data is MCAR. Its advantages include its simplicity and it uses the complete dataset for analysis. The disadvantages are that it overestimates standard errors and results may be difficult to interpret (Sterne et al., 2009).

Multiple Imputation

Multiple Imputations can be defined as a statistical technique for addressing missing research data that resulted from participants' failure of responding to a survey, and it addresses missingness by developing multiples of plausible imputed data sets, which are subsequently averaged in order to produce valid inferences (Azur et al., 2011). Multiple Imputation is regarded as the gold standard methodology for dealing with data values that are missing at random (MAR). It also has the ability to handle both MCAR and MNAR, and its wide acceptance across the research field is because multiple imputation develops several (multiple) versions of the same data which are then combined in order to calculate the best values for the missing data. When used appropriately, multiple imputation has been shown to improve the validity of results and conclusions because it produces unbiased estimates and improves precision (accurate standard errors).

One widely cited method of implementing multiple imputations is known as Multivariate Imputation by Chained Equations (MICE). This algorithm has been heralded as one of the foremost methods for dealing with missing data, particularly because MICE has an

exceptional capability of dealing with all three possible types of missingness (MCAR, MAR and MNAR) (Resseguier et al., 2011, Galimard et al., 2016, Mirmohammadkhani et al., 2012), including missingness due to non-response (Buuren and Groothuis-Oudshoorn, 2010, Miri et al., 2016, Durrant, 2005). It also has the ability to conveniently handle up to 80% missingness (Azur et al., 2011). MICE works by using every available data and conventional predictive (statistical) methods to generate several multiple "complete datasets" (Azur et al., 2011), and finally, the missing values are imputed with the most plausible value (Azur et al., 2011). It is widely suggested that between 5 and 20 iterations are sufficient to generate accurate values of the missing data (Galimard et al., 2016).

Complete Case Analyses

Complete case analysis is a type of statistical analysis where only participants (cases) with complete information on all variables are included in the analysis, i.e., those participants with sections of their data missing are excluded from the analysis (Mukaka et al., 2016). This method is the easiest and most convenient method and can only be applicable when data is MCAR (Pedersen et al., 2017). However, it has a higher propensity of biasing the odds ratios of the remaining non-missing variables (Vach and Blettner, 1995, Azur et al., 2011). Reduction of sample size, resulting from complete case analysis, ultimately reduces statistical power and also causes loss of precision because of the large standard errors that result from the analysis.

5.2.3.1 Method used to deal with missingness in CDHS 2013

In this study, two sets of datasets were developed and analysed by the researcher from the CDHS 2013. The 2 sets of datasets put together by the researcher were done because of the complexity and the incompleteness of the available CDHS datasets. The first set of data available from the CDHS is the non-imputed, "complete at clinic only" dataset (i.e., those who completed the first part of the oral survey), which are all the children that participated in the clinical examination. This data has the largest number of complete information, i.e., after completely excluding cases (patients) with one or more missing variables in their data. It contains SDH information like gender, free school meal, country, urban-rural and region of residence, area-deprivation, ethnicity, and dental attendance of responding adult were collected. The second set of data is the data that is "complete at both clinic and at questionnaire level", i.e., those who completed both the 1st and 2nd part of the oral survey. This data is expected to be fewer, as not everyone will return questionnaires. The missing data values are imputed using MICE imputation technique described above, using the MICE package in R software (version 3.4). The benefit of this method, as stated earlier, is that it helps to maintain the original sample size available for further analysis, therefore reducing the likelihood of loss of statistical power and precision.

5.2.3.2 Dimensional reduction

The potential SDH variables were then subjected to a stepwise "forward, "backwards" and "both" elimination processes using the Akaike Information Criteria (AIC) from the stepAIC functions from the MASS packages in R software. This was important in order to eliminate

non-contributing (redundant) variables and ensure that only parsimonious risk factors, i.e., as few predictors as possible, are used to explain ECC risk model (Genell et al., 2010).

5.2.4 Data analysis approach

The data analysis approach in this section included univariate, bivariate, multivariate and multilevel analyses, which was applied to both imputed and non-imputed datasets, as recommended by Sterne et al (Sterne et al., 2009). In order to ensure transparency, Sterne et al guidelines recommended results of both complete-case analyses and multiple imputation methods should be reported, and the results should highlight areas of differences in both analyses (Sterne et al., 2009).

5.2.4.1 Univariate, Bivariate analyses and Multivariate Analysis

The initial analysis carried out was univariate analysis to describe the frequency distribution of all the explanatory and outcome variables in the CDHS 2013 and possibly identify patterns that exist within it. Univariate analysis is known to provide an overview of the dataset and also provides the foundation for subsequent further analyses like bivariate and multivariate analyses. Bivariate analyses were also conducted in order to explore the direct relationships between the independent (predictor) variables and outcome variable (caries). Multivariate analysis is a statistical analytical approach that evaluates multiple variables at the same time.

5.2.4.2 Multilevel analysis

This section also examines the role of contextual, neighbourhood characteristics in developing caries in children. This multilevel analysis was considered, owing to suggestions

from published studies that caries risk factors can also operate hierarchically (Antunes et al., 2006, Bramlett, 2010, Piovesan et al., 2011, Aida et al., 2008). Overlooking the possibilities of hierarchical risk factors may lead to the underestimation of both the environmental influence and the standard errors of regression coefficient (Maas and Vermunt, 2004). Multilevel analyses is a statistical technique that is known for its ability to separate risk factors operating at an individual level from those operating at a neighbourhood or contextual level; i.e., they can establish how much of the variations in developing ECC is due to individual or family circumstances and how much is due to area-level (e.g. Index of Multiple Deprivation level) differences (Sellström and Bremberg, 2006).

Therefore, the aim of the multilevel modelling was to separately investigate and account for the social context influence on caries risk in preschool children. Using the multilevel conceptual model developed in chapter 4 as a guide (Figure 13), a two-level logistic regression model was conducted in this chapter, in order to establish the association between caries and individual-level SDH factors, that is, nested in "area-deprivation" (contextual) factors. The first level considered were those variables at individual (child) level, which include all the first three colour rings (red, blue, purple); and the second level considered was at area-level, which are variables found in the black ring.

The final multilevel models developed were guided by the recommendations of Laflamme et al., where four sets of models were developed in turn, in order to assess the impact of neighbourhood factors on individual-level caries risk in five-year old children (Laflamme et al., 2009). The first model (baseline) is a single-level linear regression model with no predictor variable known as "null model" (model 0), i.e., a model with only an intercept and

contextual level effects, as recommended by Rasbash et al. (2009). This was developed in order to determine the baseline for the fixed effect estimates. It is advised that the fixed effects estimates of the multilevel models subsequently developed should be like those achieved from single level models (Rasbash et al., 2015), and the amount of community dependence be determined by the Intraclass Correlation Coefficient (ICC) (Maas and Hox, 2005). For binary response, ICC is calculated from variance using the equation:

$$\rho = \frac{\sigma^2{}_{\upsilon}}{\sigma^2{}_{\upsilon} + \frac{\pi^2}{3}}$$

Where, ρ = Intraclass Correlation Coefficient; $\sigma^2{}_{u}$ is the variance at neighbourhood level and $\frac{\pi^2}{3}$ = 3.2865 (Tassew et al., 2019, Browne et al., 2005). Both the measures of association (fixed effects) and measures of variation (random effects) are also reported.

The second model (Model 1) is a univariate model that involved evaluating each variable, one at a time (Laflamme et al., 2009). In Model 2, all the variables without area-level variables were included at once, and Model 3 involved simultaneously including all child-level variables with area-level variable as recommended by Sterne et al (Sterne et al., 2009). All analyses in this study were performed in R.

5.2.4.3 Developing caries risk prediction models

Developing a risk prediction model is a step further in multivariate analysis where exposures with predictive abilities are identified and modelled in order to be able to predict caries in children. This is a classification task and several classification multivariate techniques were considered, including the classic Multivariate Logistic Regression (MLR), as well as, more recent statistical techniques used in multivariate analyses collectively known as machine

learning techniques (Molina et al., 1994, Goebel and Plötz, 2019). Machine learning technique is still a relatively new in the field of dentistry, however, they are gradually gaining substantial momentum of use in biomedical studies because of their innate ability to search all possible variable relationships (Yoon et al., 2018). They are equally very effective in the identification and exclusion of redundant variables within the datasets, i.e., variables not contributing to the explanation of total variance observed in the risk model (Yoon et al., 2018). The techniques applied in this study include the traditional Multivariate Logistic Regression (MLR), as well as six most commonly used machine learning algorithms, which are, Naives Bayes (NB), Decision Trees (DT), Random Forest (RF), Conditional Random Forest (CRF), Support Vector Machine (SVM), and Gradient Boosting Machine (GBM) (Namous et al., 2018, Jamali et al., 2016). All analyses was done using statistical packages from R software (version 3.4).

5.2.4.4 Methods for assessing the generalisability of caries risk model developed

This section presents the planned steps for evaluating the generalisability of the predictive performance of all the developed models and in order to assess this metric, predictive performance of the models developed were evaluated using a statistical method known as ten-fold (k-fold) cross-validation (Refaeilzadeh et al., 2009). In cross-validation, the dataset is divided into 10 random (k-fold) subsets, and for each k-fold, the algorithms were modelled on nine-tenth of the dataset and the remaining one-tenth used as the validation dataset. Cross validation method is regarded to be a better way for assessing performance of predictive models (Bengio and Grandvalet, 2004).

Furthermore, since this is a classification exercise, the three main metrics used to evaluate the predictive models were Receiver operating characteristic (ROC) curve, sensitivity and specificity. The ROC curve is a tool (graphical plot) that is used to evaluate and compare the performance or the diagnostic ability (true positives and negatives) of predictive models (Hajian-Tilaki, 2013). That is, it assesses the ability of the model to correctly predict a positive as a positive, and a negative as a negative. Specificity in predictive modelling is defined as the proportion of observed negatives (i.e., those without the disease) that were correctly predicted to be negative, and on the other hand, sensitivity is defined as proportion of observed positives (i.e., those with the disease) that were correctly predicted to be positive (Waegeman et al., 2008). The model that predicts none out of 100 predictions right (100% wrong) will have an Area Under Receiver Curve (AUC) of 0.0, and the model that predicts all out of possible 100 predictions right (100% correct) will have an AUC of 1.0 (Carter et al., 2016). An ROC of 0.5 is regarded as not better than a simple random guessing, indicating the model is a poor classifier (Carter et al., 2016). Therefore, the higher the ROC curve, the better the model in distinguishing between “disease” (e.g. caries) and “no disease”, and the rule of thumb when choosing the best predictive model is to select the model with the highest ROC score (Carter et al., 2016).

5.2.4.5 Comparability of model with existing caries risk assessment tools

Finally, the accuracy obtained after 10-fold cross validation was ranked against the results of the four widely-used caries risk assessment tools in dentistry (Gao et al., 2013). These tools include:

1. Caries Risk Tool (CAT), developed by American Academy of Paediatric Dentistry

2. The Caries Management by Risk Assessment (CAMBRA), developed by American Dental Association
3. Computer-based Cariogram developed by Malmo University, Sweden
4. National University of Singapore model (NUS-CRA)

5.2.4.6 Evaluation of paths from proposed DAG diagram

One of the weaknesses of DAG highlighted in section 4.6.4 is that the multi-interactions between SDH variables makes the graph complex to interpret. Therefore, the aim of this section is to reduce the complexity of the proposed pathways of the SDH variables in the hypothesised DAG diagram, which was achieved in two ways: firstly, the SDH variables available in the CDHS 2013 dataset were mapped on to the proposed framework, and pathways of unavailable variables were excluded. Secondly, the bivariate associations between the remaining variables were assessed using chi-squared test in order to determine whether any two variables are related or independent of each other or not. The statistical summary of these relationships were assessed for strength of association and the statistical probability that demonstrates the likelihood of the pathway to have existed by chance (Duijster, 2014). After examining basic correlations between the SDH variables of interest, Structural Equation Modelling (SEM) was used to test the theoretical path model, using the Lavaan package in R software (version 3.4). SEM is preferred at this stage because of its ability to simultaneously test the inter-relationships between predictor variables (Duijster, 2014), and the critical sample size considered to be adequate for fitting this SEM model is 200 (Hoe, 2008). Finally, the overall goodness-of-fit measures were assessed in order to determine whether the data is able to adequately fit the proposed model. The

goodness-of-fit measures that was used is the comparative fit index (CFI) and Root Mean Square Error of approximation (RMSEA). CFI values greater than 0.90 and RMSEA less than 0.06 are indicative of a good model fit (Hooper et al., 2008, Hoe, 2008). A model path was accepted when all the goodness-of-fit measures criteria were fulfilled, and paths that did not improve the overall fit were dropped (Hooper et al., 2008, Hoe, 2008).

5.2.5 Ethical Considerations

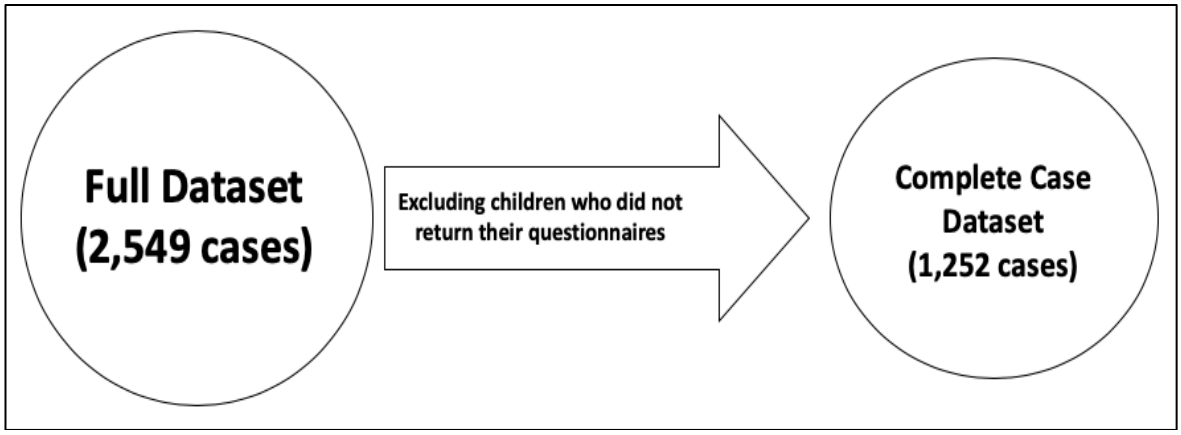
The Health and Social Care Information Centre commissioned the Child Dental Health survey (CDHS) 2013. Participation in the survey was voluntary and it obtained informed consent from a parent or guardian of a 5-year-old, before any examinations are conducted. This dataset used in this study was anonymised data; therefore, no ethical approval was required. The data is stored and easily accessed from the UK Data Service, which is funded by the Economic and Social Research Council (ESRC).

5.3 Results from CDHS 2013

5.3.1 Mapping of CDHS 2013 to Proposed Model

In the CDHS 2013 dataset, the overall number of five-year old that had clinical examination in this study, after excluding 8, 12, 15-year old children, was 2,549, however, only 1,252 participants returned their questionnaires Figure 22.

Figure 22: The two sets of data in this study



Similarly, there were 989 possible independent information collected on each child examined, consisting of clinical, behavioural and socio-demographic information. Each of these variables were later classified into two broad groups: SDH and Non-SDH variables, and a total of 945 non-SDH variables were excluded. Subsequently, the 44 remaining SDH variables were further sub-grouped into four different sub-categories: demography, socio-economic, behavioural-related and "others" sub-groups. Variables categorised under demography included ethnicity, sex, country of residence, urban or rural residence and region of residence etc. Socioeconomic variables included eligibility of child for free school meal, school type (independent or not), parents' employment type and employment status. The Index of Multiple Deprivation (IMD) criteria, categorised into five groups (quintiles) that

ranges from least deprived to most deprived, was used to establish area-level deprivation. Behavioural-related groups were mainly: routine oral hygiene and dental attendance pattern of child and parents etc; and “others” group include variables such as, difficulty finding NHS dentist etc.

In conclusion, the research student highlighted the components of an “ideal ECC conceptual framework” in the first column (“PhD student conceptual framework” column) of Table 8, which was informed by the findings of the systematic review undertaken in Chapter 3. The second column (“PhD student validated framework”) showed the results of the mapping of the retrievable SDH information from CDHS 2013. This column revealed that only 16 out of the 24 (i.e., 64%) possible SDH risk factors were able to map on to the ideal ECC framework (Column 1). Finally, all existing caries frameworks summarised in Table 2 were subsequently mapped against the ideal framework in column 1, in order to assess their gaps (see Table 8 below), and the SDH contents found in existing frameworks ranged between 0% and 48%. This clearly shows that the new, validated framework more effectively represents SDH than what currently exists.

Table 8: Mapping of CDHS variables to conceptual model

	PhD Student Conceptual Framework	[1]PhD Student Validated Framework	Keyes	Holst et al	Cynthia Pine et al	Patrick et al	Selewitz et al	Fisher-Owens et al	Watt and Sheiham	Seow	Chi	Duijster et al	Brunotto et al	Qiu et al	De Silva et al	Roncalli et al
Access to care	✓	✓			✓	✓		✓	✓						✓	✓
Age child start tooth brushing	✓	✓														
Area-Deprivation	✓	✓		✓												✓
Gender	✓	✓						✓	✓				✓			
Child living with family	✓	✓														
Parental Education	✓	✓					✓	✓		✓		✓	✓		✓	
Employment Status	✓	✓		✓			✓	✓	✓							
Employment type	✓	✓		✓			✓		✓							
Maternal age at child birth	✓	✓														
Parent's Oral Health Behaviour	✓	✓						✓	✓							
Race/Ethnicity	✓	✓			✓			✓	✓	✓		✓			✓	
Single Parenthood	✓	✓						✓								
Socioeconomic Status	✓	✓		✓	✓		✓	✓	✓	✓			✓	✓		✓
Support of routine oral hygiene	✓	✓						✓	✓							
Urban-Rural	✓	✓														
Water Fluoridation	✓				✓	✓		✓			✓				✓	
Child's birth order	✓															
Household family size	✓															
Family Caries History	✓							✓								
Family Smoking History	✓															
Access to dental insurance	✓							✓						✓		
Mode of delivery	✓															
Prematurity	✓															
Birth Weight	✓															
Data Validation		✓										✓	✓	✓		
Total	24	16	0	4	4	2	4	12	8	3	1	3	4	3	4	4
Percent	96%	64%	0%	16%	16%	8%	16%	48%	32%	12%	4%	12%	16%	12%	16%	16%

[1]PhD Student Validated Framework = Model was validated using data from CDHS 2013

5.3.2 Univariate Analysis

Following the mapping exercise, analyses (univariate, bivariate and multivariate analyses) of the CDHS were undertaken as stated in the objectives highlighted at the beginning of this chapter. The analyses were undertaken to validate the model and the results are presented in the following sections. The results of the univariate analysis for both complete and imputed CDHS dataset is summarised in Table 9 below. This table showed that the imputed dataset is very similar to the complete case dataset, where percentage difference seen between imputed and complete case, per variable, is less than or equal to 2% (Table 9). Univariate analysis showed that 32% of the five-year old in this study had caries. The results also showed that 60% of the participants were from England, whilst 21% and 19% were from Northern Ireland and Wales respectively. The analysis also showed that majority of the children (76%) lived in urban area, and the results of the participants by English region of residence ranged between 5% in East of England and 8% in both North West and South East. Similarly, the distribution of the examined children by area-deprivation was also examined, and the analysis showed that the range of distribution was between 14% and 23%, with majority of the participants residing in the most deprived region (23%), and the fewest in the least deprived region (14%) as shown in Table 9 below.

Table 9: Univariate Analysis

	Complete Case			Imputed		
SDH	**Freq.**	**%**		**Freq.**	**%**	**% difference between imputed and complete case**
Caries						
No	846	68		846	68	0
Yes	406	32		406	32	0
Gender						
Males	615	49		615	49	0
Females	637	51		637	51	0
Free School Meal						
No	1,009	83		1,040	83	0
Yes	207	17		212	17	0
Country						
England	689	55		689	55	0
Wales	242	19		242	19	0
Northern Ireland	321	26		321	26	0
Region						
1	69	6		69	6	0
2	71	6		71	6	0
3	79	6		79	6	0
4	61	5		61	5	0
5	62	5		62	5	0
6	63	5		63	5	0
7	80	6		80	6	0
8	115	9		115	9	0
9	89	7		89	7	0
Wales	242	19		242	19	0
Northern Ireland	321	26		321	26	0
Index of Multiple Deprivation						
Most deprived quintile	277	23		280	22	1
2nd	265	22		267	21	1
3rd	292	24		298	24	0
4th	223	18		231	19	1

	Complete Case			Imputed		
SDH	**Freq.**	**%**		**Freq.**	**%**	**% difference between imputed and complete case**
Least deprived quintile	172	14		176	14	0
Urban Rural						
Urban	367	30		374	30	0
Rural	861	70		878	70	0
Ethnicity						
Whites	1,052	87		1,067	85	2
Mixed	42	3		58	5	2
Asians	74	6		80	6	0
Blacks	32	3		34	3	0
Others	8	1		13	1	0
Qualification						
Degree level or above	543	44		550	44	0
Another type of qualification	582	47		590	47	0
No qualifications	109	9		112	9	0
Job Category						
Higher managerial, admin, professional occupations	526	42		526	42	0
Intermediate	288	23		288	23	0
Routine	326	26		326	26	0
Never worked	42	3		42	3	0
Not Classified	70	6		70	6	0

5.3.3 Bivariate, Multivariate and Multilevel Analyses

5.3.3.1 Summary of multilevel analysis (Model 0)

Results of the null model (model 0), i.e., null model without including independent variables, showed that the estimated intercept was -0.54. Variance of the distribution of the random effects is 0.11 (null model) and an intraclass correlation coefficient of 0.003, suggesting that 0.3% of the total of individual differences is attributable to neighbourhood effect, that is, the proportion of variance in ECC risk explained by area-deprivation is 0.3% (Table 10).

5.3.3.2 Place of Residence

Table 10 summarises the results of the bivariate, multivariate and multilevel analyses. It should be noted that bivariate analysis is the unadjusted odds ratio (OR), describing the relative odds of the SDH risk factors on the risk of developing caries. The results for place of residence analysis showed that the risk of developing caries is more than twice in children living in the most deprived quintile in the country compared with the least deprived quintile [Bivariate: 2.79 (2.06-3.82, p<0.001); Multivariate: 1.82 (1.23-2.71, p=0.003). Similarly, children living in the North West region [Bivariate: 2.71 (1.81-4.08, p<0.001); Multivariate: 2.38 (1.56-3.65, p<0.001); Multilevel: 2.34 (1.52-3.58, p<0.001)]; East Midlands [Bivariate: 1.93 (1.24-3.00, p=0.003); Multivariate: 1.81 (1.15-2.87, p=0.011); Multilevel: 1.79 (1.13-2.84, p=0.013)]; Wales [Bivariate: 1.82 (1.30-2.59, p=0.001); Multivariate: 1.68 (1.18-2.42, p=0.005); Multilevel: 1.67 (1.17-2.40, p=0.005)]; and Northern Ireland [Bivariate: 1.66 (1.18-2.35, p=0.004); Multivariate: 2.15 (1.49-3.14, p<0.001); Multilevel: 2.13 (1.47-3.11, p<0.001)] are significantly higher risk of developing caries compared with South East region (base with least prevalence).

Finally, contrary to what was found in the systematic review in chapter 3, children living in urban areas are more at risk of developing caries in the England, Wales and Northern Ireland [Bivariate: 1.30 (1.08-1.58, p=0.006)]. This urban-rural risk was further explored using the 8-class Office for National Statistics Output Area Classification (2011) described above, where it showed that children living in areas regarded as Multicultural Metropolitans [Bivariate: 1.69 (1.27-2.25, p<0.001); Multivariate: 1.97 (1.31-2.98, p=0.001); Multilevel: 1.83 (1.19-2.82, p=0.006)], Constrained City Dwellers [Bivariate: 2.17 (1.54-3.05, p<0.001); Multivariate: 2.29 (1.49-3.51, p<0.001), Multilevel: 2.05 (1.29-3.25, p=0.002)] and Hard-Pressed Living [Bivariate: 2.00 (1.57-2.55, p<0.001); Multivariate: 2.15 (1.56-2.98, p<0.001); Multilevel: 1.98 (1.39-2.82, p<0.001)] are at much higher risks of developing caries. Children living in Cosmopolitan [2.28 (1.01-5.14, p=0.046)] and Ethnicity Central areas [1.82 (1.00-3.31, p=0.050)] also had higher risk of developing caries relative to those in rural areas, which is significant at multivariate level only.

5.3.3.3 Race/Ethnicity

Analysis showed that children of Asian origin had significantly higher risk of developing caries when compared with children of White origin (baseline) [Bivariate: 1.61 (1.19-2.19, p=0.002); Multivariate: 1.65 (1.17-2.33, p=0.004); Multilevel: 1.63 (1.16-2.30, p=0.005)]. Children categorised under “Other undefined ethnicities” also had significant risk relative to children of White origin Bivariate: 3.16 (1.45-7.40, p=0.005); Multivariate: 3.37 (1.49-8.14, p=0.005); Multilevel: 3.34 (1.44-7.73, p=0.005).

5.3.3.4 Occupation

This study compared children of parents that have higher managerial, administrative, professional occupations with other children, and results showed that children living with parents with lower occupation level are at higher risk of developing caries compared with their counterparts living with parents on the highest occupation level. Intermediate occupations (clerical, sales, service) had an odds ratio of [Bivariate: 1.92 (1.40-2.62, p<0.001); Multivariate: 1.68 (1.20-2.36, p=0.003); Multilevel: 1.65 (1.17-2.32, p=0.004)] compared to children whose parents are managers; Routine and manual occupations [Bivariate: 2.12 (1.57-2.87, p<0.001); Multivariate: 1.62 (1.14-2.30, p=0.007); Multilevel: 1.62 (1.14-2.30, p=0.008)]; Never worked and long-term unemployed [Bivariate: 3.92 (2.07-7.52, p<0.001); Multivariate: 2.21 (1.06-4.60, p=0.033); Multilevel: 2.19 (1.05-4.55, p=0.036)].

5.3.3.5 Gender

The odds of a five-year old girl developing ECC, relative to a boy of the same age is lower, but this is not significant [Bivariate: 0.92 (0.78-1.08, p=0.286); Multivariate: 0.92 (0.78-1.09, p=0.333); Multilevel: 0.93 (0.78-1.09, p=0.358)].

5.3.3.6 Religion

Information on Religion was not available in the CDHS 2013 and therefore its influence on caries inequalities at child-level is unknown.

5.3.3.7 Education

Caries risk was assessed across three levels of educational attainment which are: “Degree level and above”, “Another qualification” and “No qualification”, and this study showed that risk was inversely proportional to educational attainment, i.e., the lower the qualification attainment, the higher the risk of developing caries. Children whose parents had another type of qualification had higher risk of developing caries, compared to children of parents with at least a degree [Bivariate: 1.41 (1.09-1.81, p=0.009); Multivariate: 1.07 (0.80-1.43, p=0.660); Multilevel: 1.09 (0.81-1.45, p=0.581)]. This risk was even higher in children whose parents had no qualification [Bivariate: 3.05 (2.01-4.64, p<0.001); Multivariate: 1.68 (1.04-2.72, p=0.035); Multilevel: 1.70 (1.05-2.76, p=0.031)].

5.3.3.8 Socioeconomic Status

Evaluating the risk posed by family socioeconomic (using eligibility to free school meal) showed that children assessed to be eligible for free school meal had significantly higher risk of developing caries [Bivariate: 1.61 (1.34-1.94, p<0.001); Multivariate: 1.39 (1.14-1.69, p=0.001); Multilevel: 1.35 (1.10-1.66, p=0.004)], than children who are not eligible for free school meal.

5.3.3.9 Social Capital

The two social capital risk variables identified in the CDHS 2013 were “single parenthood” and “problems finding NHS dentist”. Single Parenthood was significant only at bivariate level of analyses: Bivariate: 1.39 (1.03-1.86, p=0.029), and children who reported they had challenges with accessing an NHS dentist had slightly higher risk of developing caries, but

this was not significant [Bivariate: 1.18 (0.85-1.62, p=0.311); Multivariate: 1.08 (0.76-1.51, p=0.670); Multilevel: 1.07 (0.76-1.51, p=0.694)].

5.3.3.10 Other Risk Factors (PLUS)

Two other relevant wider determinants risk factors (identified in the systematic review) explored were whether child gets support with oral hygiene, as well as, the dental attendance pattern of the main carer. Children whose parents never visited the dentist (or they only visited when in pain) had much higher risk of ECC than children whose parent attended for regular check-up [Bivariate: 1.73 (1.32-2.26, p<0.001); Multivariate: 1.42 (1.06-1.89, p=0.019); Multilevel: 1.43 (1.07-1.92, p=0.017)]. Finally, when children whose main carer is solely responsible for oral hygiene are compared with other children, this study showed that the risk of developing caries is not different in children who had help with oral hygiene and those who did not [Bivariate: 1.18 (0.93-1.50, p=0.171); Multivariate: 1.04 (0.81-1.34, p=0.738); Multilevel: 1.04 (0.81-1.34, p=0.758)].

Table 10: Bivariate, Multivariate and Multilevel Analyses

SDH	Sound Teeth (n=1,537)	Caries (n=1,012)	Null Model Model 0	OR (Bivariate) Model 1	OR (Multivariable) Model 2	OR (Multilevel) Model 3
Variance			0.11			
Standard error			0.16			
Intraclass Correlation Coefficient			0.003			
Fit Statistics – AIC			3382.8			
Fit Statistics – BIC			3394.5			
Intercept			-0.54			
Odds Ratio			0.58			
p-value			0.0006			
Least deprived quintile	204 (74.7)	69 (25.3)	-	-	-	Reference
Most deprived quintile	401 (51.4)	379 (48.6)	-	**2.79 (2.06-3.82, p<0.001)**	**1.82 (1.23-2.71, p=0.003)**	Reference
2nd	342 (57.5)	253 (42.5)	-	**2.19 (1.60-3.02, p<0.001)**	**1.63 (1.14-2.35, p=0.008)**	Reference
3rd	332 (64.0)	187 (36.0)	-	**1.67 (1.21-2.32, p=0.002)**	**1.45 (1.03-2.05, p=0.036)**	Reference
4th	258 (67.5)	124 (32.5)	-	**1.42 (1.01-2.02, p=0.047)**	1.33 (0.94-1.90, p=0.114)	Reference
South East	145 (69.7)	63 (30.3)	-	-	-	-
North East	115 (71.0)	47 (29.0)	-	0.94 (0.60-1.47, p=0.790)	0.75 (0.47-1.19, p=0.226)	0.72 (0.45-1.16, p=0.179)
North West	91 (46.0)	107 (54.0)	-	**2.71 (1.81-4.08, p<0.001)**	**2.38 (1.56-3.65, p<0.001)**	**2.34 (1.52-3.58, p<0.001)**
Yorkshire and the Humber	107 (63.3)	62 (36.7)	-	1.33 (0.87-2.05, p=0.190)	1.27 (0.81-1.98, p=0.304)	1.28 (0.82-2.01, p=0.282)
East Midlands	80 (54.4)	67 (45.6)	-	**1.93 (1.24-3.00, p=0.003)**	**1.81 (1.15-2.87, p=0.011)**	**1.79 (1.13-2.84, p=0.013)**
West Midlands	106 (62.0)	65 (38.0)	-	1.41 (0.92-2.17, p=0.114)	1.11 (0.71-1.76, p=0.645)	1.10 (0.70-1.74, p=0.677)
East of England	90 (65.7)	47 (34.3)	-	1.20 (0.76-1.90, p=0.434)	1.25 (0.77-2.00, p=0.362)	1.23 (0.76-1.98, p=0.393)
London	119 (67.6)	57 (32.4)	-	1.10 (0.71-1.70, p=0.659)	0.85 (0.48-1.48, p=0.561)	0.83 (0.47-1.46, p=0.513)
South West	101 (63.9)	57 (36.1)	-	1.30 (0.84-2.02, p=0.243)	1.25 (0.79-1.99, p=0.338)	1.27 (0.80-2.02, p=0.303)
Wales	275 (55.8)	218 (44.2)	-	**1.82 (1.30-2.59, p=0.001)**	**1.68 (1.18-2.42, p=0.005)**	**1.67 (1.17-2.40, p=0.005)**
Northern Ireland	308 (58.1)	222 (41.9)	-	**1.66 (1.18-2.35, p=0.004)**	**2.15 (1.49-3.14, p<0.001)**	**2.13 (1.47-3.11, p<0.001)**
Rural	393 (65.1)	211 (34.9)	-	-	-	-
Urban	1144 (58.8)	801 (41.2)	-	**1.30 (1.08-1.58, p=0.006)**	1.07 (0.81-1.41, p=0.654)	1.03 (0.77-1.37, p=0.855)

SDH	Sound Teeth (n=1,537)	Caries (n=1,012)	Null Model Model 0	OR (Bivariate) Model 1	OR (Multivariable) Model 2	OR (Multilevel) Model 3
Rural Residents	329 (68.0)	155 (32.0)	-	-	-	-
Cosmopolitans	16 (51.6)	15 (48.4)	-	**1.99 (0.95-4.15, p=0.065)**	**2.28 (1.01-5.14, p=0.046)**	2.19 (0.97-4.96, p=0.060)
Ethnicity Central	89 (64.5)	49 (35.5)	-	1.17 (0.78-1.73, p=0.442)	**1.82 (1.00-3.31, p=0.050)**	1.65 (0.89-3.07, p=0.111)
Multicultural Metropolitans	193 (55.6)	154 (44.4)	-	**1.69 (1.27-2.25, p<0.001)**	**1.97 (1.31-2.98, p=0.001)**	**1.83 (1.19-2.82, p=0.006)**
Urbanities	216 (70.8)	89 (29.2)	-	0.87 (0.64-1.19, p=0.400)	1.01 (0.68-1.48, p=0.979)	1.02 (0.69-1.51, p=0.917)
Suburbanites	247 (66.9)	122 (33.1)	-	1.05 (0.78-1.40, p=0.749)	1.14 (0.79-1.63, p=0.482)	1.19 (0.82-1.73, p=0.355)
Constrained City Dwellers	95 (49.5)	97 (50.5)	-	**2.17 (1.54-3.05, p<0.001)**	**2.29 (1.49-3.51, p<0.001)**	**2.05 (1.29-3.25, p=0.002)**
Hard-Pressed Living	352 (51.5)	331 (48.5)	-	**2.00 (1.57-2.55, p<0.001)**	**2.15 (1.56-2.98, p<0.001)**	**1.98 (1.39-2.82, p<0.001)**
Whites	1303 (61.2)	825 (38.8)	-	-	-	-
Mixed	71 (59.2)	49 (40.8)	-	1.09 (0.75-1.58, p=0.652)	1.11 (0.73-1.66, p=0.625)	1.10 (0.73-1.65, p=0.640)
Asians	90 (49.5)	92 (50.5)	-	**1.61 (1.19-2.19, p=0.002)**	**1.65 (1.17-2.33, p=0.004)**	**1.63 (1.16-2.30, p=0.005)**
Blacks	64 (69.6)	28 (30.4)	-	0.69 (0.43-1.08, p=0.109)	0.69 (0.41-1.14, p=0.154)	0.67 (0.40-1.11, p=0.117)
Other Ethnicities	9 (33.3)	18 (66.7)	-	**3.16 (1.45-7.40, p=0.005)**	**3.37 (1.49-8.14, p=0.005)**	**3.34 (1.44-7.73, p=0.005)**
Higher managerial, admin, professional occupations	402 (76.4)	124 (23.6)	-	-	-	-
Intermediate	181 (62.8)	107 (37.2)	-	**1.92 (1.40-2.62, p<0.001)**	**1.68 (1.20-2.36, p=0.003)**	**1.65 (1.17-2.32, p=0.004)**
Routine	197 (60.4)	129 (39.6)	-	**2.12 (1.57-2.87, p<0.001)**	**1.62 (1.14-2.30, p=0.007)**	**1.62 (1.14-2.30, p=0.008)**
Never worked	19 (45.2)	23 (54.8)	-	**3.92 (2.07-7.52, p<0.001)**	**2.21 (1.06-4.60, p=0.033)**	**2.19 (1.05-4.55, p=0.036)**
Not Classified	47 (67.1)	23 (32.9)	-	1.59 (0.91-2.69, p=0.093)	1.32 (0.73-2.34, p=0.350)	1.34 (0.75-2.40, p=0.329)
Male	749 (59.3)	515 (40.7)	-	-	-	-
Female	788 (61.3)	497 (38.7)	-	0.92 (0.78-1.08, p=0.286)	0.92 (0.78-1.09, p=0.333)	0.93 (0.78-1.09, p=0.358)
Degree level or above	403 (73.3)	147 (26.7)	-	-	-	-
Another type of qualification	390 (66.1)	200 (33.9)	-	**1.41 (1.09-1.81, p=0.009)**	1.07 (0.80-1.43, p=0.660)	1.09 (0.81-1.45, p=0.581)
No qualifications	53 (47.3)	59 (52.7)	-	**3.05 (2.01-4.64, p<0.001)**	**1.68 (1.04-2.72, p=0.035)**	**1.70 (1.05-2.76, p=0.031)**
Ineligible for Free school meal	1232 (63.0)	723 (37.0)	-	-	-	-

SDH	Sound Teeth (n=1,537)	Caries (n=1,012)	Null Model Model 0	OR (Bivariate) Model 1	OR (Multivariable) Model 2	OR (Multilevel) Model 3
Eligible for Free School Meal	305 (51.3)	289 (48.7)	-	**1.61 (1.34-1.94, p<0.001)**	**1.39 (1.14-1.69, p=0.001)**	**1.35 (1.10-1.66, p=0.004)**
Independent school	37 (69.8)	16 (30.2)	-	-	-	-
Primary school	1463 (60.3)	962 (39.7)	-	1.52 (0.86-2.82, p=0.165)	1.09 (0.59-2.07, p=0.794)	1.05 (0.56-1.96, p=0.886)
Academy or free school (England)	37 (52.1)	34 (47.9)	-	2.12 (1.02-4.57, p=0.049)	1.68 (0.77-3.75, p=0.201)	1.62 (0.73-3.58, p=0.234)
Two parents	702 (69.0)	316 (31.0)	-	-	-	-
Single parent	144 (61.5)	90 (38.5)	-	**1.39 (1.03-1.86, p=0.029)**	1.07 (0.75-1.52, p=0.715)	1.10 (0.77-1.57, p=0.611)
Adult brushes teeth	491 (69.2)	219 (30.8)	-	-	-	-
Child brushes teeth	355 (65.5)	187 (34.5)	-	1.18 (0.93-1.50, p=0.171)	1.04 (0.81-1.34, p=0.738)	1.04 (0.81-1.34, p=0.758)
Problem finding NHS Dentist – No	719 (68.2)	336 (31.8)	-	-	-	-
Problem finding NHS Dentist – Yes	127 (64.5)	70 (35.5)	-	1.18 (0.85-1.62, p=0.311)	1.08 (0.76-1.51, p=0.670)	1.07 (0.76-1.51, p=0.694)
Dental attendance of responding adult: Regular check up	668 (70.6)	278 (29.4)	-	-	-	-
Dental attendance of responding adult: When trouble/never	178 (58.2)	128 (41.8)	-	**1.73 (1.32-2.26, p<0.001)**	**1.42 (1.06-1.89, p=0.019)**	**1.43 (1.07-1.92, p=0.017)**

5.3.4 Predictability of the caries model

After the application of eight commonly cited and used classification models in the medical field, Support Vector Machine (SVM) model had the best ROC value. As stated in section 5.2.4.4, the rule of thumb is to choose the predictive model with the highest ROC score (Carter et al., 2016), and the SVM model had an ROC Curve (AUC) of 0.76 (CI = 0.61 – 0.86), i.e., the model predicts 76 out of 100 cases correctly, after the 10-fold cross-validation (Figure 23 and Table 11). The SVM model has a specificity value of 0.91 (CI = 0.79 -1.00) i.e., 91 out of 100 actual negative cases (no caries) that were correctly predicted and sensitivity value of 0.34 (CI = 0.18 – 0.54) i.e., 34 out of 100 actual positive cases (caries) were correctly predicted.

Figure 23: Results of predictability of models after 10-fold cross validation

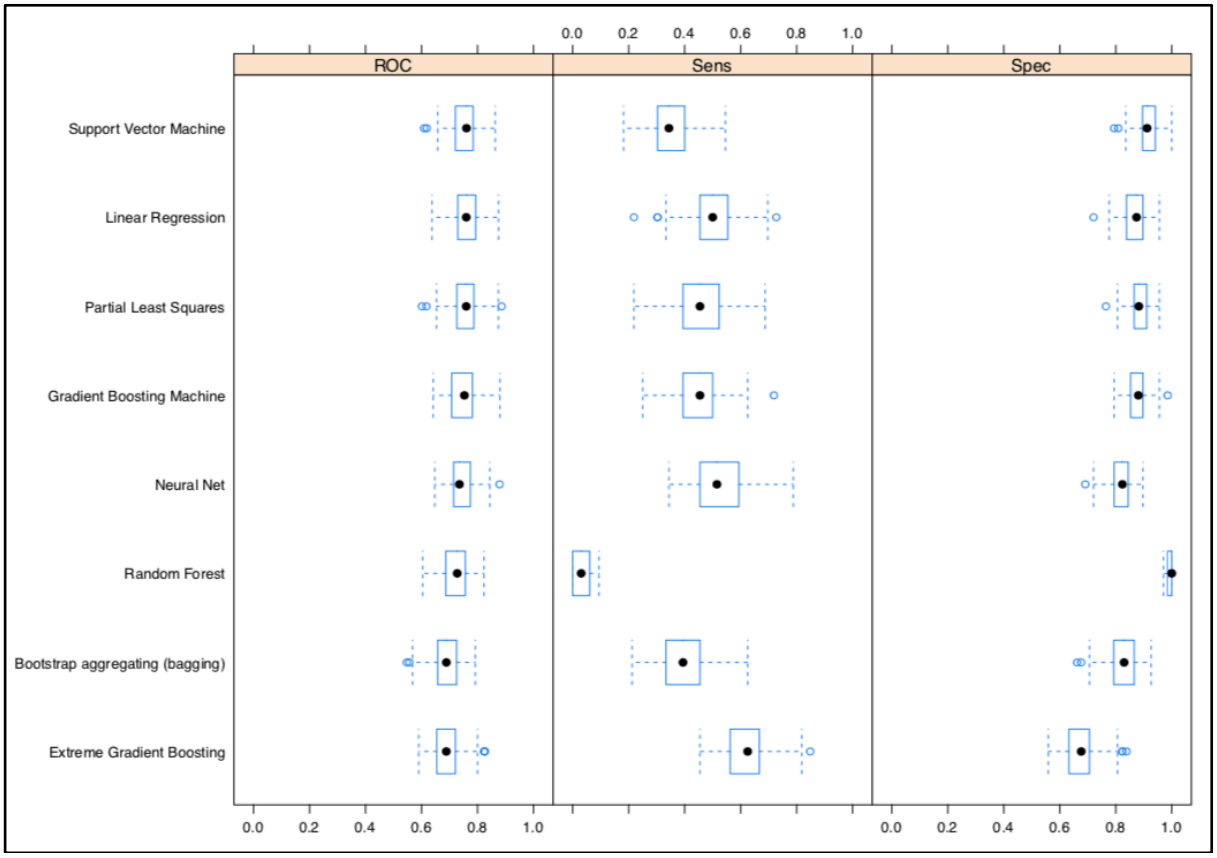


Table 11: A table comparing seven predictive risk models

ROC Curve	Minimum	1st Quartile	Median	Mean	3rd Quartile	Maximum
Partial Least Squares	0.6015	0.7268	0.7596	0.7575	0.7864	0.8860
Gradient Boosting Machine	0.6413	0.7080	0.7531	0.7466	0.7818	0.8801
Logistic Regression	0.6373	0.7298	0.7597	0.7580	0.7931	0.8750
Neural Net	0.6474	0.7145	0.7353	0.7437	0.7734	0.8787
Bootstrap aggregating (bagging)	0.5470	0.6589	0.6886	0.6859	0.7244	0.7920
Support Vector Machine	**0.6094**	**0.7215**	**0.7604**	**0.7524**	**0.7832**	**0.8631**
Extreme Gradient Boosting	0.5900	0.6551	0.6885	0.6913	0.7207	0.8261
Random Forest	0.6041	0.6879	0.7274	0.7229	0.7557	0.8232

Sensitivity	Minimum	1st Quartile	Median	Mean	3rd Quartile	Maximum
Partial Least Squares	0.2188	0.3939	0.4545	0.4559	0.5192	0.6875
Gradient Boosting Machine	0.2500	0.3939	0.4545	0.4474	0.5000	0.7188
Linear Regression	0.2188	0.4545	0.5000	0.4984	0.5497	0.7273
Neural Net	0.3438	0.4545	0.5152	0.5253	0.5938	0.7879
Bootstrap aggregating (bagging)	0.2121	0.3333	0.3939	0.3893	0.4545	0.6250
Support Vector Machine	**0.1818**	**0.3030**	**0.3438**	**0.3544**	**0.3970**	**0.5455**
Extreme Gradient Boosting	0.4545	0.5625	0.6250	0.6260	0.6667	0.8485
Random Forest	0.0000	0.0000	0.0303	0.0324	0.0606	0.0938

Specificity	Minimum	1st Quartile	Median	Mean	3rd Quartile	Maximum
Partial Least Squares	0.7647	0.8657	0.8824	0.8839	0.9104	0.9559
Gradient Boosting Machine	0.7941	0.8529	0.8806	0.8758	0.8971	0.9853
Linear Regression	0.7206	0.8382	0.8741	0.8709	0.8971	0.9559
Neural Net	0.6912	0.7941	0.8235	0.8168	0.8414	0.8971
Bootstrap aggregating (bagging)	0.6618	0.7933	0.8297	0.8249	0.8657	0.9265
Support Vector Machine	**0.7941**	**0.8955**	**0.9118**	**0.9135**	**0.9412**	**1.0000**
Extreme Gradient Boosting	0.5588	0.6324	0.6765	0.6781	0.7059	0.8382
Random Forest	0.9701	0.9853	1.0000	0.9950	1.0000	1.0000

*__Number of resamples: 100__

Comparison of SDH-based model result with other caries risk assessment tools

Finally, the accuracy of the risk model developed is ranked among the four main types of caries risk assessment tools (Fontana, 2015, Gao et al., 2013). The table in Appendix 40 below summarises the components (risk factors) of each risk assessment tool and the result of the ranking is presented in Table 12. It should be emphasised that the model developed in this chapter is the only model without one or more of clinical, microbiological or salivary test component in it (see Appendix 40).

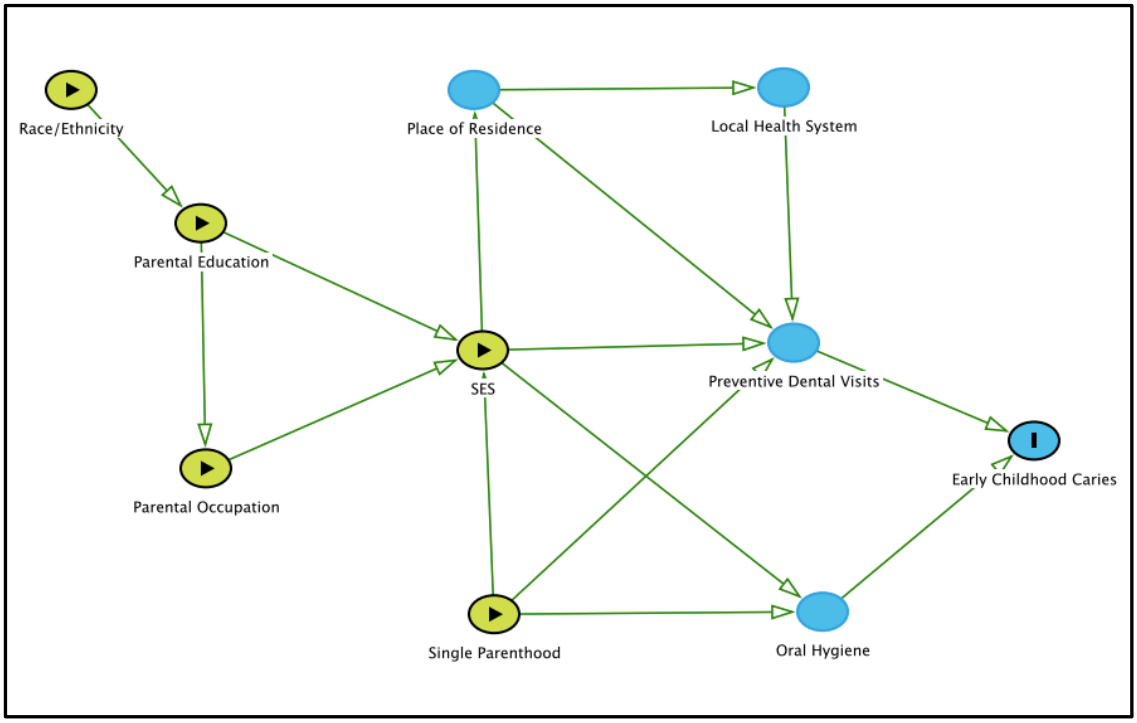
Table 12: Ranking of Risk prediction tool (Fontana, 2015, Gao et al., 2013).

Risk Prediction Model	**Accuracy**	**Specificity**	**Sensitivity**
NUS-CRA with microbiological test	83	85	78
SDH Based Model developed by PhD Student	**76**	**91**	**34**
Cariogram with microbiological test	74	79	65
CAMBRA with salivary/microbiological test	71	63	84
CAT screening with salivary/microbiological test	39	4	100

5.3.5 Path Analysis

The proposed path model was modified, based only on the available variables from the CDHS 2013 data that maps on to the variables found in the systematic review (Figure 24). In this figure, one-way causal arrows were used to demonstrate all hypotheses related to developing caries and it showed the factors that contributed to caries inequalities seen in 5-year old children in the United Kingdom.

Figure 24: Proposed model diagram based on available datasets from CDHS 2013



Testing proposed model diagram using Structural Equation Model (SEM)

The theory being advanced in this thesis is that SDH predisposes or determines oral behavioural patterns, therefore, the modified path model consists of nine major items: race, parental education, parental occupation, socio-economic status, place of residence, single parenthood, local health system, preventive dental visits and oral hygiene. The recommended sample size to fit a SEM (path model) is 200 (Wolf et al., 2013, Kline, 2015), and therefore this model was tested using non-imputed variables, as the sample size of 1,252 was deemed adequate to fit a path model. The values of the fit indices for this model are shown in the Table 13 below, which fall within acceptable limits of Comparative Fit Index (CFI) > 0.9, and Root Mean Square Error of Approximation <0.10.

Table 13: Results of Fit Index for Preschool Caries Path Analysis

Degree of Freedom	P-value (Chi-square)	Comparative Fit Index (CFI)	Tucker-Lewis Index	Root Mean Square Error
35	<0.001	0.95	0.92	0.04

The final (tested) caries model in children is now shown below in Figure 25. Further analysis from the path model showed that the two major factors contributing directly to the caries status among children were significant (Figure 25 and Table 14). The first factor was preventive dental visits showed that children who do not have preventive dental visits were 1.18 times more likely to develop caries (OR: $e^{0.163}$ = 1.18, 95%CI = 1.05 – 1.31; p=<0.001) compared with those who did; and the second factor was oral hygiene that showed children with poor oral hygiene being 1.32 times more likely to develop caries than (OR: $e^{0.279}$ = 1.32; CI= 1.20 - 1.45; p=<0.001). Similarly, there were 12 indirect pathways in the final path model, linking all the SDH variables, and all the indirect paths were significant, except in three pathways: Place of residence determining Local Health System (OR: $e^{-0.08}$ = 0.92; CI = 0.84 - 1.03; p= 0.15); Single Parenthood determining Child's Preventive Visits (OR: $e^{-0.19}$ = 0.83; 0.63 - 1.09; p=0.18); and Single Parenthood determining Child's Oral Hygiene (OR: $e^{0.02}$ = 1.02; 0.78 - 1.32; p=0.91).

In this path model, highest level of Parental Education was a significant predictor for both Parental Occupation (OR: $e^{0.66}$ = 1.02; 0.78-1.3; p <0.001), and Family Socioeconomic Status (SES), which was assessed using eligibility for free school meal (OR: $e^{0.52}$ = 1.68 (1.45 - 1.95); p <0.001). The path showed that the lower parental education, then the lower the SES; and the lower the parental occupation level. SES is a direct predictor of SDH factors: Place of Residence, which is measured by the post-code based Index of Multiple Deprivation (IMD) (OR: $e^{-0.39}$ = 0.68 (0.63 - 0.72); p <0.001); Preventive Dental Visits (OR: $e^{0.19}$ = 1.21 (1.06 -

1.39); p<0.001); and Poor Oral Hygiene (OR: $e^{0.10}$ = 1.11 (1.00 - 1.22); p = 0.05). The lower the SES, the lower the preventive visits, the poorer the oral hygiene and the more deprived their place of residence is. Single parenthood on its own is a direct predictor of oral hygiene (OR: $e^{0.02}$ = 1.02 (0.78 - 1.32); p = 0.91, preventive visits (OR: $e^{-0.19}$= 0.83 (0.63 - 1.09); p = 0.18) and SES (OR: $e^{1.31}$ = 3.71 (3.03 - 4.48); p <0.001), however, the only significant path in single parenthood is SES.

Figure 25: Path Analysis demonstrating early childhood caries pathway (odds ratio in parenthesis)

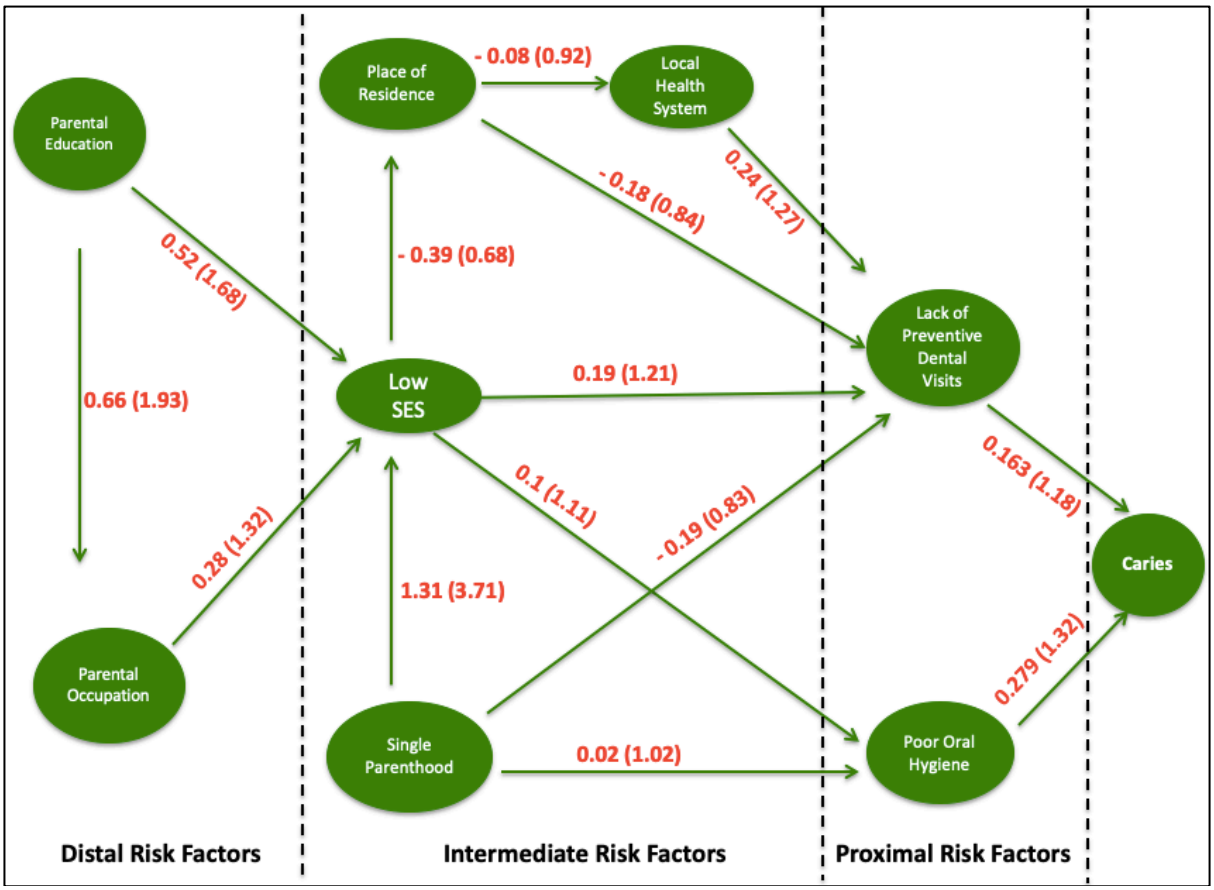


Table 14: Path coefficient table

	Coefficient						Odds Ratio					
Path	**Estimate**	**P(>|z|)**	**Lower CI**	**Upper CI**	**Std.lv**	**Std.all**	**Estimate**	**P(>|z|)**	**Lower CI**	**Upper CI**	**Std.lv**	**Std.all**
SES ~Education	**0.52**	**0.00**	**0.37**	**0.67**	**0.52**	**0.27**	**1.68**	**0.00**	**1.45**	**1.96**	**1.68**	**1.31**
Occupation ~Education	**0.66**	**0.00**	**0.55**	**0.77**	**0.66**	**0.39**	**1.93**	**0.00**	**1.74**	**2.15**	**1.93**	**1.47**
SES ~Occupation	**0.28**	**0.00**	**0.18**	**0.37**	**0.28**	**0.24**	**1.32**	**0.00**	**1.20**	**1.44**	**1.32**	**1.27**
Place ~SES	**-0.39**	**0.00**	**-0.46**	**-0.33**	**-0.39**	**-0.47**	**0.68**	**0.00**	**0.63**	**0.72**	**0.68**	**0.63**
Preventive Visits ~Place	**-0.18**	**0.00**	**-0.29**	**-0.06**	**-0.18**	**-0.18**	**0.84**	**0.00**	**0.75**	**0.94**	**0.84**	**0.83**
Local Health System ~Place	**-0.08**	**0.15**	**-0.18**	**0.03**	**-0.08**	**-0.08**	**0.93**	**0.15**	**0.83**	**1.03**	**0.93**	**0.92**
Preventive Visits ~Local Health	**0.24**	**0.00**	**0.11**	**0.38**	**0.24**	**0.24**	**1.27**	**0.00**	**1.11**	**1.45**	**1.27**	**1.27**
Preventive Visits ~SES	**0.19**	**0.00**	**0.06**	**0.33**	**0.19**	**0.24**	**1.21**	**0.00**	**1.06**	**1.38**	**1.21**	**1.27**
SES ~Single Parenthood	**1.31**	**0.00**	**1.11**	**1.50**	**1.31**	**0.40**	**3.69**	**0.00**	**3.03**	**4.50**	**3.69**	**1.50**
Preventive Visits~Single Parenthood	-0.19	0.18	-0.46	0.09	-0.19	-0.07	0.83	0.18	0.63	1.09	0.83	0.93
Oral Hygiene ~Single Parenthood	0.02	0.91	-0.25	0.28	0.02	0.01	1.02	0.91	0.78	1.32	1.02	1.01
Caries ~Oral Hygiene	**0.28**	**0.00**	**0.37**	**0.18**	**0.28**	**0.28**	**1.32**	**0.00**	**1.45**	**1.20**	**1.32**	**1.32**
Oral Hygiene ~SES	**0.10**	**0.05**	**0.00**	**0.20**	**0.10**	**0.13**	**1.11**	**0.05**	**1.00**	**1.22**	**1.11**	**1.13**
Caries~ Preventive Visits	**0.16**	**0.00**	**0.27**	**0.05**	**0.16**	**0.16**	**1.18**	**0.00**	**1.31**	**1.05**	**1.18**	**1.18**

***Paths with bold fonts are significant**

5.4 Discussion

5.4.1 Main findings

In this study, caries was not only prevalent in the sample used in the CDHS 2013 (32% of respondents), but further analyses confirmed that it was also found to be associated with SDH-based deprivation. It showed that the identified social determinants risk factors from systematic review influence the risk of developing caries in five-year old children. The key inference from this observation is that the predisposition of 5-year-old children to caries is shown to be through a very complex SDH mechanism that goes beyond just diet, oral health behaviour or oral bacteria as proposed by Keyes (Keyes, 1962).

Although the overall impact of neighbourhood area on developing ECC is negligible (0.3% from multilevel analysis conducted), however, a series of separate area-based analyses conducted showed that the "place of residence" is one of the most crucial SDH factor that determines caries risks in children. It was the only risk factor that had four possible indices of assessment: area-deprivation (IMD), region, urban-rural and area-type. Children living in the most deprived quintiles across England and Wales were almost twice at risk of developing caries when compared with those in least deprived quintiles [1.82 (1.23-2.71, p=0.003)]. The association between area deprivation and morbidity is well recognised and the difference in risk observed between the most and least deprived areas has been linked to the socioeconomic composition differences seen within the population (Reijneveld et al., 2000, Charlton et al., 2013). Living in one of the North West, East Midlands, Wales or Northern Ireland regions also poses a

significant caries risk in children and these three regions are listed among the poorest regions in the United Kingdom (Office for National Statistics, 2018). Bivariate analysis also showed that children from urban areas also had significantly higher risk than their rural counterparts, which is contrary to the findings of the systematic review reported in chapter 3. However, this significance is lost in multivariate analysis and the possible explanation for this is that the differences between rural and urban areas have diminished in the UK, and rural area are more connected and closer to urban areas in the UK than other countries like the United States (Midouhas and Flouri, 2015). In terms of deprivation, it is also documented that only 4% of those in the most deprived population live in rural area in England, whilst 26% of the most deprived live in urban area (Milojevic et al., 2017). Children living in urban areas have more caries experience because they consume more sugars than their rural counterparts (Amalia et al., 2012).

Another finding that is consistent with other studies is that children of Asian origin showed statistically higher risk of developing caries, even after adjusting for deprivation (Bedi and Elton, 1991, Conway, 2007, Dugmore and Rock, 2005). Asian children had almost twice the risk of caries compared to White children, and reviewing earlier studies showed that this inequality by ethnic origin has persisted in the UK for more than 30 years (Prendergast et al., 1997, Bedi, 1989). A few explanations have been put forward for this caries risk by ethnic origin, one of which is the language barriers that some immigrants face, and therefore those with poor English may be unable to access preventive oral health information in time (Conway, 2007). Rouxel et al., study also concluded that of all mothers, mothers of Asian origin are less likely to

consider the sugar content of food and less likely to avoid teeth damaging foods (Rouxel et al., 2018). Their study specifically identified Pakistani mothers as more likely to introduce sweetened drinks and foods at an early age compared with mothers from other ethnic background (Rouxel et al., 2018).

Another important finding is regarding the risk prediction model developed, which showed promising characteristics, when ranked among the four highly regarded caries risk assessment tools (Fontana, 2015, Gao et al., 2013). The uniqueness of this risk model is that it is the only one without clinical, microbiological or salivary test, and its overall accuracy was ranked second in the list (Table 12 and Appendix 40). The SDH-based risk model has a high specificity (91%) and low sensitivity (34%) score, and the implication of this is that it has the ability to produce only a few false positive results. That is, 91% specificity correctly identifies 91% of the children without ECC as true negatives, but the remainder (9%) without the disease (ECC) are incorrectly classed as having ECC (false negatives). Although the ideal but impracticable scenario is to have a risk assessment tool that is 100% accurate (100% specificity and 100% sensitivity), trade-offs between specificity and sensitivity are acceptable (Hennekens et al., 1987). However, where a choice need to be made, it is generally not advisable to use a test with low specificity as a screening test because majority of people without the disease will screen positive (false positive) (Hennekens et al., 1987). Similarly, a model with high sensitivity but low specificity falsely classify disease free patients as having disease, and therefore they will be subjected to unnecessary investigations or diagnostic procedures (Hennekens et al., 1987). Lalkhen and McCluskey and Kobilinsky et al., both recommended that a test with high sensitivity, low

specificity characteristics should be the first line of risk assessment, known as presumptive screening. All the positive patients from the presumptive screening must then be subjected to a confirmatory test that is high specificity/low sensitivity test, like the one developed in this thesis, therefore making the results more reliable (Kobilinsky et al., 2005, Lalkhen and McCluskey, 2008).

Finally, in the temporal-based path analysis model developed in section 4.6.3, the findings showed that SDH had no direct relationship with caries inequalities in children, but they mediate their effects via two behavioural factors, which are preventive visits and routine oral hygiene (Figure 25). This conceptual model showed that there are two important distal factors triggering caries inequalities in a five-year old and these are "Parental Education" and "Parental Occupation" in that order, which is followed by four intermediate determinants, which are socioeconomic status of the family, single parenthood status, place of residence and local health system. Finally, the proximal factors directly responsible for caries inequalities are lack of preventive visits and poor oral hygiene. The possible benefit of this evidence-based path modelling is the opportunity to provide guidance on the level at which caries inequalities can be modified or controlled, and the likely impact this control will provide.

This study demonstrates novelty in at least two ways. Firstly, it is the first known early childhood caries modelling study to develop conceptual modelling from systematic review in order to identify all possible caries risk factors relevant to preschool children only, then used directed acyclic graphs and path analysis to explain the relationship these risk factors have with

one another, which ultimately adds depth to previous caries modelling studies. The validity of the developed model was also tested, using real world epidemiologic data, which is recommended in conceptual risk modelling (Cox, 2002, Jabareen, 2009, Saroyan et al., 2012).

Secondly, this research was the first in the field, to the best of researcher's knowledge, to develop caries risk prediction models using only social determinants variables entirely, that is, excluding all behavioural variables like oral hygiene, sugar consumption and preventive measures in the model. Prior to now, the accuracy of existing caries models is highly dependent on the accuracy of the self-reported history obtained from parents, however, self-reported history are usually inaccurate because it is dependent on excellent recall abilities. Outcomes from models built on behavioural history can also be misleading because primary care-givers are prone to withhold important information for fear of being blamed (victim-blaming) or held responsible for their children's poor oral health outcomes (Heilmann et al., 2016). Therefore, the total exclusion of behavioural factors is a novel approach to disease risk modelling and its advantage could be that the model provides a much better risk attribution that is independent of past behavioural histories.

5.4.2 Strengths and Limitations

One of the strengths of this study include the relatively large study size and the methodology used to collect the CDHS 2013 dataset, where the overall sample size used for modelling was 1,252 five-year old children from different regions across three out of the four countries that make up the United Kingdom. The survey data was also carried out using a multi-stage stratified

random sampling technique across England, Wales and Northern Ireland, which therefore improves the generalisability of the study. The dataset itself contains adequate information on behavioural and wider determinants characteristics influencing five-year old children. Another strength is that the predictive risk model was developed using relatively modern statistical methods such as machine learning that are capable of achieving better outcomes than traditional logistic regression.

The weakness of the study is largely that it was developed from survey data (cross-sectional design), which is a one-time measurement of exposure and outcome, making it difficult to ascertain causality i.e., whether exposure precedes outcome. Surveys are also known to be susceptible to biases, such as recall bias, non-response bias and interviewer bias (Setia, 2016, Sedgwick, 2014). The other weakness in this study is in the large amount of missing data recorded in the CDHS 2013, where only 53% of those who participated in the clinical examination (phase 1) returned their completed questionnaire (phase 2). Data samples with large missing data are prone to be unrepresentative of the population and also lead to biased estimation of parameters (Sedgwick, 2014, Kang, 2013).

Another weakness in this study is with the use of secondary data analysis for explanation of a concept. This CDHS 2013 data was not collected primarily for SDH research study, therefore, it lacked some important information such as which of the children was exposed to water fluoridation and dietary intake that would have biased the conclusions from the risk model and conceptual model respectively. When CDHS 2013 was mapped to the important SDH variables

identified from systematic review, the result showed that there was about 64% retrievable SDH information from CDHS 2013, i.e., 15 out of the 24 possible SDH risk factors influencing ECC. This rises slightly to 64% (16 out of the 25) when data validation is assessed. The mapping showed that there were no data information on: mode of delivery, prematurity, birth weight, child's birth order, household family size, family caries history, parental smoking history, access to dental insurance and water fluoridation (Table 8).

There were also obligatory changes to the oral survey methodology when it comes to consent. Parental consent for the survey changed from being opt-out to opt-in, which have resulted in a decreased response rate for the examination phases consent (Spence et al., 2015). It has been argued that opt-in surveys are more likely to introduce response bias, because of their propensity to lower participation rate and a less representative sample (Spence et al., 2015). Caries epidemiologists also believe that parents of children with higher level of caries experience are less likely to consent, thus compromising the data quality (Spence et al., 2015, Vernazza et al., 2016a).

5.5 Implications of CDHS Study

5.5.1 Implication for policymakers

The prevalence of caries in this study is 32% which is higher than the published 27.9% reported by Public Health England in 2012, i.e., one in three preschool children had caries. Similarly, the number of tooth extraction in children aged four and under increased by 24% within a decade, between 2006-07 and 2015-16 (The Royal College of Surgeons of England, 2017). Caries also

impacts on the national health budget, where children who were admitted for caries-related diseases cost the NHS England £30 million in 2012 – 2013 (Faculty of Dental Surgery, 2015a). The findings of this study therefore have implications for both intervention strategies and resource allocation that will be useful to achieve significant reductions in both caries prevalence and inequalities, which are discussed further below.

Firstly, the conceptual framework developed highlighted the importance of wider determinants by demonstrating them either as an ancestor, intermediate or descendant variables in the caries causal pathway (Figure 25). Therefore, this framework will help health planners to identify possible areas of intervention that are likely to yield greatest outcomes.

Similarly, the SDH-based risk model developed has the ability to compute “expected risk” of five-year olds for developing ECC, therefore preventive programmes can be better targeted based on evidence. Prior predictive risk models to this all have one or more combination of sugar consumption, oral hygiene or preventive visits in their model, mainly because of the relative proximity of behavioural risk factors to the outcome (Figure 25).

Risk models based on behavioural factors may disproportionately place greater emphasis on behavioural risks as the most important factors in caries risk models, than they should on the wider determinants (“causes of the causes”). This observation is likely the major reason why majority of existing preventive interventions focus mainly on diet and encouraging oral hygiene and preventive dental visits, and do less in improving the "the conditions in which people are born, grow, live, work and age" (World Health Organisation, 2010). It has also been

documented that healthy oral behaviours are actually achieved when people are empowered to make healthy choices, by addressing the "causes of the causes" (Kallestal et al., 2000, World Health Organisation, 2003). The study by Khan found a correlation between wider determinants and the decisions, choices and actions people take regarding their oral health (Khan, 2017). His argument is that being empowered gives the ability to have control not only to avoid adverse exposures, but equally to be able to deal with them, even when exposed, without falling ill (or developing caries) (Khan, 2017). Therefore, policy makers need to think about the development of oral health policies and interventions that address both SDH and oral behavioural factors at the same time.

The third implication is that this model can influence the manner in which public health messages are being promoted, for example, the exclusion of behavioural factors from the pathway will potentially shift blame away from parents and primary carers. Examining the four most recent recommendations made by the Public Health England, in order to combat tooth decay showed that the entirety of the recommendations is to the individual (Public Health England, 2018a).

1. Swap sugary drinks for lower or no sugar alternatives
2. Limit fruit juice and smoothies to a total of 150ml per day
3. Ensure children brush twice a day with fluoride toothpaste (once before bedtime and once during the day).
4. Brushing should start as soon as the first tooth appears and children should be supervised up to the age of 7.

Recommendations like this tend to place the entire caries-prevention responsibilities on the parents and primary carers and that usually makes them feel absolutely responsible and guilty for their children's poor oral health status (Pahel et al., 2007, Naidu et al., 2016). It can also be argued that recommendations like these above equally absolves the government and Health Authority from commitments to improve on the wider causes of caries prevalence. However, this study clarified, using the principles of causality, that main reason why children or parents will indulge in any of the four risks highlighted by PHE above, is attributable to their unequal exposures to the "causes of the causes". For example, a five-year old living in the North West region, which is largely deprived, has more than twice the risk of developing caries, when compared to a five-year old living in the South East region, which is considered relatively affluent. That can also be said of ethnicity, e.g., being Asian, and those children with "no qualifications attained" parents, therefore, the overall implication is for PHE to invest more in targeted services based on wider determinants needs identified, i.e., major responsibilities of caries incidence transferred from primary carers to the government, or at least shared between both parties.

5.5.2 Implications for clinicians

The clinical significance of this study is that the model can support practitioners in determining children who are susceptible to developing caries, such that preventive measures can be targeted especially when resources are limited. Implementing it in the clinic will certainly aid clinicians in classifying children in high and low risk with an overall accuracy of 76%. Up until now, primary care dentists rely almost entirely on oral health behaviour history (especially diet

and oral hygiene), however, evidence suggests that this history is seldom accurate, due to the reasons earlier discussed above (section 5.4.1). However, requesting further tests like clinical and microbiological tests to supplement oral behaviour history will definitely have cost implications on the patient or health system (Fontana, 2015, Gao et al., 2013). Therefore, an SDH-based predictive model could be a much-preferred tool because of the convenience it offers.

5.5.3 Implications for patients

Implementing this risk model also has implications on patients and their carers. Children that are deemed to be at higher risk, due to their relative SDH caries-risk exposures, will be prescribed more frequent dental clinic attendance for preventive treatments. However, there are published studies that have linked regular dental attendance and the possibility of over-treating the patients (Sheiham et al., 1985, Beirne et al., 2007).

5.6 Conclusion

In conclusion, this chapter has shown that although behavioural factors are the direct cause (precursor) of caries, but largely, they (behavioural factors) only act as a mediator; and itself being determined by the child's differential exposures to SDH (i.e., "causes of the causes"). The conceptual model, evaluated against standardised fit measures, also provided the information on the possible pathways from SDH risks to caries, mediated by behavioural risks (oral hygiene and preventive visits). Finally, the risk model developed in this chapter showed very good predictive abilities and ranked well among commonly used caries risk assessment tools.

However, the use of SDH-based caries risk model is novel and promising, considering that it can further be improved on when additional wider determinants that were not collected routinely, such as the one summarised in Table 8, are included.

The next chapter (chapter 6) will further explore caries data collected at local authority (LA) level in order to identify and understand SDH risk factors that determine caries prevalence at LA level. In this chapter, “place of residence” was seen to play some role in developing ECC, where ECC risk varied by area-level deprivation, region of residence, urban-rural area and finally by postcode-based Output Area Classification. It is therefore important to investigate and understand the important ECC risk factors influencing caries prevalence at local authority level.

CHAPTER SIX: DEVELOPING CARIES PREVALENCE RISK PREDICTION MODEL USING ADMINISTRATIVE DATASETS

6.1 Introduction

The previous study in chapter 5 explored the relationship between Social Determinants of Health (SDH) and developing early childhood caries (ECC) at an individual child-level and the findings revealed that SDH risk factors predisposing five-year olds to developing caries exist more in deprived, urban areas, and in the North West, East Midlands, Northern Ireland or Wales. Therefore, the place where a child resides appears to be one of the most important caries inequalities risk factor. This study therefore proposes to further explore other area-level characteristics that goes beyond those explored in chapter 5. The aim of this chapter is to identify all the factors responsible for the caries risk differences observed between different geographic (Local Authority) areas in England, to understand how "place of residence" exerts its detrimental influence on a child's teeth, and to use that understanding to develop an 'area-level' caries prevalence risk prediction model. Therefore, this chapter will provide answer to the question: "What are the factors responsible for the differences in caries risks seen between two local authority areas in England?", and the objectives are as follows:

Objectives

1. To identify and describe the relevant datasets used and map variables in these datasets under PROGRESS PLUS
2. To develop a caries risk model that describes the relationship between SDH and caries prevalence at local authority level
3. To determine the predictability of the risk model for caries outcome, using appropriate statistical methods.

6.2 Materials and Method

6.2.1 The data source

This study is a secondary analysis of three main administrative datasets that provide Local Authority (LA) level information on both the prevalence of ECC and on the prevalence of SDH (PROGRESS-PLUS). These datasets include Public Health England datasets, UK census 2011, and Office for National Statistics datasets. The Public Health England (PHE) oversees the National Dental Epidemiology Programme for England and they conduct oral health surveys in five-year olds every two years. The latest survey, which is the fourth in the series, is the survey undertaken in England in the academic year 2016 to 2017. The preference for the oral health survey dataset in this study is because it is the same dataset used by the National Health Service (NHS) and local authority commissioners to monitor health improvement and plan health needs assessment in England (Davies et al., 2018). The data is also the singular source of caries prevalence information for both Public Health Outcomes Framework and NHS Outcomes Framework (Public Health England, 2019, Department of Health and Social Care, 2016).

Similarly, the UK Data Service hosts the largest collection of social, economic and population data resources (Pollard et al., 2017). One of the datasets within the UK Data Service is the UK census dataset that is regarded as having the largest collection of social determinants of health information in the UK (Ware, 2002). The census dataset was the main dataset used in this study, however, this was supplemented with data obtained from the Office for National Statistics (ONS), whenever research student found deficiency in the census dataset, as demonstrated by the Centre of Disease Control (Centers for Disease Control and Prevention, 2018). The mapping of SDH risk factors and data source used is summarised in Table 15.

Table 15: Social Determinants of Health Datasets

Domain	Data Source	Table Name
Place	Census 2011	Lower Tier Local Authority Area
	Office for National Statistics	Urban Rural Area
	Office for National Statistics	English indices of deprivation 2010
Race	Census 2011	QS203EW: Country of birth (detailed)
	Census 2011	QS204EW: Main language (detailed)
	Census 2011	QS205EW: Proficiency in English
	Census 2011	QS211EW: Ethnic group (detailed)
	Census 2011	QS802EW: Age of arrival in the UK
	Census 2011	QS803EW: Length of residence in the UK
Occupation	Census 2011	KS601EW: Economic activity, local authorities in England and Wales
	Census 2011	KS106EW: Adults not in employment and dependent children and persons
	Census 2011	KS611EW: NS-SeC, local authorities in England and Wales
	Census 2011	KS608EW: Occupation, local authorities in England and Wales
	Office for National Statistics	Jobseeker's Allowance with rates and proportions
Gender	Office for National Statistics	Lower layer Super Output Area population estimates (2016/17)
Religion	Census 2011	QS208EW: Religion
Education	Census 2011	KS501EW: Qualifications and students
Socio-Economic Status	Office for National Statistics	Regional gross disposable household income (GDHI) by local authority
	Office for National Statistics	Annual survey of hours and earnings - resident analysis - 2011

Social Capital	Census 2011	KS405EW: Communal establishment residents
	Census 2011	KS107EW: Lone parent households with dependent children
	Census 2011	KS103EW: Marital and civil partnership status
	Census 2011	KS301EW: Health and provision of unpaid care
	Census 2011	KS105EW: Household composition
PLUS	No data found at LA Level	PLUS 1: Child's Birth Order
	No data found at LA Level	PLUS 2: Parental Oral Health Behaviour
	No data found at LA Level	PLUS 3: Maternal Age
	No data found at LA Level	PLUS 4: Support with Oral Hygiene
	No data found at LA Level	PLUS 5: Health Insurance
Others	Public Health England	Oral health Survey 2016/17
	Public Health England	Water Fluoridation

6.2.2.1 Public Health England datasets

The Public Health England is responsible for facilitating the national programme for dental epidemiology surveys, and one of the surveys they oversee is the Oral Health Survey of 5-year-old children. This survey takes place every two years and the dataset used in this chapter is the Oral Health Survey of 5-year-old children, conducted in 2016/17 academic year. The main aim of the Oral Health Survey is to determine the prevalence of caries among 5-year-old children at local authority level.

Further details of the Oral Health Survey of 5-year-old children data showed that the survey 2016/17 was carried out in 134 out of 152 upper-tier local authorities (LAs) in England, which results in the survey data being collected from 303 out of 324 lower-tier local authorities (i.e., lower-tier is the primary sampling unit). Five-year old children attending mainstream schools were randomly selected and the children were examined, after obtaining explicit consent from the parents, and these data were linked to home postcodes in order determine both their lower super output areas and their deprivation level, using index of multiple deprivation (IMD) scores.

Finally, the outcome variable of interest in this dataset is caries prevalence, which is defined as the percentage of five-year old with decayed, missing and filled teeth (due to caries) that is greater than 0, by local authority area. The strength of the Oral Health Survey is that it contains the largest sample size for assessing caries prevalence in five-year old children, with a total of 96,005 consented children had clinical examinations, representing 96.2% of the main consented sample. This sample size represents 13.6% of five-year population attending mainstream schools. Also, these datasets were aggregated at both upper and

lower tier level, therefore making it easier to compare caries prevalence and inequalities between one local authority area and the other.

Water Fluoridation dataset

Public Health England (PHE) has a legal duty to monitor and report on the health effects of water fluoridation schemes on the population living in water fluoridated areas in England years (Public Health England, 2018c). This report is produced every four years and the latest data used in this study is the 2018 monitoring report that has details of all local authority areas that have "ever been water fluoridated" between 2005 and 2015 (Appendix 45).

6.2.2.2 Census datasets 2011

The census survey is a legal exercise that is planned and implemented by the government. Census is carried out every ten years in the United Kingdom, and the most recent census was conducted in 2011 by three organisations: the Office for National Statistics (ONS) for England and Wales, National Records of Scotland (NRS), and the Northern Ireland Statistics and Research Agency (NISRA). The strength of using census data in this exercise is that it contains the most complete data source holding both the socioeconomic and demographic characteristics for the United Kingdom (Hakim, 1982, UK Data Service, 2013). This data is aggregated into small area local authority in England and Wales that makes it possible to compare one area with another.

6.2.2.3 Office for National Statistics (ONS) datasets

The ONS is the primary UK government agency that is tasked with the collection, analysis and publication of statistical information relating to the economy, population and society of the UK (Jackson, 2013). They are the largest independent producer of official statistics in the UK (Anderson and Newing, 2015). This study considered ONS as a good resource because it collects population level data on health and social care, as well as, information on the labour market, education and skills, economy and environment, to mention a few.

Ethical Considerations

This study is based on secondary analysis of publicly available survey datasets, where all patient identifiers have been excluded from the oral health datasets.

6.2.2 Analytical methods

This section described a multi-step statistical process used to analyse the data. The first step carried out was data exploration, where each variable was assessed for missingness, skewness and multicollinearity and mapped to SDH variables identified in Table 8. Descriptive univariate analyses were then conducted on variables in order to demonstrate the characteristics of each variable, their measures of central tendencies (mean, median, maximum, minimum, and standard deviation). Step two involved dealing with the issues of missing data, where all missing data were imputed using MICE as described in chapter 5 (Section 5.2.4). The dataset used in this chapter has high-dimension, i.e., there are many columns (variables) in the dataset, therefore step three is the dimensionality reduction phase in order to reduce high dimensions (and inadvertently, reduce the collinearity) in the dataset. The final step (Step 4) is the development of a predictive model, including model

evaluation. All analyses were performed using the R statistical software, version 3.5.0 (The R Foundation for Statistical Computing; http://www.r-project.org).

6.2.2.1 Methods for dealing with missingness

As discussed in chapter 5, it is always advantageous to impute missing numbers because it helps to maintain the original sample size available for further analysis, therefore reducing the likelihood of loss of statistical power and precision (Vach and Blettner, 1995, Azur et al., 2011). Multivariate Imputation by Chained Equations (MICE) are preferred because MICE is capable of dealing with all three possible types of missingness (MCAR, MAR and MNAR) (Galimard et al., 2016, Mirmohammadkhani et al., 2012, Resseguier et al., 2011), including missingness due to non-response (Buuren and Groothuis-Oudshoorn, 2010, Miri et al., 2016, Durrant, 2005). Azur et al also mentioned that MICE has the ability to handle up to 80% missingness (Azur et al., 2011).

6.2.2.2 Dealing with Multicollinearity

One of the main challenges usually encountered when using a dataset with many independent variables (high-dimensional) datasets such as census data, is the high level of collinearity between the variables. Multicollinearity is defined as a situation whereby two or more predictor variables are highly correlated, i.e., when there is a linear relationship between two predictor variables, such that one predictor variable can be used to predict the other (predictor) variable (Iacobucci et al., 2016). While it is generally agreed that multicollinearity does not influence the predictive power of a model, the consequence of including variables with high correlation in predictive models is that the model produces

implausible regression coefficients estimates that are difficult to interpret (Chen, 2012). Multicollinearity is assessed using tolerance and the variable inflation factor (VIF), and results of tolerance greater than 0.10, or VIF greater than 10 imply high collinearity (Hair et al., 2006). Several techniques have been recommended for dealing with multicollinearity such as Dimensionality Reduction (Best Subset Regression and Stepwise Regression) which is a technique that eliminates redundant variables. Other techniques include the application of statistical methods that are very capable of addressing existing multicollinearity within the variables, such as Penalised Regression models or Principal Component Analysis or a combination of both. Each of these methods is later described below, including their advantages and disadvantages that influenced the method used in this thesis.

6.2.2.3 Feature Selection (Dimensionality reduction)

Feature selection is a process of reducing the dimension (independent variables) of the full model dataset by excluding redundant variables that do not contribute or that reduce the overall predictive accuracy, and selecting only the least number of variables that will give the best predictive accuracy (i.e., parsimonious model) (Devi and Rajagopalan, 2011). Dimensionality reduction is a necessary step in predictive modelling, because it helps us to select the more important variables, in order to avoid the issues of overfitting the model with too many variables. Overfitting occurs when a model fits a particular data so well, however, the resulting model cannot be generalised to another population (Prashanth and Roy, 2018). Another advantage of reducing high-dimensional datasets is that the decreased size also reduces the issues of collinearity when redundant variables (i.e., variables not contributing positively to the model) are eliminated. The possible disadvantage of

dimensionality reduction is the potential loss of valuable information from excluded variables, known as omitted variable bias (Chen, 2012). There are four main types of variable selection, which include: stepwise regression, criterion based (AIC, BIC and Adjusted R-squared) procedures, penalised regression and dimensionality regression (Faraway, 2002, Mooney and Pejaver, 2018, Neuvirth et al., 2011). Each of these techniques is further discussed below.

Criterion Based: Best Subset Regression

The Best Subset Regression is a process where every possible model combination is generated from the entire variables and the model with the lowest Akiake's Information Criterion (AIC) is considered the best. The advantages of Best Subset is that, unlike the stepwise models described below, it is not based solely on the single model that accounts for the highest amount of variability in the outcome variable, therefore, reducing the risk of missing important variables within the model (Goodenough et al., 2012). However, the weakness of the method is that it is computationally expensive, especially high-dimensional datasets. For a model with y variables, best subset regression produces 2^y possible models. Goodenough et al demonstrated that 1,073,741,823 models will need to be generated in a data with 30 predictor variables. They acknowledged that this will take over a year to run the model (Goodenough et al., 2012). It will therefore be practically challenging to use this technique on the developed caries dataset with 137 variables, which will generate $6.64613997892458 \times 10^{35}$ possible models.

Stepwise Regression

The stepwise regression is a process of adding and excluding predictor variables in an iteratively manner, until the best performing (predictive) subset of variables with the lowest predictive error is achieved. There are three types of stepwise regression (James et al., 2014, Bruce and Bruce, 2017): Forward Selection, which starts initially with no predictors and repeatedly adds variables that improve the model; Backward selection, which starts with the full model (all predictors), and removes predictors that are not contributing to the predictive model; Stepwise regression is a combination of the two, however, its main disadvantage is the likelihood of eliminating (highly correlated) explanatory variables that are important predictors of caries prevalence (Jabeur, 2017).

Penalised Linear Regression

Penalised Regression models, Principal Component Regression and Partial Least Squares Regression are the most popular modelling techniques used to address highly collinear datasets (Li, 2010). These modelling techniques are able to eliminate multicollinearity without needing to sacrifice any of the potential predictor variables, unlike in dimensionality reduction described above (Chen, 2012). Penalised regression model is a model of choice where the number of variables is large. This is because the model penalises the variables that are not adding to the regression by shrinking their regression coefficients to zero or close to zero (James et al., 2014, Bruce and Bruce, 2017). These models have been shown to produce better predictive performance in the presence of multicollinearity (Oyeyemi et al., 2015). There are three common types of penalised regression models, which are: Ridge Regression, Least Absolute Shrinkage and Selection Operator (LASSO model), and Elastic Net Regression, depending on their shrinkage tuning parameter (lambda) used.

Principal Component Analysis (PCA)

Principal Component Analysis (PCA) is a multivariate statistical technique that was introduced by Karl Pearson in 1901. PCA is considered a statistical analysis of choice when there are too many variables in a dataset, because it transforms original variables (without losing too much statistical information) into a new set of orthogonal or linearly uncorrelated variables known as principal components. The original variables are transformed into principal component variables using the formula below:

$$\frac{\boldsymbol{x_i} - \mathbf{mean(x)}}{\mathbf{sd(x)}}$$

Where mean(x) is the mean of x values, and sd(x) is the standard deviation (SD).

PCA also functions as a tool used to reduce high-dimension data because it creates a reduced number of variables that explains most of the variation seen in the original dataset. These principal components are arranged in the order of variables with most variance, i.e., the first principal component accounts for the largest possible variance in the dataset, followed by the second, and so on.

To conclude this section, research student used a combination penalised regression and stepwise regression in order to reduce the observed multicollinearity within the acceptable value (VIF <10). Penalised regression model is a model of choice in this study because of its ability to deal with very large dimensional datasets and also able to deal with multicollinearity (Oyeyemi et al., 2015). They are also able to extract the most important risk factor variables for the multivariable analysis by excluding all variables not contributing to the variance seen in the data.

6.2.2.4 Developing Caries Prevalence Predictive Model

Ten supervised machine learning techniques were applied in this study, including Linear, Stepwise Regression, Penalised (Ridge, LASSO, Elastic Net) regression, Partial Least Square, Support Vector Machine (SVM), Gradient Boosting Machine (GBM), Extreme Gradient Boosting (Xgboost) and Random Forest. These algorithms have been selected because of their abilities to use both parametric and non-parametric approaches, and have currently been used in several published medical literature (Seligman et al., 2018) (Namous et al., 2018, Jamali et al., 2016).

6.2.2.5 Model Evaluation

This section presents the planned steps for comparing the model performance of all the developed models. Predictive performance of the models developed were evaluated using a method known as ten-fold (k-fold) cross-validation, where the dataset is divided into 10 random subsets (Refaeilzadeh et al., 2009). For each k-fold, the algorithms were modelled on nine-tenth of the dataset and the remaining one-tenth used as the validation dataset, where the accuracy of the model is tested. Cross validation method is regarded to be a better way for assessing performance of predictive models (Bengio and Grandvalet, 2004). The three main metrics that were used to evaluate the models were R-squared, Root Mean Squared Error (RMSE) and Mean Absolute Error (MAE). R-squared provides information on the relative measure of fit of the model, MAE and RMSE both measure the average magnitude of errors between the predicted value and the true value. It should also be noted that the lower the MAE or RMSE, the better the model fit, and the higher the R-squared

values the better the model fit. Finally, the model with the highest R-squared was then selected among the models applied.

6.3 Results

6.3.1 Descriptive Statistics

A total of 96,005 children across 302 local authorities were clinically examined in this caries prevalence study (2016), and a total of 142 potential SDH variables matched on to one of the nine PROGRESS-PLUS classes identified in the systematic review criteria that was developed from chapter 3 (Appendix 44). Table 16 below shows a few of the important area-based characteristics by English region, however, the full statistical description of each variable such as minimum value, maximum value, mean (average), standard deviation, variance, skewness and kurtosis is also demonstrated in Appendix 42 to Appendix 45.

Table 16: Summary of area-based characteristics by region

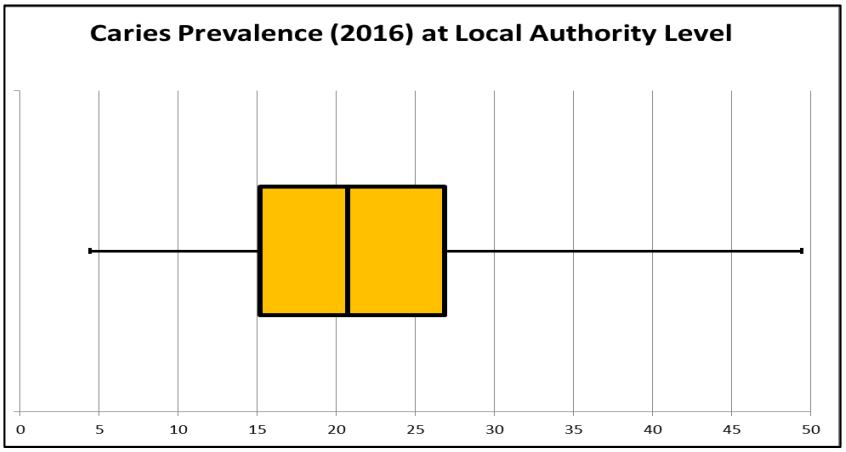
Region	*LA	Water Fluoridation	Average Caries Prevalence	% of "Area most deprived"	% "Non White"	% with "No Qualification"	% "Never worked"
East Midlands	40	8	22.4%	23%	7%	24%	4%
East of England	47	0	16.6%	4%	8%	23%	4%
London	32	0	25.6%	50%	39%	18%	8%
North East	12	2	23.8%	42%	5%	27%	7%
North West	39	2	32.5%	44%	8%	25%	6%
South East	67	0	16.0%	4%	9%	19%	4%
South West	36	0	19.8%	6%	4%	21%	3%
West Midlands	30	20	21.7%	20%	10%	25%	5%
Yorkshire and the Humber	21	1	27.0%	24%	7%	25%	5%
National Average			**23%**	**20%**	**11%**	**22%**	**5%**

***LA = Number of Local Authority**

The caries prevalence (2016) is summarised using the box and whiskers plot below (Figure 26). This plot showed significant variations in the proportion of five-year olds with caries

across English LA in 2016, with a minimum recorded prevalence of 4.4% in Horsham Local Authority in West Sussex (South East England) and the highest prevalence recorded was 49.4% in Pendle Local Authority in Lancashire (North West England). The average prevalence of caries in 5-year olds for all the local authorities was 21.7 (median = 20.8; standard deviation = 8.6)

Figure 26: Caries Prevalence 2016 Box and Whiskers plot



6.3.2 Dealing with Missingness

Twenty-two out of the 324 local authority areas had missing caries prevalence reports, which indicate 6.8% missingness. There are statistical evidence that have suggested that the failure to impute missingness are more likely to cause a reduction in the overall power of the study, and statisticians encourage missing data to be imputed, using appropriate statistical techniques (Sterne et al., 2009). Multivariate Imputation by Chained Equations (MICE) was used to input missing data, as discussed in chapter 5. Finally, two sets of datasets were developed and evaluated from the Oral Health Survey 2017: the first is a complete case dataset, which completely excluded cases (Local Authority areas) with missing caries prevalence data. The second dataset was the imputed dataset that replaced

missing values using MICE technique, which helped to maintain the original sample size. The summary statistics (e.g. mean, median, mode, etc.) of the dataset before and after the imputation is seen in Table 17 below, which shows that the characteristics of the final (imputed) dataset did not compromise the original spread of the data (Table 17).

Table 17: Data characteristics of Oral Health Survey 2017

Summary Statistics	**Raw data**	**Imputed data**
Count	302	324
Mean	21.7	21.7
Standard Error	0.5	0.5
Median	20.8	20.8
Standard Deviation	8.7	8.5
Sample Variance	74.8	72.6
Kurtosis	0.2	0.2
Skewness	0.6	0.6
Range	45.0	45.0
Minimum	4.4	4.4
Maximum	49.4	49.4

6.3.3 Multivariate Analysis

The penalised regression (elastic net) model had the best median R-square value (0.70) and the least MAE error of 3.6 after 10-fold cross-validation. A total of 32 (out of 142) important variables make up the elastic net model, therefore making the remaining 110 variables redundant. Furthermore, another 17 variables became redundant after subjecting the model to both forward and backwards stepwise regression and restricting to a model cut-off of p=0.05. The final caries prediction risk model was developed from 15 variables that are relevant to the prevalence of ECC as demonstrated in Figure 27 (see below).

Figure 27: Summary of dimensionality reduction process

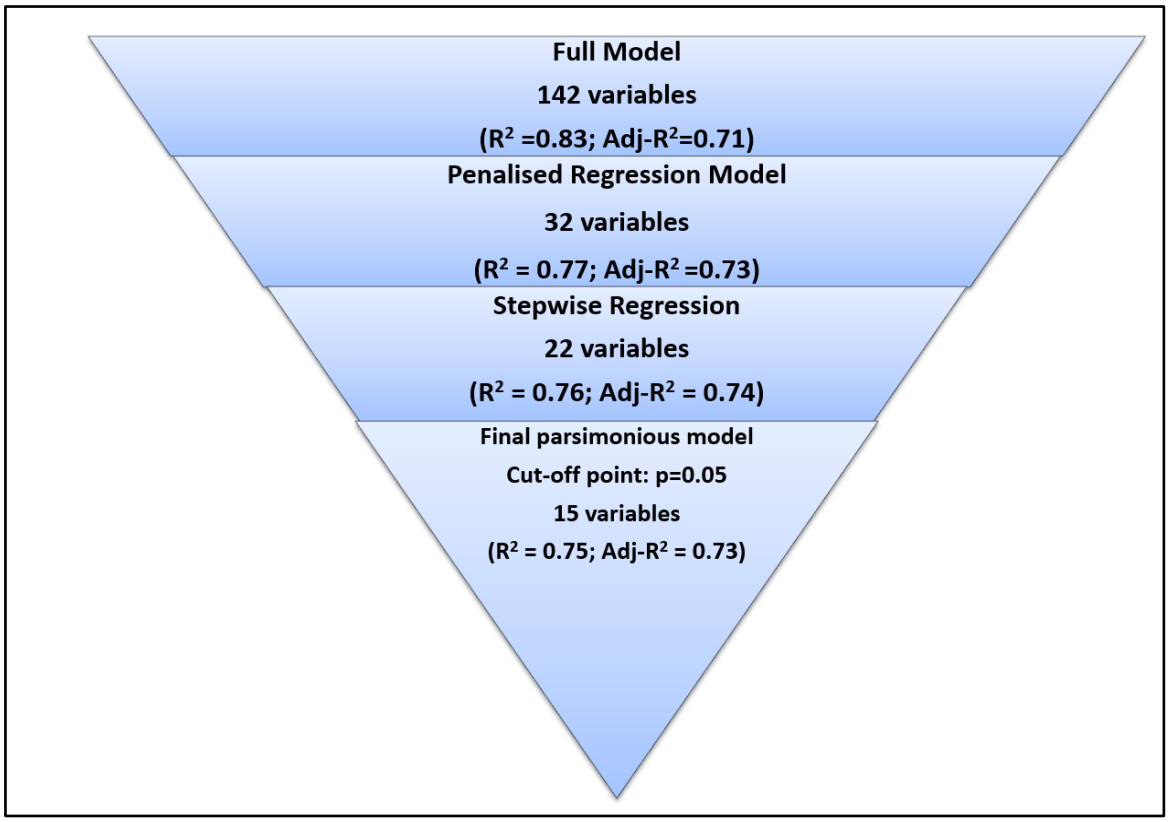


Table 18 (see below) shows the final variables used to develop the multivariate linear regression (MLR) analysis demonstrating the relationship between SDH and caries prevalence in five-year olds. In MLR, the relative relationship of each of the variables to the output (results) is expressed as the regression coefficient (third column in Table 18), unlike in logistic regression where odds ratios are presented. Where regression coefficients are positive, it demonstrates that the predictors vary directly with the outcome, and where they are negative, then the predictors and outcome have an inverse relationship. The characteristics of the model is interpreted as follows: The overall R-squared of 0.75 (Adjusted R-squared: 0.73) implies that approximately 75% of the variations seen in caries prevalence can be explained by this area-level risk model.

Table 18: Multivariate analysis demonstrating the relationship between SDH and prevalence of ECC in 5-year old in England

PROGRESS-PLUS Domain	SDH Risk	Coefficient	[1]LCI	[2]UCI	P-Value	code
	(Intercept)	8.38	-9.26	26.03	0.35	
PLACE	No Access to Water Fluoridation	3.72	1.95	5.49	0.00	***
PLACE	Region: East of England	-3.66	-5.32	-2.00	0.00	***
PLACE	Region: North West	5.63	3.61	7.64	0.00	***
PLACE	Region: South East	-3.20	-4.79	-1.61	0.00	***
PLACE	Region: Yorkshire and the Humber	4.37	2.17	6.57	0.00	***
RACE	Pakistani	0.64	0.43	0.84	0.00	***
OCCUPATION	Caring, leisure and other service occupations	0.88	0.24	1.52	0.01	**
OCCUPATION	Lower supervisory and technical occupations	-1.33	-2.24	-0.43	0.00	**
OCCUPATION	Routine occupations	1.49	1.12	1.86	0.00	***
OCCUPATION	Long-term unemployed	-4.33	-6.41	-2.24	0.00	***
OCCUPATION	Full-time employee	0.31	0.14	0.48	0.00	***
SOCIAL CAPITAL	Married	-0.28	-0.46	-0.11	0.00	**
SOCIOECONOMIC STATUS	Lone parent not in employment	0.33	0.16	0.50	0.00	***
RELIGION	Other religion	5.27	2.42	8.12	0.00	***
RELIGION	No religion	-0.47	-0.61	-0.32	0.00	***

Multiple R-squared: 0.75; Adjusted R-squared: 0.73; P-value < 0.001 = '***'; p-value < 0.01 = '**'

[1]LCI = Lower Confidence Interval; [2]UCI = Upper Confidence Interval

The entire 324 LA areas are grouped into nine regional areas, which is shown in Appendix 42 below. This table also demonstrates the number of local authorities, spread of deprivation (most and least deprived), and water fluoridation status by region. The risk model shows that a 1% increase in the population of 5-year old children living in either of the North West or Yorkshire and the Humber region also increases regional caries prevalence by 5.6% (3.61-7.64) and 4.37% (2.17-6.57) respectively. Children living in areas like the East of England and South East have also shown statistically significant protective capabilities with regression coefficients of -3.66 (-5.32 to -2.00) and -3.2 (-4.79 to -1.61) respectively. Finally, an important observation in this model is that it was able to underscore the relationship between water fluoridation and caries prevalence, where it showed that lack of access to water fluoridation increases caries prevalence of that local authority area by 3.72 (1.95 to 5.49). Caries prevalence are also influenced by how racially diverse the local authority is, where the findings of this study showed ECC prevalence is higher in diverse local authorities, especially Pakistani communities. The model showed that for every percentage increase in the Pakistani (five-year) population, there is a 0.64 (0.43-0.84) percent increase in caries prevalence of the LA.

This study also found that the higher the percentage of those classed as “routine workers”, “Caring, leisure and other service occupations”, and “Full-time employee” in the population, the higher the caries prevalence of the area. For every percent rise in routine workers in the area, the caries prevalence rises by 0.88 (0.24 - 1.52). On the other hand, the percentage of parents doing “Lower supervisory and technical” work in the population also influences caries, and reduces caries prevalence by approximately -1.33% (-2.24 to -0.43) for every percent increase. The percentage of those classified under “Other religion” influences caries

by a factor of 5.27 (2.42 - 8.12), and those classified under “No religion” reduces prevalence by -0.47 (-0.61 to -0.32). This model showed that “long term unemployment” has an inverse relationship with caries prevalence by a factor of -4.33(-6.41 to -2.24), however, an increase in “lone parent not in employment” increases caries prevalence by 0.33 (0.16 to 0.50). In this model, the percentage of married families per local authority area was found to be a significant protective factor and able to reduce caries prevalence by -0.28% (-0.46 to -0.11). Finally, the mathematical equation for caries prevalence at local authority level can be represented using the formula:

Caries Prevalence (Local Authority Area) = *8.38 + 3.72A – 3.66B + 5.63C - 3.2D +4.37E + 0.64F + 0.88G – 1.33H +1.49I - 4.33J + 0.31K – 0.28L + 0.33M + 5.27N – 0.47O*

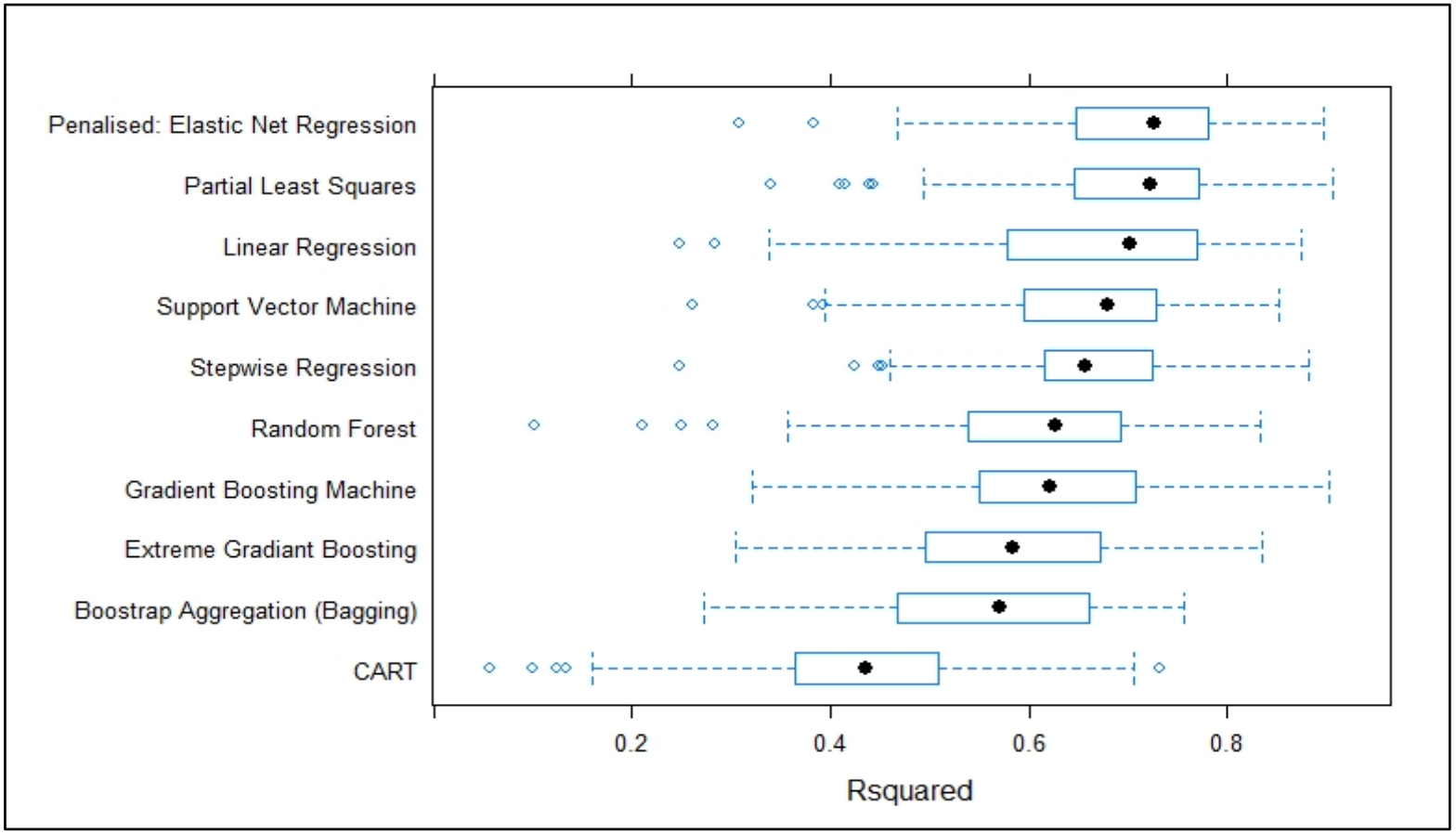
Where:
A = “No Access” to Water Fluoridation; B = Region: East of England; C = Region: North West; D = Region: South East; E = Region: Yorkshire and the Humber; F = Race: Pakistani; G = Occupation: Caring, leisure and other service; H = Occupation: Lower supervisory and technical; I = Occupation: Routine occupation group; J = Occupation: Long-term unemployed; K = Occupation: Full-time employee; L = Two-parent family; M = Lone parent not in employment; N = “Other” religion; O = No religion

6.3.4 Developing caries prevalence predictive model using administrative datasets

This phase of the research involved the development and the evaluation of several risk prediction models for caries prevalence at local authority level, where the entire (imputed) 324 Local Authorities were available for the analysis. Evaluating the predictability of the risk models summarised in Table 18, using linear and machine learning techniques, showed penalised regression to be the best predictive risk model out of the 10 supervised models explored. Penalised ridge regression model produced an average R-squared value of 0.74,

after 10-fold cross-validation, and predictive error showed mean absolute error (MAE) of 3.41, and root mean square error (RMSE) of 4.29 (Figure 28 and Appendix 46).

Figure 28: Figure showing the “R-Squared” rankings of the 10 models used (The higher, the better)



6.4 Discussion

Early childhood caries is an important public issue in the UK and globally and its aetiology is multifactorial and complex (Wiley et al.). Despite the recent decline in caries morbidity across the globe, children from lower SDH resources continue to develop caries at higher rates than children with means (Anil and Anand, 2017). This study, informed by the systematic review, provides a comprehensive evaluation of SDH risks underlying caries inequalities at local authority level. The aim of this study was to develop a caries risk model that contributes to a better understanding of the interrelationships between social determinants of health that is aggregated at local authority level and caries prevalence. Through this study, an understanding of characteristics of the areas with higher risk of caries is understood. The study findings suggest the possibility of predicting caries prevalence of an area from SDH. It has demonstrated that the characteristics of the place (i.e., geography) where a child spends most of their first five years is one of the most important caries inequalities risk factor. This occurs because wider determinants have been shown to be differentially distributed by place of residence concentrated (Kibria et al., 2018). The overall relative influence analysis from the best predictive model showed that growing up particularly in the North West and Yorkshire and the Humber posed a great risk to developing caries, followed by living in areas without water fluoridation. The North West region is one of the poorest regions and has the second largest number of Local Authorities in the most deprived quintile after London (Table 16). Studies have also demonstrated that there is a link between area level deprivation and access to dental services, where those living in deprived communities have been shown to be less likely to use dental services (Lang et al., 2008). Similarly, the observation of this study is consistent with other studies

that have equally reported similar direct correlation between exposure to water fluoridation and caries prevalence (McLaren and Singhal, 2016, Ran et al., 2016).

When it comes to ethnicity, this study also showed that there is a statistically significant higher level of caries risk in Pakistani children, relative to White children, whilst Black African/Caribbean children showed a lower (albeit not statistically significant) level of caries risk. This observation is consistent with previous UK studies, and some of the explanations for this observation is that Pakistani children become exposed to cariogenic food substance earlier than other children (Rouxel et al., 2018), and their mothers are more likely than other ethnic groups to add sugar to children's drinks (Gibbs et al., 2016). Researchers have also linked language barriers experienced by Pakistani mothers as well as their cultural beliefs about oral health (Marcenes et al., 2013).

The percentage of "Routine occupations" in a local authority is another important variable found to influence overall caries prevalence. According to the Office for National Statistics Socio-Economic Classification (NS-SEC) definition, "Routine occupations" are defined as *"positions with a basic labour contract, in which employees are engaged in routine occupations. These positions have the least need for employee discretion and employees are regulated by a basic labour contract."* This group of occupation are at the lowest rung of the ladder, after "not in work" group, in the eight-class occupation classification (Table 19). Some explanations for this is that children of parent with lower ranked occupation are more likely to consume energy-dense food than their counterparts (Vilela et al., 2015), and more unlikely to visit a dentist when requiring treatment (Kidd, 2012). Bremberg's study also highlighted that the proportion of adults in manual occupation is a strong marker for overall

health of an area's population (Bremberg, 2002). Occupation has also been used in other studies as a marker for socioeconomic position (Adomaviciute et al., 2015, Thomas et al., 2012, Yau et al., 2018)

Table 19: ONS Standard Occupational Classification (2010)

Class	Definition
1	Higher managerial, administrative and professional occupations
2	Lower managerial, administrative and professional occupations
3	Intermediate occupations
4	Small employers and own account workers
5	Lower supervisory and technical occupations
6	Semi-routine occupations
7	Routine occupations
8	Never worked and long-term unemployed

Finally, there were two unexpected findings observed in this risk model, which will require to be further investigated. The first was that the larger the population of parents with "long term unemployment" status, the lower the caries prevalence in the area become [-4.33; CI = --6.41 to -2.24; P-value = 0.000]. This observation is consistent with the study by Al-Sudani et al that showed that there is an association between "Long-term unemployment" in mothers and higher number of sound teeth and fewer number of filled teeth (Al-Sudani et al., 2015), however, their study was unable to describe the pathway of how "Long-term unemployment" exerts its effects on caries. "Long-term unemployment" is directly proportional to material deprivation, income poverty, poor housing quality and poor neighbourhood conditions (Delgado-Angulo et al., 2009), and therefore, one would expect caries risks of preschool children to be greater in such scenario. The second unexpected finding of this study showed that the larger the population without religion, the lower the caries prevalence of the area [-0.47; CI =-0.61to -0.32; P-value < 0.000]. This group were made up of 24.7% of the entire population in the 2011 census. Similarly, the larger the

percentage of those classed as “other religion” in the society, the greater the caries prevalence of the area [5.27; CI= 2.42-8.12; p-Value = 0.000]. Other religion was defined in census data as those not practising one of the big six religions in the UK, which are Christianity, Muslim, Hinduism, Sikhism Judaism, and Buddhism), and this group is made up of 0.4% of the entire population in the 2011 census. Conway et al study emphasised that there is usually a strong relationship between religion and ethnicity, and the study cited an example where they found that there is a strong association between reported Pakistani ethnic origin and Muslim religion (Conway, 2007).

6.5 Strengths and Limitations

The strength of this study is that it is the first known study to consider the risks posed by all the possible “place of residence” characteristics at once. This study also used entirely wider determinants variables to develop the caries prevalence risk model. The main strength of this chapter is that all risk factors (SDH) used to develop the predictive models were obtained from publicly available datasets, while none of the oral behavioural risk factors was included in the model. These models were also subjected to a series of machine learning techniques, which are statistical techniques that are able to assess several millions of variable relationships, in order to learn from the data and ultimately produce the best model combination with the best risk score (Krittanawong et al., 2017). Machine learning is fairly new in medical science, and the techniques have been encouraged mainly because of their remarkable accuracy and reliability in predictive modelling, which can assist clinicians in making better clinical decisions (Krittanawong et al., 2017).

Another strength of the study is that it is the first study to link multiple SDH datasets from four different publicly available data sources together into one main SDH dataset that can be used in other social inequalities studies. This is an important step because it is assumed that humans are exposed to several thousands of SDH risks at the same time, and linking all available SDH sources into one provides the opportunity to test the impact of all SDH, simultaneously, on developing diseases. In this study, further analyses on the composite data helped to extract relevant SDH variables from redundant variables, which achieved better results than the popular variable pre-selection that is based on a best educated guess.

The final strength of the study is that the study is based on a very large sample size of 96,005 five-year old children across the country. A large sample size ensures that the study has enough power to detect differences between groups, and it provides a more precise estimate of model statistics in almost all cases, with only a few exception (Biau et al., 2008, Nayak, 2010). Large sample size is also advocated for in epidemiological studies because it guarantees representativeness of the population and therefore improves the generalisability of the result (Biau et al., 2008).

There are two major limitation of this study, the first is the use of survey data from different sources to develop a risk model. Administrative datasets are known to be relatively more prone to inaccuracies from data entry than data from other study type (Shin and Scherer, 2009, Tirschwell and Longstreth, 2002). The second limitation observed was the lack of detailed information on the actual numbers of children who have moved into or out of the local authority area ("loss to follow up"), although, data from the Office for National

Statistics suggests this as an average of 5.2% of five-year olds born in 2012 experienced internal migration in 2017 (Office for National Statistics, 2016) (Table 20). A study of this type would benefit more from longitudinal studies, where the children born during the 2011 census are followed up and assessed for preschool caries after five years.

Table 20: Rate of internal migration in five-year old starting from 2012 to 2017

Year	Age (Years)	% internal migration
2012	0	3.6%
2013	1	6.6%
2014	2	6.2%
2015	3	6.0%
2016	4	4.8%
2017	5	4.1%
	Average	**5.2%**

6.6 Implications of Oral Health Survey Prevalence Study

Implications for policymakers

One of the implications of this study is that it encourages the government to review the current oral health preventive programmes, where emphasis placed on oral behaviour is more than that placed on wider determinants (Heilmann et al., 2016, Watt, 2015). Current Oral health prevention programmes may be reviewed such that it incorporates governments commitments to addressing wider determinants too, especially place-based SDH, and relatively more health investments should be considered in areas predicted (based on SDH needs) to have higher caries prevalence, using a universal proportionate approach, proposed in the Marmot Review (Fair Society, Healthy Lives) (Marmot, 2010). According to a Public Health England report, dental commissioners are currently piloting innovative commissioning in order to improve child oral health in 13 priority areas in England (Public

Health England, 2017). One advantage of employing a predictive model in health commissioning is that it will help to determine which areas need to be prioritised, and ensure decisions for health investments are made based on predicted prevalence of the local authority area, and not after the damage has already been done, which is what currently takes place. This also ensures that the area-level risk of developing ECC is quickly abated even before they manifest in children's teeth. The tool could also be substituted for the rigorous and more expensive routine oral health surveys that is carried out every two years in England and Wales, or used in-between the surveys to assess caries prevalence, which will ultimately result in some financial cost savings to the NHS. Incentives may also be needed to encourage dentists to set up practice in areas predicted to be of greatest need, as the findings of this study showed that areas with greater number of dental clinics per local authority area fared better than areas that are not. Regions like the North West area and Yorkshire and Humber need significant dental investments in order to reduce the risk of developing caries in five-year old children living in those areas.

Another important implication is policymakers will be propagating unfairness if there is no programme to deliberately target Pakistani children separately. Pakistani children have equally demonstrated higher risk of developing caries when they are compared with non-Pakistani children, and this is consistent with literature and with the result findings in chapter 5, using a completely different data source and methodology. Pakistani children will need to be identified earlier in order to administer appropriate preventive measures. There is also a need to develop training packs that raise awareness of SDH risks to look out for, which can be included as a verifiable continuing professional development for dental professionals. Finally, the current NICE guidance on caries risk assessment criteria

recommended for dentists will also need to be reviewed because it is lacking in majority of the relevant evidence-based wider determinants that have been identified as risk factors of developing caries in preschool children. This NICE guidance only had two relevant SDH risk factors questions (high caries in mother and siblings and exposure to water fluoridation) out of the possible 24 questions required to assess caries preventive need and the frequency of recall.

Implications for children

One major implication of this for children is that advice for preventive visits will vary per groups of children. Up until now, routine dental recall is mainly based on age of the patients, unless there is already a caries history in the child or family. So, five-year old children are advised for a recall visit between 3 – 12 months, depending on identified needs that is based on risk assessment (NICE, 2004). However, implementing this model nationally will imply that some groups of children should receive more frequent recalls than others, i.e., frequency of recalls for groups of children are informed by SDH-based caries model. For example, a group of five-year old Pakistani child of a low-educated parent, living in North West region have a higher risk of developing caries and have enough justification for more frequent recalls than another group of five-year old non-Pakistani children living in South East region, and of well-educated parents. This section concludes by acknowledging that some negative implications would be expected, where some groups will feel unfairly targeted or discriminated on, by suggesting that their risk is higher and therefore require more recall visits, even after they have demonstrated excellent oral health behaviour.

In summary, this chapter was able to demonstrate that it is possible to predict caries prevalence of a local area, with over 70% accuracy, using publicly available administrative datasets.

CHAPTER SEVEN: GENERAL DISCUSSION AND CONCLUSIONS

7.1 Introduction

In this thesis, the wider determinants responsible for caries inequalities in preschool children were explored using a combination of epidemiological tools, such as, systematic review, path analysis, regression analyses and machine learning techniques. The systematic review was carried out to investigate all possible social deprivation risk factors that influence caries inequalities in five-year old children, and to demonstrate best practices in caries risk modelling that was lacking in existing conceptual models. This study is the first study to seek and retrieve evidence using the Cochrane-validated equity tool (PROGRESS-PLUS). Using PROGRESS-PLUS helped to ensure that all possible caries risk factors driving inequalities are explored, and also helped to identify where there are gaps or absence of evidence within any of the PROGRESS-PLUS categories. Two different approaches were undertaken in this thesis, the first investigated the SDH factors operating at child-level and the other explored risk factors that are important in determining area-level caries prevalence. Path analysis was then carried out to understand the direct and indirect causal relationships of all the risk factors identified from the review, and create a conceptual model of how they contribute to causing caries, using evidence-based causal theories. Finally, series of statistical analyses were conducted to validate the caries inequalities risk model developed, using publicly available oral health surveys and administrative datasets.

This chapter is divided into four sections. The first section synthesises the findings from the four previous chapters (chapters 3-6) and discusses the relationship of key findings to existing literature. The second section explored strengths and limitations of the study and the third

section discusses the implications of this PhD for public health policy, clinical practice and patients, and for further research. The final section provides recommendations and summarises the main conclusions of the thesis.

7.2 Section 1: Summary of the findings

7.2.1 Research objective one: To carry out a systematic review in order to identify wider determinants influencing development of caries in in children.

The findings of the review identified a range of 24 wider determinant risk factors, from 67 studies, that exert their influence on caries experience during the first six years of a child's life. The studies identified in this review showed that there are four important area-based characteristics that could act as a risk factor of caries in preschool children, which are local policy of the area (exposure to water fluoridation, well-funded, equitable local health system and access to universal dental insurance), region of residence, deprivation of the area and exposure to environmental smoking. The findings of this review is also consistent with that of other studies, for example, those living in rural or deprived areas have limited access to preventive dentistry infrastructures (Ohsuka et al., 2009), limited access to healthy food options (Ju et al., 2016), as well as, limited job opportunities (Ju et al., 2016). Caries experience was also higher in children from "non-indigenous" background, in children whose parents were born abroad, and those children living in families where mother tongue was different from the official national language. Relatively lower income observed in non-indigenous families is one explanation for the ethnic risk differences in caries in the United Kingdom (Marcenes et al.,

2013), and the lack of adequate financial resources eventually leads to inability of the child to attend preventive dental visit or to afford healthy diet (Kim et al., 2012). This is the same mechanism (relatively lower income) by which parental occupation, parental education and family socioeconomic status mainly exert their influence (Wardle et al., 2002, Ball et al., 2002). For social capital, mothers' marital status provided the biggest influences on caries and on overall development of the child (Goodman and Greaves, 2010, Fisher, 2010, Nikolaou, 2012). Goodman and Greaves' study particularly highlighted that combined resources (from both parents) provide the family with better healthy options to choose from (Goodman and Greaves, 2010).

7.2.2 Research objective two: To map existing caries models against SDH identified from review in order to identify gaps within existing models.

Although there have been several conceptual risk models developed in order to understand the relationship between the various risk factors in ECC, however, the findings of this study showed that none of these models was developed with a consideration of the full wider determinants that are responsible for health inequalities. According to findings from McTiernan et al (1997), the precision of risk models hinges on robust identification of risk factors and the demonstration of how these factors operate in the presence or absence of other factors (McTiernan et al., 1997). This mapping exercise helped to identify the weaknesses of existing conceptual models and, therefore, can safely extrapolate that existing risk models are more likely to be inadequate to address the issue of caries inequalities because none of the existing caries inequalities model considered (and incorporated) anything greater than 48% of the 24

possible wider determinants identified from the systematic review (Table 2 and Table 8). The two-possible reason for the omission of important wider determinants are, firstly, risk model developers' main aim might have centred only on the reduction of caries prevalence, without the consideration of reducing inequalities. Secondly, in those who considered inequalities, the model developers possibly overlooked or underestimated the importance of a critical step during risk model development, which is the use of best evidence when developing conceptual risk modelling. A joint paper from the World Bank and WHO explained that the solution to current health inequalities is through the introduction of equitable health models within our societies (Kim and Chan, 2013). Inclusion of wider determinant risk factors in disease models also increases the chances that preventive measures, built on SDH models, will be able to address both prevalence and inequalities. In conclusion, there is an obvious gap to improve existing caries risk models and the findings of this study demonstrated current models are inadequate and may not be fit for purpose of reducing inequalities in preschool children. The lack of consideration for factors contained in a robust equity-focused caries model in prevention decisions may be the cause of the current low prevalence, but widening/stagnant caries inequalities pattern seen school children aged 6 years and under globally. Social deprivation disempowers people, which ultimately prevents them from making healthy behaviour choices than those not deprived (Friel, 2009). This PhD equally demonstrated that SDH are the ancestors of oral health behaviours like oral hygiene and preventive dental visits (Figure 25). Therefore, wider determinants derived from a thorough primary research or systematic review exercise could always be a major component of conceptual caries models' development, in order to avoid perpetuating the unfairness and avoidable caries inequalities

among our socially, economically, demographically or geographically diverse preschool children.

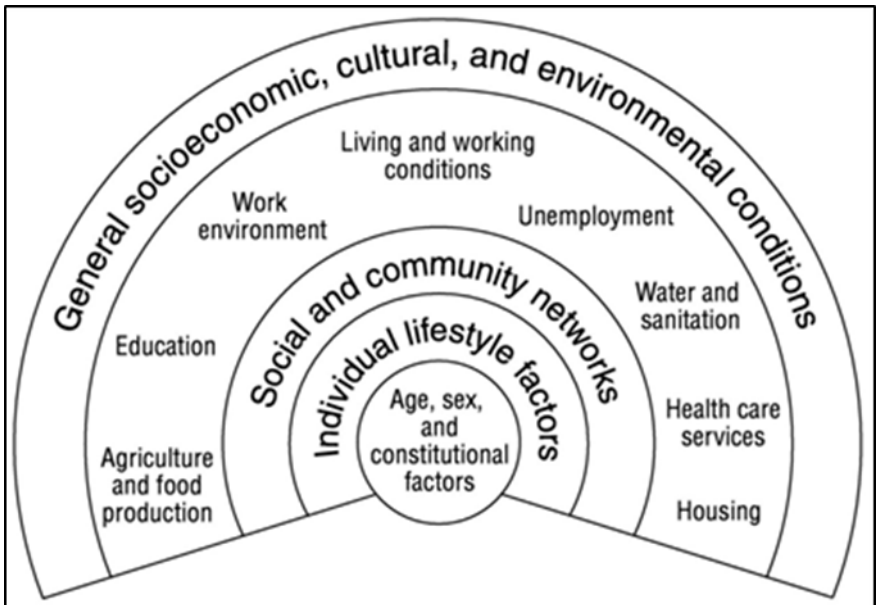
7.2.3 Research objective three: To develop a conceptual caries model that describes causal pathways between wider determinants and caries in children

The lack of an appropriate caries risk model increased the need for developing a new equity-focussed caries model for children under the age of six. Understanding how the risk factors interrelate to one another in a causal pathway is the first step in disease prevention. This knowledge makes explicit the causes of causes and thus highlights the most appropriate combination of interventions that are likely to be effective. Having synthesised the information gathered from the first three chapters, this thesis therefore demonstrated that the process of developing a robust conceptual caries inequalities model is best implemented in a three-dimensional space, that is; the disease modelling process employed in this thesis ensured that the length (association), width (temporality) and depth (hierarchy) of the caries model was well defined. Chapter 4 emphasised that these three planes are the main rudiments of causality and are best incorporated into conceptual modelling in order to ensure that the "interrelationship" between the risk factors is known, the "time of the risk" (i.e., the time between "cause" and "effect") is ascertained and the "level of operation" of the risk (i.e., the level at which the risk is exerting its influence) is established. This is the second gap that was observed in existing preschool caries models, where caries modelers failed to consider these three domains in their models. The recommendation to define the three planes is important for conceptual caries modelling because it ensures that disease pathways are clearly understood, which will aid

policy makers during preventive policy development. The first dimension defines the domain of "what" to intercept (association), the other (temporality) describes "when" to intercept, and the third dimension is about understanding "where" the best level pitching the prevention. Of these three, temporality is the only known, absolutely essential factor in causality (Staplin et al., 2016).

This thesis finally developed a new caries inequalities conceptual model that can be adapted to meet the needs of most countries, depending on the type of health system implemented and routine data collected in that country. Contrary to existing models, the conceptual model developed showed that parental education and occupation are the most distal determinants of caries inequalities in five-year old children. In the United Kingdom, a child's "place of residence" and the "local health system" they have access to, are offspring of their parental level education and occupation. This is the third weakness observed in existing caries models, where these models have been patterned against a universal SDH template, such as the most cited SDH rainbow proposed by Dahlgren-Whitehead (Figure 29) or the WHO Structural Determinant Conceptual template (Figure 2). These universal SDH templates rightly place all environmental related risks as relatively distal, and others such as parental education and occupation as relatively proximal. However, the findings of chapter 4 demonstrated that parental education and parental income kick-start the caries inequalities risk model for children under 6 years of age.

Figure 29: An example of SDH proposed by Dahlgren-Whitehead



One explanation why published caries models would have taken this approach can possibly be attributed to their lack of conceptualisation of risk models in the three-dimensional space (association, time and hierarchy) prior to model development. Lämmle et al. defined a distal determinant as a risk factor, relative to other risks in the model, that is able to influence health behaviours via proximal determinants (Lämmle et al., 2013). Published evidence also support that parental education and parental occupation both determine family socioeconomic status, which in turn influence where a five-year-old resides (Kato et al., 2017). The issue of applying a universal approach to modelling is better illustrated using this example that highlights that there are different causal pathways between adult caries and childhood caries. In adult caries, environmental risks such as “place of residence” and “local health system” may be relatively distal to one’s education and occupation and socioeconomic status; however, causal pathway reverses in early childhood caries, where the most distal risk in the caries pathway is determined by the “kind of parent” the child has in those first six years (Figure 25). If we also

consider the level of difficulty of modifying the risks within the first six years, distal risk factors in this new model pose the most challenging to address. In fact, Nancy Krieger's article, Proximal, Distal, and the Politics of Causation, discouraged the use of "distal" and "proximal" risk factors because of this type of misuse in application (Krieger, 2008), and advocated for the use of levels and pathways of action, in order to demonstrate simultaneous complex interrelationships between wider determinants (Krieger, 2008, Shi and Singh, 2011). Finally, the proposed caries conceptual model incorporated all three dimensions described above, which was then validated using the Child Dental Health Survey 2013 dataset.

7.2.4 Research objective four: To use knowledge from outcome three to develop a caries predictive risk model, using administrative datasets

7.2.4.1 Individual level risk model

This section of the thesis used administrative data to extend the argument that wider determinants risk factors, after excluding behavioural risk factors, might be sufficient to reproduce the caries pattern seen in five-year old children. This quantitative model can also be used to predict future caries in an individual or predict caries prevalence in a local authority area. The first quantitative risk model evaluated risk at individual (child-level) and the findings highlighted that caries risk of a five-year old child may more than double significantly, depending on where the child lives. The explanations for the caries risk differences by place of residence are mainly due to the differences in access to healthcare, as well as, the

socioeconomic divide, especially between North and South of England, that have already been highlighted in other studies (Doran et al., 2004). This risk model also confirmed that some ethnicities predispose some children more to caries than others. In the UK, children of Asian background, particularly Pakistani children, are more likely to develop caries by the age of five. This is in agreement with other published studies and the two main links between Asian children and caries is the level of parental education (Conway, 2007) and this children are also known to be introduced earlier to cariogenic food substances (Rouxel et al., 2018).

This study equally emphasised that parental level of education is an essential distal determinant of childhood caries. Crocombe et al (2018) study revealed that children whose parents had higher education levels have nearly half the relative risk of developing caries, when they are compared with children whose parents had low levels of education (Crocombe et al., 2018). In a systematic review and meta-analysis, the authors found that the association between educational background and developing caries was relatively (and significantly) higher in countries with high human development index (HDI), especially western countries like United Kingdom and the United States (Schwendicke, 2015), and education is the main determinant of parents' labour market status, which eventually determines their income, housing (place of residence) and other material resources. Finally, being educated ensures that one is well informed about the use and availability of services, and it empowers the citizens about their (health and wellbeing) rights and responsibilities.

The risk prediction model developed was ranked among the four highly regarded caries risk assessment tools (Fontana, 2015, Gao et al., 2013) and its uniqueness is that it is the only tool

that assessed wider determinants without clinical, microbiological or salivary tests, and achieved an accuracy that was ranked second in the list (Table 12 and Appendix 40). The SDH-based risk model also demonstrated a high level of specificity (91%) and low sensitivity (34%) scores.

7.2.4.2 Area-level prevalence risk model

The development of an area level caries model was a decision that came after investigating caries risk factors at child level, where all area-based characteristics assessed (IMD deprivation, region, Urban-rural and Output Area Classification (2011) groups showed significance at bivariate, multivariate and multilevel analyses), therefore, the aim of the second area-based risk model was to provide answers to the question: **"*What are the factors responsible for the differences in caries risks seen between two local authority areas in England?*"**. This is the first known study to put together a predictive risk model for caries prevalence at local authority level.

The findings revealed that the determinants of caries prevalence within a local authority area goes beyond these four area characteristics explored at individual risk level, which are, water fluoridation status, Output Area Classification (2011) characteristics, regional area, and area deprivation status. The other relevant caries-prevalence influencing factors include: the percentage of non-indigenous families within the population, the distribution of the parental job types within the population, percentage of single-parent families, and religious practices. The implication of this knowledge, when taken into consideration, is that it can help to inform

Public Health on resource allocation. Local authorities with larger percentage of “at risk” groups of children might be able to address an impending high ECC prevalence by committing relatively more caries preventive resources than others.

Another contribution of this study is that it helped to emphasise the need to explore the “religion gap”, where there was absence of evidence in the influence of religion on developing caries. This thesis showed that religion is important and should be considered an important caries prevalence risk factor. The evaluation of wider determinants at area-level (chapter 6) showed that the larger the percentage of those with “no religion” (24.7% of population in 2011 census) in the society, the lower the caries prevalence of the area, that is, not having a religion exerts a protective influence on the population and this observation is statistically significant [-0.47 (-0.61 to -0.32)]. On the contrary, the percentage of those classed as “other religion” in the society, although this is a very small population (0.4% of population in 2011 census), also influenced (negatively) caries prevalence in a local authority area [5.27(2.42-8.12)].

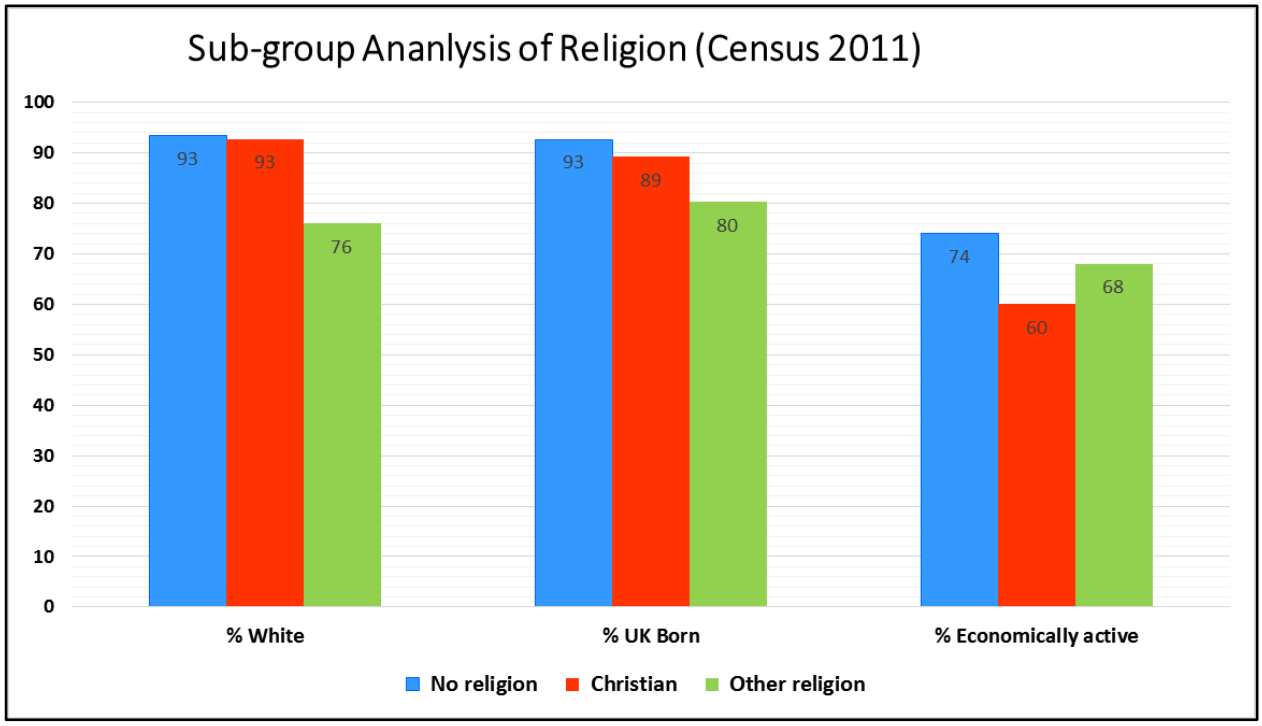
This link between lack of religion and caries remains unclear, but a few possible explanations from further sub-group analysis showed that majority of those without religion in this study lived in the southern part of England, with the exception of London (Table 21). Similarly, the 2011 census report also revealed that majority (93.4%) of those without religion were White compared to 76% White in “other religion”, 93% are born in the UK, compared to 80% with “no religion” and 74% are economically active (compared to 68% in “other religion”) (Office for National Statistics, 2013) (Figure 30). The protective effect posed by having larger people with

"no religion" is possibly mediated by a combination of other underlying socioeconomic differences between the children. Therefore, religion could be used as a proxy for other relevant SDH risk factors.

Table 21: Ranked "No Religion" by region of residence

Region	Percentage of "No Religion"	Average Caries Prevalence
South West	28	19.8
East of England	27.8	16.6
East Midlands	27.1	22.4
South East	27.1	16.0
Yorkshire and the Humber	24.9	27.0
North East	23.5	23.8
West Midlands	22.9	21.7
London	21.1	25.5
North West	19.4	32.5

Figure 30: Subgroup analysis of religion (Office for National Statistics, 2013)



In conclusion, this PhD found that the risk of a five-year old developing caries is determined essentially by parental education, parental occupation, the place where the child lives, ethnicity, and household composition (single or two parent). Dental awareness level of the main carer was also found to be significant. Similarly, the risk of a relatively higher caries prevalence is determined by place of residence (exposure to water fluoridation and region of residence), race (percentage of non-indigenous children per local authority), type of occupation (percentage of caring, leisure and other service occupations, lower supervisory and technical occupations, routine occupations, full-time employee per local authority, long-term unemployed, lone parent not in employment), religion (other religion, no religion) and social capital (percentage of single-parent families).

7.3 Section 2: Strengths and Weaknesses

One of the main strengths of this study is the quality of evidence used in the systematic review. A robust search strategy was developed that purposefully excluded studies unable to demonstrate temporal precedence, such as, cross-sectional studies. Establishing temporal precedence is the single most important factor in causality (Mann, 2003), therefore, this exclusion helped to improve the quality of evidence used in the systematic review. The search strategy developed was also guided by the Cochrane – JBI validated PROGRESS-PLUS equity focussed tool, which therefore ensured that all possible SDH risk factors were considered in the thesis. There were also an abundance of relevant papers retrieved, 67 papers, which therefore

allows for the use of cohort studies entirely in situations where there are enough cohort study papers.

Secondly, this study developed two separate conceptual ECC frameworks, instead of one, thus highlighting the two possible relationships that SDH risk factors have with one another – temporal relationship and hierarchical relationship. This study observed that temporal and hierarchical ECC risk factor relationships follow different pathways, and therefore will be quite challenging to conceptualise temporality on hierarchical pathway. This may be one reason why majority of existing frameworks did not include temporality; and was only two out of the 14 existing ECC frameworks explored that attempted to demonstrate it in their frameworks.

The validity of the temporal model was then tested, using epidemiological data, which is regarded as a good practice in risk modelling (Kruijshaar et al., 2002, Walker, 2012, Liu et al., 2011). The data used is a relatively large dataset of five-year old children, with adequate information on behavioural, parental and wider determinants characteristics of each child. Modern statistical techniques, such as, structural equation model for the conceptual framework and machine learning for the predictive models were employed. These modern statistical techniques have been shown, from literature, to achieve better outcomes than traditional logistic regression (Krittanawong et al., 2017); and they also yielded better results in this thesis.

Limitation of the study

The main limitation in this study is with the use of secondary data (oral health surveys) to explain a concept. The oral health survey data was also not collected primarily for SDH research study; therefore, it lacked important information, such as, information on exposure to water

fluoridation (which of the children was exposed to water fluoridation) and frequency of sugar intake that would have improved the conclusions from the study. The predictive models developed in chapters 5 and 6 would also have benefitted from additional wider determinants that were not collected routinely, such as birth order, parents' caries history, exposure to smoking, to mention a few. Similarly, the SDH risk factors assessed were self-reported, which can lead to possibility of a recall bias in the study.

The other limitation of this study is seen in the obligatory changes in oral survey methodology when it comes to consent. Parental consent for the survey changed from being opt-out to opt-in, which have resulted in a decreased response rate for the clinical examination phases consent (Spence et al., 2015). Epidemiologists have also argued that opt-in surveys are more likely to introduce response bias, because of their propensity to lower participation rate and a less representative sample (Spence et al., 2015). Caries epidemiologists also believed that parents of children with higher level of caries experience are less likely to consent, thus compromising the data quality (Spence et al., 2015, Vernazza et al., 2016b).

7.4 Section 3: Implications of this PhD

Predicting the risk of developing diseases is no longer new to the field of medicine and the process has provided significant opportunities especially within preventive medicine, where individuals at risk of developing diseases are identified much earlier, at a point when treatment is at greatest benefit (Custodio-Lumsden, 2013, Spencer, 2004, Avery et al., 2010). These risk models developed therefore provide epidemiologists with the opportunity of providing interventions that is proportional to individual or area level needs (predicted risk). The Marmot

Review: Fair Society, Healthy Lives calls this approach “Proportionate Universalism”, where universal health policies are developed at a scale and intensity that is proportionate to the level of risk identified or predicted (Marmot, 2010). This strategy came into prominence in public health in 2010, and has been heralded as the way forward to reducing health inequalities between the most and least affluent areas (Guillaume et al., 2017).

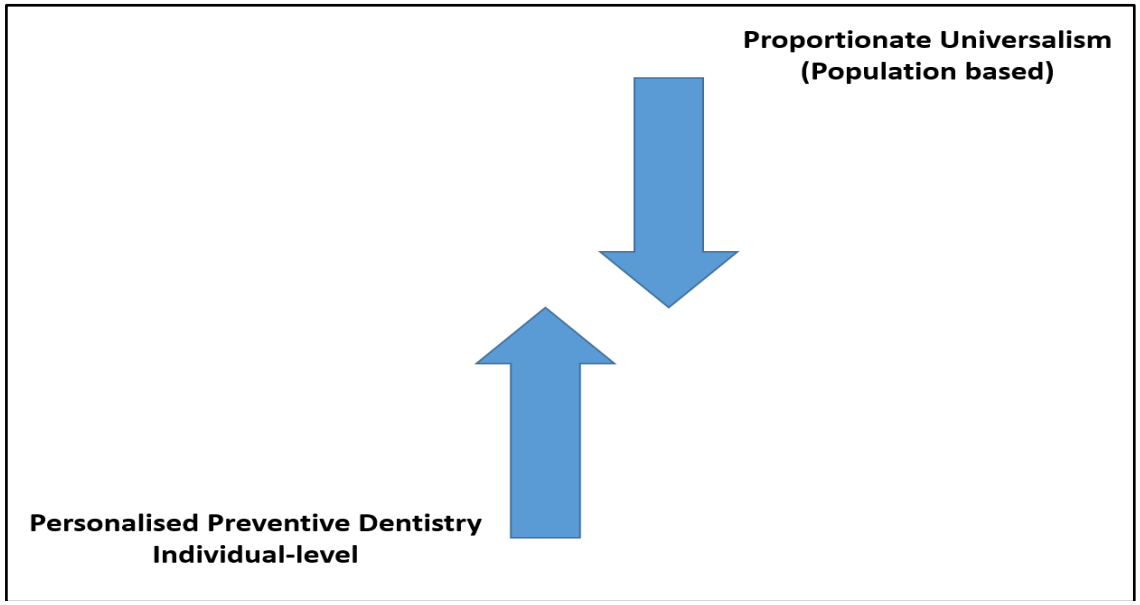
On the other hand, the individual level caries risk model developed in this thesis offers an opportunity to adopt a personalised preventive treatment approach to patients attending dental clinics, by specifically targeting children with similar risks identified in the risk model. The US National Library of Medicine National Institute of Health defined personalised medicine as “an emerging practice of medicine that uses an individual’s genetic profile to guide decisions made in regard to the prevention, diagnosis and treatment of disease”. However, this definition can be expanded to include using individual’s risk profiles to guide decisions made in regard to the prevention, diagnosis and treatment of disease (Azzopardi-Muscat, 2015).

Personalised preventive medicine helps the general dental practitioner to develop patient-tailored and individualised preventive treatments for children relative to their risks. With the current advances and gains in biomedical research, it has been suggested that the traditional one-size-fits-all approach to disease prevention would soon be abolished and replaced with a more individualised and tailor-made preventive methods. Implementing personalised preventive dentistry will help the general dental practitioner to develop custom-tailored individualised preventive treatments for preschool children relative to their SDH risks, even before these children develop caries. This approach is complementary to the population-based

proportionate universalism discussed above, and both strategies are proposed in this thesis in order to reduce existing caries inequalities (Figure 31).

Social inequalities in caries also have significant consequences to the government, such as its impact on overall cost to the National Health Service, and employing both approaches will ensure that the limited health resources are best utilised in a way that will reduce both caries prevalence and inequalities simultaneously. In addition to this, the SDH model developed in this study could be substituted for the rigorous and more expensive routine oral health surveys that is carried out every two years in England and Wales, or used in-between the surveys to assess caries prevalence, which will ultimately result in some financial cost savings to the NHS that could be diverted towards caries prevention. Furthermore, it can guide dental services planning such that more dental practices to set up practice in areas predicted to be of greatest need.

Figure 31: A Comprehensive Strategy for reducing caries inequalities in preschool population



7.5 Section 4: Recommendations for future research

Directions for future research relating to caries risk modelling in children fall into three main areas: the first is epidemiologic studies that are able to measure and ascertain exposure (and possibly length of exposure) of children to the wider determinants factors that were shown to be missing in Table 8. Details of exposure to water fluoridation, especially, will be a good practice to link into subsequent child dental health surveys and caries models. The inclusion of water fluoridation (and other missing SDH exposures) will have two major impact on future research: the first is that the real impact of water fluoridation in ECC will be highlighted, especially in the advent of sophisticated statistical modelling tools that are able to run several millions of machine learning models and techniques. The outcome will help to put rest to the age-long debate of the benefits of water fluoridation. It should also be emphasised that further research is equally needed on treatment plan options and recall intervals that should be considered for high-risk children, thus effecting a form of personalised preventive dentistry that is based on the relative exposures of the children to wider determinants risks.

Another recommendation is to ascertain the actual causal link between religion and ECC, and some of the questions that need to be addressed (or pathways that need to be eliminated) will include whether the practice of religion (aggregated together) adds more stress to the family such that parents are unable to supervise OH behaviour, or does it influence overall access to wider determinants resources. Further research should also be explored on how SDH-based predictive risk models can be replicated across other health needs and diseases, and to evaluate if it is a cheaper option to routine oral and general health and wellbeing surveys.

Health commissioners can also explore the impact of using SDH-based predictive risk models in subsequent Health Needs Assessments. It will also be important to understand the pathway between 'long term' unemployment and lower caries prevalence.

7.6 Conclusion

Early Childhood Caries (ECC) is one of the major global public health challenges that continues to experience difficulties in closing the SDH-related inequalities gap (Do et al., 2010). ECC has been shown to negatively impact on the children, their parents and overall health economy, where latest figures showed caries-related admission costing the NHS approximately £7.8 million per annum (Public Health England, 2017).

A literature review undertaken, using systematic methods, was able to identify 24 SDH risk factors capable of influencing ECC in children under the age of six and the association between these was demonstrated temporally and hierarchically. These interrelationships was further explored to develop a caries risk prediction model in preschool children using two separate oral health surveys. The child level risk model emphasised that the place where a child spends first six years of their life is one of the most important predictors of ECC, and this "place" is mainly determined in the UK by three ancestor risk factors, which are, parental education, parental occupation and overall socio-economic status of the family, using the conceptual model developed. General Dental Practitioners who are keen on preventive dentistry can also use the individual-level model to risk assess preschool children attending their clinics, and administering appropriate preventive measures to children who are at high risk of developing dental caries before this is evident in the child's mouth.

The caries prevalence model developed also showed that it is possible to predict, with a good level of accuracy, area-level ECC prevalence from SDH risk factors operating at local authority level. This could be a relevant tool to public health, because it can help inform policy developers who are willing to implement Proportionate Universalism. The caries prevalence tool also proffers the opportunity to estimate year-on-year caries prevalence at local authority level, with some degree of accuracy. These predicted local authority estimates could therefore be the guide for preventive health investments, where resource allocation is determined by relative need at local authority level, i.e., local authorities with poorly predicted estimates receive relatively more investments than others.

Statement of originality

As far as the research student is aware, this is the first known caries modelling study to go through the entire process of evidence-based disease risk modelling, starting from systematic review in order to identify all possible caries factors, then using causal theories, directed acyclic graphs and path analysis to explain the relationship these risk factors have with one another, which ultimately adds depth to previous early childhood caries modelling studies. Secondly, this is the first in the field, to the best of researcher's knowledge, to only use social determinants variables, i.e., excluding all behavioural variables like oral hygiene, sugar consumption and preventive measures in the analysis of caries risk in children. The model is developed using modern statistical methods such as machine learning to develop predictive risk models that are capable of predicting the likelihood of a five-year old developing caries. The reason for this exclusion is that, prior to now, the accuracy of existing caries models has been highly dependent on an accurate self-reported history from main carers or parents. However, self-reported history is known to be misleading largely because of fear of parents being blamed or held responsible for poor oral health consequences (Heilmann et al., 2016). Therefore, the total exclusion of behavioural factors is a novel approach to disease risk modelling and its main advantage is that the model provides better risk attribution that is independent of past behavioural histories.

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APPENDIX

Appendix 1: Search Strategy in EBSCO for six databases

(Medline, CINAHL, Dentistry and Oral Sciences, PsycINFO,

Search ID	Search Terms	Number of hits
S64	S41 AND S51 AND S63	23,761
S63	S52 OR S53 OR S54 OR S55 OR S56 OR S57 OR S58 OR S59 OR S60 OR S61 OR S62	5,345,970
S62	(paediatric* or pediatric*)	1,119,227
S61	(underage* or ("under" N0 age*))	22,620
S60	("child" or children* or childhood* or childcare* or schoolchild*)	4,427,085
S59	("toddler" or "toddlers")	27,082
S58	("baby" or "babies")	130,400
S57	("kid" or "kids")	42,606
S56	infant*	1,629,311
S55	girl*	291,199
S54	boy*	383,167
S53	"minors"	15,522
S52	MH "Child" OR MH "infant" OR MH "pediatrics"	2,553,452
S51	S29 OR S42 OR S43 OR S44 OR S45 OR S46 OR S47 OR S48 OR S49 OR S50	18,673,286
S50	(MH "Social Capital") OR "SOCIAL CAPITAL"	26,060
S49	(MH "Religion+") OR "RELIGION"	215,581
S48	(MH "Religion")	52,104

S47	(MH "Educational Status+") OR "EDUCATION ATTAINMENT"	83,151
S46	(MH "Female")	9,822,058
S45	(MH "Male")	9,485,445
S44	(MH "Occupations") OR "occupation"	101,399
S43	(MH "Ethnic Groups") OR (MH "Population Groups") OR (MH "Continental Population Groups") OR (MH "Emigrants and Immigrants") OR (MH "Family Characteristics") OR (MH "Residence Characteristics") OR (MH "European Continental Ancestry Group") OR (MH "Asian Continental Ancestry Group") OR (MH "Oceanic Ancestry Group") OR (MH "African Continental Ancestry Group") OR (MH "Race Relations") OR "race"	616,182
S42	(MH "Residence Characteristics+") OR "residence"	245,517
S41	S30 OR S31 OR S32 OR S33 OR S34 OR S35 OR S36 OR S37 OR S38 OR S39 OR S40	160,895
S40	(dental N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*))	104,860
S39	((dental or tooth or teeth or enamel or dentin*) and (decay* or cavit* or deminerali* or reminerali* or "white spot*"))	87,027
S38	("DMF Index" or dmf* or ICDAS)	24,475
S37	("white spot*" or "Early Childhood Caries")	7,154
S36	(MH "Tooth Demineralization")	4,184
S35	root N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*)	7,545
S34	dentin N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*)	5,440
S33	enamel N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*)	8,517
S32	tooth N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*)	43,037
S31	teeth N4 (cavit* or caries or carious or decay* or lesion* or deminerali* or reminerali*)	43,037
S30	(MH "Dental Caries")	69,289

S29	S1 OR S2 OR S3 OR S4 OR S5 OR S6 OR S7 OR S8 OR S9 OR S10 OR S11 OR S12 OR S13 OR S14 OR S15 OR S16 OR S17 OR S18 OR S19 OR S20 OR S21 OR S22 OR S23 OR S24 OR S25 OR S26 OR S27 OR S28	8,374,750
S28	(MH "Healthcare Disparities") OR (MH "Socioeconomic Factors+") OR "inequalities"	771,744
S27	(MH "Psychosocial Deprivation") OR (MH "Paternal Deprivation") OR (MH "Maternal Deprivation") OR (MH "Cultural Deprivation") OR "deprivation"	134,015
S26	(MH "Socioeconomic Factors+") OR (MH "Social Class+") OR (MH "Social Environment+") OR (MH "Population Characteristics+") OR (MH "Health Status Disparities") OR "socioeconomic"	3,244,053
S25	(TI workless OR AB workless OR SU workless) OR (TI worklessness OR AB worklessness OR SU worklessness)	216
S24	TI welfare OR AB welfare OR SU welfare OR TI benefits OR AB benefits OR SU benefits	1,357,732
S23	(TI vulnerable N2 population* OR AB vulnerable N2 population* OR SU vulnerable N2 population*) OR (TI vulnerable N2 population* OR AB vulnerable N2 population* OR SU vulnerable N2 population*) OR (TI vulnerable N2 communit* OR AB vulnerable N2 communit* OR SU vulnerable N2 communit*) OR (TI vulnerable N2 people OR AB vulnerable N2 people OR SU vulnerable N2 people) OR (TI vulnerable N2 person* OR AB vulnerable N2 person* OR SU vulnerable N2 person*)	38,701
S22	TI urban OR AB urban OR SU urban OR TI rural OR AB rural OR SU rural	631,728
S21	(TI underinsur* N2 health OR AB underinsur* N2 health OR SU underinsur* N2 health) OR (TI underprivile* OR AB underprivile* OR SU underprivile*) OR (TI unemployed OR AB unemployed OR SU unemployed) OR (TI unemployment OR AB unemployment OR SU unemployment) OR (TI uninsur* N2 health OR AB uninsur* N2 health OR SU uninsur* N2 health) OR (TI occupation OR AB occupation OR SU occupation) OR (TI employe* OR AB employe* OR SU employe*) OR (TI work OR AB work OR SU work) OR (TI job* OR ...	2,884,941
S20	(TI soci* N0 status OR AB soci* N0 status OR SU soci* N0 status) OR (TI soci* N0 variable* OR AB soci* N0 variable* OR SU soci* N0 variable*) OR (TI standard N2 living OR AB standard N2 living OR SU standard N2 living) OR (TI state N0 benefits OR AB state N0 benefits OR SU state N0 benefits) OR (TI uncompensated N0 care OR AB uncompensated N0 care OR SU uncompensated N0 care)	181,781
S19	(TI soci* N0 circumstance* OR AB soci* N0 circumstance* OR SU soci* N0 circumstance*) OR (TI soci* N0 factor* OR AB V OR SU soci* N0 factor*) OR (TI soci* N0 gradient* OR AB soci*N0 gradient* OR SU soci* N0 gradient*) OR (TI soci* N0 health N0 difference* OR AB soci* N0 health N0 difference* OR SU soci* N0 health N0 difference*) OR soci* N0 position	741,596

S18	(TI soci* N0 economic N0 position OR AB soci* N0 economic N0 position OR SU soci* N0 economic N0 position) OR (TI soci* N0 economic N0 status OR AB soci* N0 economic N0 status OR SU soci* N0 economic N0 status) OR (TI soci* N0 economic N0 variable* OR AB soci* N0 economic N0 variable* OR SU soci* N0 economic N0 variable*) OR (TI soci*economic N2 attribut* OR AB soci*economic N2 attribut* OR SU soci*economic N2 attribut*) OR (TI soci*economic N2 attribut* OR AB soci*economic N2 attribut* ...	24,313
S17	(TI soci* N0 economic N0 attribut* OR AB soci* N0 economic N0 attribut* OR SU soci* N0 economic N0 attribut*) OR (TI soci* N0 economic N0 circumstanc* OR AB soci* N0 economic N0 circumstanc* OR SU soci* N0 economic N0 circumstanc*) OR (TI soci* N0 economic N0 circumstanc* OR AB soci* N0 economic N0 circumstanc* OR SU soci* N0 economic N0 circumstanc*) OR (TI soci* N0 economic N0 gradient* OR AB soci* N0 economic N0 gradient* OR SU soci* N0 economic N0 gradient*) OR (TI soci* N0 economic ...	13,538
S16	(TI soci* N0 position OR AB soci* N0 position OR SU soci* N0 position) OR (TI soci* N0 security OR AB soci* N0 security OR SU soci* N0 security) OR (TI soci* N2 variation* OR AB soci* N2 variation* OR SU soci* N2 variation*) OR (TI soci* N0 welfare OR AB soci* N0 welfare OR SU soci* N0 welfare) OR (TI soci* N2 exclu* OR AB soci* N2 exclu* OR SU soci* N2 exclu*)	100,954
S15	(TI soci* N0 inclusion OR AB soci* N0 inclusion OR SU soci* N0 inclusion) OR (TI soci* N0 inequalit* OR AB soci* N0 inequalit* OR SU soci* N0 inequalit*) OR (TI soci* N0 inequit* OR AB soci* N0 inequit* OR SU soci* N0 inequit*) OR (TI soci* N0 isolat* OR AB soci* N0 isolat* OR SU soci* N0 isolat*) OR (TI soci* N0 justice OR AB soci* N0 justice OR SU soci* N0 justice)	117,577
S14	(TI soci* N2 disparit* OR AB soci* N2 disparit* OR SU soci* N2 disparit*) OR (TI soci* N0 environment OR AB soci* N0 environment OR SU soci* N0 environment) OR (TI soci* N2 exclu* OR AB soci* N2 exclu* OR SU soci* N2 exclu*) OR (TI soci* N2 factor* OR AB soci* N2 factor* OR SU soci* N2 factor*) OR (TI soci* N2 gradient* OR AB soci* N2 gradient* OR SU soci* N2 gradient*)	533,468
S13	(TI soci* N0 capital OR AB soci* N0 capital OR SU soci* N0 capital) OR (TI soci* N0 class OR AB soci* N0 class OR SU soci* N0 class) OR (TI soci* N2 condition* OR AB soci* N2 condition* OR SU soci* N2 condition*) OR (TI soci* N2 depriv* OR AB soci* N2 depriv* OR SU soci* N2 depriv*) OR (TI soci* N2 difference* OR AB soci* N2 difference* OR SU soci* N2 difference*)	264,719
S12	(TI occupation* N0 status OR AB occupation* N0 status OR SU occupation* N0 status) OR (TI poverty OR AB poverty OR SU poverty) OR (TI psychosocial N2 depriv* OR AB psychosocial N2 depriv* OR SU psychosocial N2 depriv*) OR (TI rural N2 health OR AB rural N2 health OR SU rural N2 health) OR (TI “SES” OR AB “SES” OR SU “SES”) OR (TI “Socioeconomic” OR AB “Socioeconomic” OR SU “Socioeconomic”) OR (TI “Socio-economic” OR AB “Socio-economic” OR SU “Socio-economic”)	674,821

S11	(TI material* N2 depriv* OR AB material* N2 depriv* OR SU material* N2 depriv*) OR (TI medical* N2 indigen* OR AB medical* N2 indigen* OR SU medical* N2 indigen*) OR (TI uninsured OR AB uninsured OR SU uninsured) OR (TI medicare OR AB medicare OR SU medicare) OR (TI multipl* N2 depriv* OR AB multipl* N2 depriv* OR SU multipl* N2 depriv*)	158,949
S10	(TI jobless* OR AB jobless* OR SU jobless*) OR (TI job N2 insecurit* OR AB job N2 insecurit* OR SU job N2 insecurit*) OR (TI low N0 income OR AB low N0 income OR SU low N0 income) OR (TI marginali* OR AB marginali* OR SU marginali*)	135,182
S9	(TI income * OR AB income OR SU income) OR (TI indigent OR AB indigent OR SU indigent) OR (TI inequit* OR AB inequit* OR SU inequit*) OR (TI health N2 insurance OR AB health N2 insurance OR SU health N2 insurance) OR (TI insurance N2 status OR AB insurance N2 status OR SU insurance N2 status) OR (TI state N2 insurance OR AB state N2 insurance OR SU state N2 insurance) OR (TI private N2 insurance OR AB private N2 insurance OR SU private N2 insurance)	519,432
S8	(TI health N2 inequalit* OR AB health N2 inequalit* OR SU health N2 inequalit*) OR (TI health N2 inequit* OR AB health N2 inequit* OR SU health N2 inequit*) OR (TI "health service* access*" OR AB "health service* access*" OR SU "health service* access*") OR (TI health N2 variation* OR AB health N2 variation* OR SU health N2 variation*) OR (TI income OR AB income OR SU income) OR (TI "dental access*" OR AB "dental access*" OR SU "dental access*") OR (TI "access to dentist*" OR AB "acc ...	522,829
S7	(TI financial N2 difficult* OR AB financial N2 difficult* OR SU financial N2 difficult*) OR (TI financial N2 problem* OR AB financial N2 problem* OR SU financial N2 problem*) OR (TI health N2 difference* OR AB health N2 difference* OR SU health N2 difference*) OR (TI health N2 disparit* OR AB health N2 disparit* OR SU health N2 disparit*) OR (TI health N2 equit* OR AB health N2 equit* OR SU health N2 equit*)	90,682
S6	TI soci* N2 depriv* OR AB soci* N2 depriv* OR SU soci* N2 depriv* OR TI soci* N2 disadvantage* OR AB soci* N2 disadvantage* OR SU soci* N2 disadvantage* OR TI education* N0 achieve* OR AB education* N0 achieve* OR SU education* N0 achieve* OR TI education* N0 status OR AB education* N0 status OR SU education* N0 status OR TI equit* OR AB equit* OR SU equit*	207,668
S5	TI care N0 seeking N0 behavio* OR AB care N0 seeking N0 behavio* OR SU care N0 seeking N0 behavio* OR TI care N0 seeking N0 behavio* OR AB care N0 seeking N0 behavio* OR SU care N0 seeking N0 behavio*	6,088
S4	TI avail* N2 care OR AB avail* N2 care OR SU avail* N2 care	11,835
S3	TI access N2 care OR AB access N2 care OR SU access N2 care	54,302
S2	TI access N2 healthcare OR AB access N2 healthcare OR SU access N2 healthcare	20,280

S1	TI avail* N2 healthcare OR AB avail* N2 healthcare OR SU avail* N2 healthcare	3,301

Appendix 2: Search Strategy in OVID for EMBASE database

	Search Term	Numbers of Hits
64	41 and 51 and 63	13453
63	52 or 53 or 54 or 55 or 56 or 57 or 58 or 59 or 60 or 61 or 62	2893190
62	(paediatric$ or pediatric$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	556361
61	(underage$ or ("under" adj1 age$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	16325
60	("child" or children or childhood$ or childcare$ or schoolchild$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	2401777
59	("toddler" or "toddlers").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	12518
58	("baby" or "babies").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	95274
57	("kid" or "kids").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	9863
56	infant/	546008
55	"girl".mp. or exp girl/	96992
54	"boy$".mp. or exp boy/	192709
53	"minors".mp. or exp "minor (person)"/	4749
	Search Term	Numbers of Hits
52	("Child" or "infant" or "paediatrics" or "pediatrics").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	2392178
51	29 or 42 or 43 or 44 or 45 or 46 or 47 or 48 or 49 or 50	12463802
50	("Social Capital" or "SOCIAL CAPITAL").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	3756
49	("Religion" or "RELIGION").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	67147

48	"Religion".mp. or exp religion/	68972
47	("Educational Status" or "EDUCATION ATTAINMENT").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	67439
46	female/	8069886
45	male/	7970227
44	("Occupations" or "occupation").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	66734
43	("Ethnic Groups" or "Population Groups" or "Continental Population Groups" or "Emigrants and Immigrants" or "Family Characteristics" or "Residence Characteristics" or "European Continental Ancestry Group" or "Asian Continental Ancestry Group" or "Oceanic Ancestry Group" or "African Continental Ancestry Group" or "Race Relations" or "race").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	221025
42	("Residence Characteristics" or "residence").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	58558
	Search Term	Numbers of Hits
41	30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40	88256
40	(dental adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	50184
39	((dental or tooth or teeth or enamel or dentin$) and (decay$ or cavit$ or demineral$ or remineral$* or "white spot")).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	33178
38	("DMF Index" or dmf$ or ICDAS).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	17016
37	("white spot" or "Early Childhood Caries").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	3571

36	(Tooth adj1 Demineral$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	283
35	(root adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	4568
34	(dentin adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	4217
33	(enamel adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	5498
	Search Term	Numbers of Hits
32	(tooth adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	5755
31	(teeth adj5 (cavit$ or caries or carious or decay$ or lesion$ or demineral$ or remineral$)).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	9464
30	"Dental Caries".mp. or exp dental caries/	46492
29	1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28	2767245
28	("Healthcare Disparities" or "Socioeconomic Factors" or "inequalities").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	29555
27	("Psychosocial Deprivation" or "Paternal Deprivation" or "Maternal Deprivation" or "Cultural Deprivation" or "deprivation").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	99157
26	("Socioeconomic Factors" or "Social Class" or "Social Environment" or "Population Characteristics" or "Health Status Disparities" or "socioeconomic").mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	158975

25	(workless or worklessness).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	44
24	(welfare or benefits).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	448128
23	(((((((((vulnerable adj3 population) or vulnerable) adj3 population) or vulnerable) adj3 communit*) or vulnerable) adj3 people) or vulnerable) adj3 person*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	696
22	(urban or rural).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	313947
21	((((Underinsure* adj3 health) or underprivile* or unemployed or unemployment or uninsur*) adj3 health) or occupation or employe* or work or job*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	1641217
20	(((((((((Soci* adj1 status) or soci*) adj1 variable*) or standard) adj3 living) or state) adj1 benefits) or uncompensated) adj1 care).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	371
19	(((((((Soci* adj1 circumstance*) or soci*) adj1 factor*) or soci*) adj1 gradient*) or soci*) adj1 health adj1 difference*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	29
18	(((((((((Soci* adj1 economic adj1 position) or soci*) adj1 economic adj1 status) or soci*) adj1 economic adj1 variable*) or soci$economic) adj3 attribut*) or socio*) adj3 attribut*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	656
17	(((((((((Soci* adj1 economic adj1 attribut*) or soci$) adj1 economic adj1 circumstanc*) or soci*) adj1 economic adj1 circumstanc*) or soci*) adj1 economic adj1 gradient*) or soci*) adj1 economic adj1 health* adj1 difference*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	22
16	((((((((((Soci* adj1 position) or AB soci*) adj1 position) or soci*) adj1 security) or soci*) adj3 variation*) or soci*) adj1 welfare) or soci*) adj3 exclu*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	3783

	Search Term	Numbers of Hits
15	(((((((((Soci* adj1 inclusion) or soci*) adj1 inequalit*) or soci*) adj1 inequit*) or soci*) adj1 isolat*) or soci*) adj1 justice).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	10699
14	(((((((((Soci* adj3 disparit*) or soci*) adj1 environment) or soci*) adj3 exclu*) or soci*) adj3 factor*) or soci*) adj3 gradient*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	2116
13	(((((((((Soci* adj1 capital) or soci*) adj1 class) or soci*) adj3 condition*) or soci*) adj3 depriv*) or soci*) adj3 difference*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	11810
12	((((((Occupation* adj1 status) or poverty or psychosocial) adj3 depriv*) or rural) adj3 health) or Socioeconomic or Socio-economic).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	150713
11	(((((material* adj3 depriv*) or medical*) adj3 indigen*) or uninsured or medicare or multipl*) adj3 depriv*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	1263
10	(((((jobless$ or job) adj3 insecurit$) or low) adj1 income) or marginali$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	46075
9	(((((((income or indigent or inequit* or health) adj3 insurance) or insurance) adj3 status) or state) adj3 insurance) or private) adj3 insurance).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	20420
8	((((((health adj3 inequalit*) or health) adj3 inequit*) or health service* access* or health) adj3 variation*) or income or dental access* or access to dentist*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	158491
7	(((((((((financial adj3 difficult*) or financial) adj3 problem*) or health) adj3 difference*) or health) adj3 disparit*) or health) adj3 equit*).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	5752

6	(((((((soci$ adj3 depriv$) or soci$) adj3 disadvantage$) or education$) adj1 achieve$) or education$) adj1 status) or equit$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	95470
5	(((care adj1 seeking adj1 behavio$) or care) adj1 seeking adj1 behavio$).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	1435
4	(availab$ adj3 care).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	19588
3	(access adj3 care).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	81599
2	(access adj3 healthcare).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	5608
1	(availab$ adj3 healthcare).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word, candidate term word]	1276

Appendix 3: Data extraction form for Cohort studies

Publication-level data:	**Descriptions as stated in the report/paper**
Title	
Authors	
Publication Year	
Abstract or Full paper	
Study ID (Endnote number)	
Journal /Volume/Page	
Country of Study	
Data Extractor	Olufemi Olajide
Date of extraction	

Study Aims	**Descriptions as stated in the report/paper**
Study design	
Study Setting	
How were participants recruited?	
Overview of the study	
Research question/Hypothesis/study aims	
Dates of study	
Total study duration	

Population	**Descriptions as stated in the report/paper**
Participants age	
Source of subjects	
Inclusion criteria	
Exclusion criteria	
Recruitment procedures used	
Total number of subjects recruited	
Total number of subjects in this study	
Sample size calculation, y/n	

Outcome	Descriptions as stated in the report/paper
Exposures of Interest	
Outcome of Interest	
Who reported outcome? Self or study assessor	
Data collected	
Is this outcome/tool validated?	

Analysis	Descriptions as stated in the report/paper
A priori power calculation	
Statistical analysis used	
Confounding factors and how were they handled	
Subgroup analysis	
Missing data + how addressed	

Results

Social determinants indicator	Association between socioeconomic indicators and dental caries
Outcome measures reported:	
PROGRESS-PLUS explored	
Conclusion	

Additional useful information	Descriptions as stated in the report/paper
Strength	
Limitations	
Interpretation	
Generalisibility of study	
Study funding source	
Possible conflicts of interest	
Is it an independent evaluation	
Reviewers comments and conclusion	

Xxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxx

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Risk of Bias Assessment on next page

Appendix 4: Data extraction form for Case-Control studies

Publication-level data:	**Descriptions as stated in the report/paper**
Title	
Authors	
Publication Year	
Abstract or Full paper	
Study ID (Endnote number)	
Journal /Volume/Page	
Country of Study	
Data Extractor	Olufemi Olajide
Date of extraction	

Study Aims	**Descriptions as stated in the report/paper**
Study design	**Case-control**
Study Setting	
How were participants recruited?	
Research question/Hypothesis/study aims	
Dates of study	
Total study duration	

Population	**Descriptions as stated in the report/paper**
Participants age	
Source of subjects	
If case-control: What is the source of recruitment for cases	
If case-control: What is the source of recruitment for control	
Exclusion criteria	
Total number of subjects recruited	
Total number of subjects in this study	
Sample size calculation, y/n	

Outcome	Descriptions as stated in the report/paper
Exposures of Interest	
Outcome of Interest	
Who reported outcome? Self or study assessor	
Is this outcome/tool validated?	

Analysis	Descriptions as stated in the report/paper
A priori power calculation	
Statistical analysis used	
Confounding factors and how were they handled	
Subgroup analysis	
Missing data + how addressed	

Results

Social determinants indicator	Association between socioeconomic indicators and dental caries
Outcome measures reported:	
PROGRESS-PLUS explored	
Conclusion	

Additional useful information	Descriptions as stated in the report/paper
Strength	
Limitations	
Interpretation	
Generalisibility of study	
Study funding source	
Possible conflicts of interest	
Is it an independent evaluation	
Reviewers comments and conclusion	

Risk of Bias Assessment on next page

Appendix 5: Newcastle - Ottawa Quality Assessment Scale for Cohort Studies

Note: A study can be awarded a maximum of one star for each numbered item within the Selection and Outcome categories. A maximum of two stars can be given for Comparability

Selection

1) Representativeness of the exposed cohort

a) Truly representative of the average _ (describe) in the community *

b) Somewhat representative of the average _____ _____ in the community *

c) Selected group of users e.g. nurses, volunteers

d) No description of the derivation of the cohort

2) Selection of the non-exposed cohort

a) Drawn from the same community as the exposed cohort *

b) Drawn from a different source

c) No description of the derivation of the non-exposed cohort

3) Ascertainment of exposure

a) Secure record (e.g. surgical records) *

b) Structured interview *

c) Written self-report

d) No description

4) Demonstration that outcome of interest was not present at start of study

a) Yes *

b) No

Comparability

1) Comparability of cohorts on the basis of the design or analysis

a) Study controls for _____ (select the most important factor) *

b) Study controls for any additional factor *

(All children/mothers resided in the four selected districts.)

Outcome

1) Assessment of outcome

a) Independent blind assessment *

b) Record linkage *

c) Self-report

d) No description

2) Was follow-up long enough for outcomes to occur?

a) Yes *

b) No

3) Adequacy of follow up of cohorts

a) Complete follow up - all subjects accounted for *

b) Subjects lost to follow up unlikely to introduce bias - small number lost ( ) *

c) Follow up rate < ____% (select an adequate %) and no description of those lost

d) No statement

Total Score out of 9

Appendix 6: Newcastle - Ottawa Quality Assessment Scale for Case Control Studies

Note: A study can be awarded a maximum of one star for each numbered item within the Selection and Exposure categories. A maximum of two stars can be given for Comparability.

Selection

1) Is the case definition adequate?

a) Yes, with independent validation *

b) Yes, e.g. record linkage or based on self-reports

c) No description

2) Representativeness of the cases

a) Consecutive or obviously representative series of cases *

b) Potential for selection biases or not stated

3) Selection of Controls

a) Community controls *

b) Hospital controls

c) No description

4) Definition of Controls

a) No history of disease (endpoint) *

b) No description of source

Comparability

1) Comparability of cases and controls on the basis of the design or analysis

a) Study controls for _____ (Select the most important factor.) *

b) Study controls for any additional factor * (This criteria could be modified to indicate specific control for a second important factor.)

Exposure

1) Ascertainment of exposure

a) Secure record (eg surgical records) *

b) Structured interview where blind to case/control status *

c) Interview not blinded to case/control status

d) Written self-report or medical record only

e) No description

2) Same method of ascertainment for cases and controls

a) Yes *

b) No

3) Non-Response rate

a) Same rate for both groups *

b) Non respondents described

c) Rate different and no designation

Total Score out of 9 =

Appendix 7: Details of papers excluded

Reasons for exclusion	Number of papers
SDH was not assessed	46
Cross sectional Study	43
Participants' age not between 0-6 years	39
Caries not assessed	7
Publication is not written in English	6
Ongoing Studies - results not available	3
Same study not adding anything new to SDH	3
Two separate cohorts of children assessed	3
Outcome assessed is severe ECC	2
PhD thesis	2
Review	2
Grand total	156

Appendix 8: Characteristics of Eligible Studies

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Gussy, R. Ashbolt, L. Carpenter, M. Virgo-Milton, H. Calache, S. Dashper, P. Leong, A. de Silva, A. de Livera, J. Simpson and E. Waters	Natural history of dental caries in very young Australian children	2016	Cohort	3 years	467	Australia	1	7
Ju, L. M. Jamieson and G. C. Mejia	Estimating the effects of maternal education on child dental caries using marginal structural models: The Longitudinal Study of Indigenous Australian Children	2016	Cohort	5 years	1720	Australia	1	8
Sanders and G. D. Slade	Apgar score and dental caries risk in the primary dentition of five year olds	2010	Cohort	5 years	1398	Australia	1	8
Leroy, K. Bogaerts, L. Martens and D. Declerck	Risk factors for caries incidence in a cohort of Flemish preschool children	2012	Cohort	5 years	1057	Belgium	1	8
Dos Santos Pinto, de Ávila Quevedo L, Britto Correa M, Sousa Azevedo, Leão Goettems M, Tavares Pinheiro , Demarco.	Maternal Depression Increases Childhood Dental Caries: A Cohort Study in Brazil.	2017	Cohort	2 to 3 years	538	Brazil	2	9
	Are Maternal Factors Predictors for Early Childhood Caries? Results from a Cohort in Southern Brazil	2017	Cohort	2 to 3.5 years	538	Brazil	2	7
	Maternal care influence on children's caries prevalence in southern Brazil	2016	Cohort	2 to 3.5 years	538	Brazil	2	7
Ribeiro, M. C. B. D. Silva, A. M. M. Nunes, E. B. A. F. Thomaz, C. D. S. Carmo, M. R. C. Ribeiro and A. A. M. D. Silva	Overweight, obese, underweight, and frequency of sugar consumption as risk indicators for early childhood caries in Brazilian preschool children	2017	Cohort	6 years	388	Brazil	2	7

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
	Factors underlying the polarization of early childhood caries within a high-risk population	2014	Cohort	6 years	244	Brazil	2	8
Goettems, G. G. Nascimento, M. A. Peres, Ina S. Santos, A. Matijasevich, A. J. D. Barros, K. G. Peres and Flávio F. Demarco	Influence of maternal characteristics and caregiving behaviours on children's caries experience: An intergenerational approach	2018	Cohort	5 years	1303	Brazil	2	8
	Impact of Prolonged Breastfeeding on Dental Caries: A Population-Based Birth Cohort Study	2017	Cohort	5 years	1129	Brazil	2	9
Cabral, E. L. A. Mota, M. C. T. Cangussu, M. I. P. Vianna and F. R. Floriano	Risk factors for caries-free time: longitudinal study in early childhood	2017	Cohort	<30 months	495	Brazil	2	6
Piva, J. T. Pereira, P. B. Luz, L. N. Hashizume, F. N. Hugo and F. B. d. Araujo	A Longitudinal Study of Early Childhood Caries and Associated Factors in Brazilian Children	2017	Cohort	3 to 4 years	163	Brazil	2	8
Rocha, C. A. S. Garbin, A. J. I. Garbin, O. Saliba and S. A. S. Moimaz	Longitudinal study into the determining factors of dental caries in children aged 4: socio-behavioral aspects and oral health of pregnant women	2017	Cohort	4 years	73	Brazil	2	7
Schroth, C. Lavelle, R. Tate, S. Bruce, R. J. Billings and M. E. K. Moffatt	Prenatal Vitamin D and Dental Caries in Infants	2014	Cohort	<2 years	207	Canada	1	9
Wong, H. X. Lu and E. C. M. Lo	Caries increment over 2 years in preschool children: a life course approach	2012	Cohort	5 to 6 years	358	China	2	7
Zhou, J. Y. Yang, E. C. M. Lo and H. C. Lin	The Contribution of Life Course Determinants to Early Childhood Caries: A 2-Year Cohort Study	2012	Cohort	32 months	225	China	2	7

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Guedes, C. Piovesan, I. Floriano, B. Emmanuelli, M. M. Braga, K. R. Ekstrand, T. M. Ardenghi and F. M. Mendes	Risk of initial and moderate caries lesions in primary teeth to progress to dentine cavitation: a 2-year cohort study	2016	Cohort	1 to 6 years	469	Denmark	1	7
Mattila ML, Rautava P, Sillanpää M, Paunio P.	Caries in Five-year-old Children and Associations with Family-related Factors	2000	Cohort	5 years	828	Finland	1	7
Meurman P, Pienihäkkinen K.	Factors Associated with Caries Increment: A Longitudinal Study from 18 Months to 5 Years of Age	2010	Cohort	5 years	337	Finland	1	7
Wagner and R. Heinrich-Weltzien	Evaluation of a regional German interdisciplinary oral health programme for children from birth to 5 years of age	2017	Cohort	5 years	289	Germany	1	8
Agarwal, R. Nagarajappa, S. B. Keshavappa and R. T. Lingesha	Association of maternal risk factors with early childhood caries in schoolchildren of Moradabad, India	2011	Cohort	3 to 5 years	150	India	3	6
Nishide, M. Mizutani, S. Tanimura, N. Kudo, T. Nishii and H. Hatashita	Homecare protective and risk factors for early childhood caries in Japan 11 Medical and Health Sciences	2018	Cohort	3 Years	566	Japan	1	6
Tanaka S, Maki Shinzawa, Hironobu Tokumasu, Kahori Seto, Sachiko Tanaka, Koji Kawakami	Secondhand smoke and incidence of dental caries in deciduous teeth among children in Japan: population based retrospective cohort study	2015	Cohort	3 YEARS	76,920	Japan	1	9
Tanaka, Yoshihiro;Sasaki, Satoshi;Hirota, Yoshio	Socioeconomic status and risk of dental caries in Japanese preschool children: the Osaka Maternal and Child Health Study	2013	Cohort	41-50 months	315	Japan	1	7
Yokomichi, T. Tanaka, K. Suzuki, T. Akiyama and Z. Yamagata	Macrosomic Neonates Carry Increased Risk of Dental Caries in Early Childhood:	2015	Cohort	3 years	117,175	Japan	1	8

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
	Findings from a Cohort Study, the Okinawa Child Health Study, Japan							
van der Tas, L. Kragt, J. J. Veerkamp, V. W. Jaddoe, H. A. Moll, E. M. Ongkosuwito, M. E. Elfrink and E. B. Wolvius	Ethnic Disparities in Dental Caries among Six-Year-Old Children in the Netherlands	2016	Cohort	6 years	4306	Netherlands	1	9
Schluter PJ, Durward C, Cartwright S, Paterson J.	Maternal self-report of oral health in 4-year-old Pacific children from South Auckland, New Zealand: findings from the Pacific Islands Families Study	2007	Cohort	4 years	1048	New Zealand	1	8
Grytten, I. Rossow, D. Hoist and L. Steele	Longitudinal study of dental health behaviors and other caries predictors in early childhood	1988	Cohort	3 years	231	Norway	1	7
Wigen TI1, Wang NJ.	Health behaviors and family characteristics in early childhood influence caries development. A longitudinal study based on data from MoBa	2014	Cohort	5 years	1348	Norway	1	9
	Maternal health and lifestyle, and caries experience in preschool children. A longitudinal study from pregnancy to age 5 yr	2011	Cohort	5 years	1348	Norway	1	9
	Caries and background factors in Norwegian and immigrant 5-year-old children	2010	Cohort	5 years	1348	Norway	1	9
	Family characteristics and caries experience in preschool children. A longitudinal study from pregnancy to 5 years of age	2010	Cohort	5 years	1348	Norway	1	9

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Gao, C. Y. Hsu, Y. C. Xu, T. Loh, D. Koh and H. B. Hwarng	Behavioral pathways explaining oral health disparity in children	2010	Cohort	3 to 6 years	1576	Singapore	1	7
Un Lam, L. W. Khin, A. C. Kalhan, R. Yee, Y. S. Lee, M. F. F. Chong, K. Kwek, S. M. Saw, K. Godfrey, Y. S. Chong and C. Y. Hsu	Identification of Caries Risk Determinants in Toddlers: Results of the GUSTO Birth Cohort Study	2017	Cohort	2 years	543	Singapore	1	7
Julihn, F. C. Soares, A. Hjern and G. Dahllöf	Socioeconomic Determinants, Maternal Health, and Caries in Young Children	2018	Cohort	3 years	73,658	Sweden	1	8
Boustedt, J. Roswall, S. Twetman and J. Dahlgren	Influence of mode of delivery, family and nursing determinants on early childhood caries development: a prospective cohort study	2018	Cohort	3-5 years	551	Sweden	1	7
Grindefjord, G. Dahllöf, B. Nilsson and T. Modéer	Stepwise prediction of dental caries in children up to 3.5 years of age	1996	Cohort	3.5 Years	692	Sweden	1	8
Östberg, M. S. Skeie, A. B. Skaare and I. Espelid	Caries increment in young children in Skaraborg, Sweden: associations with parental sociodemography, health habits, and attitudes	2017	Cohort	6 years	243	Sweden	1	8
Nirunsittirat, W. Pitiphat, C. M. McKinney, T. A. DeRouen, N. Chansamak, O. Angwaravong, P. Patcharanuchat and T. Pimpak	Adverse birth outcomes and childhood caries: a cohort study	2016	Cohort	3 to 4 years	860	Thailand	2	8
Peltzer, Aroonsri;Satchaiyan, Gamon;Rajchagool, Sunsanee;Pimpak, Taksin	Sociobehavioral factors associated with caries increment: a longitudinal study from 24 to 36 months old children in Thailand	2014	Cohort	3 Years	365	Thailand	2	9
Thitasomakul, S. Piwat, A. Thearmontree, O. Chankanka, W. Pithpornchaiyakul and S. Madyusoh	Risks for early childhood caries analyzed by negative binomial models	2009	Cohort	9-18 months	495	Thailand	2	7

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Kay, K. Northstone, A. Ness, K. Duncan and S. J. Crean	Is there a relationship between Birthweight and subsequent growth on the development of Dental Caries at 5 years of age? A cohort study	2010	Cohort	5 years	985	United Kingdom	1	9
Skafida, S. Chambers	Positive association between sugar consumption and dental decay prevalence independent of oral hygiene in pre-school children: a longitudinal prospective study	2018	Cohort	5 years	3770	United Kingdom	1	8
Brickhouse, R. Gary;Slade, Gary D.	Effects of enrollment in medicaid versus the state children's health insurance program on kindergarten children's untreated dental caries	2008	Cohort	Kindergarten	23936	United States	1	9
Fontana, R.;Eckert, G.;Swigonski, N.;Chin, J.;Ferreira Zandona, A.;Ando, M.;Stookey, G. K.;Downs, S.;Zero, D. T.	Identification of Caries Risk Factors in Toddlers	2011	Cohort	1.5 to 3 years	329	United States	1	8
Hong, S. M. Levy, J. J. Warren and B. Broffitt	Infant Breast-feeding and Childhood Caries: A Nine-year Study	2014	Cohort	5 years	509	United States	1	8
Ismail, W.;Lim, S.;Willem, J. M.	Predictors of dental caries progression in primary teeth	2009	Cohort	0 to 5 years	788	United States	1	6
	Determinants of Early Childhood Caries in Low-income African American Young Children	2008	Cohort	0 to 5 years	788	United States	1	7
Warren, Derek;Dawson, Deborah V.;Marshall, Teresa A.;Phipps, Kathy R.;Starr, Delores;Drake, David R.	Factors associated with dental caries in a group of American Indian children at age 36 months	2016	Cohort	3 years	232	United States	1	9

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Flaherman, J. Epstein, L. Amendola, R. Inge, J. D. Featherstone and M. Okumura	Preventive Dental Care at 6-Month Intervals Is Associated With Reduced Caries Risk	2017	Cohort	< 6 years	132,462	United States	1	6
Plonka, M. L. Pukallus, A. G. Barnett, T. F. Holcombe, L. J. Walsh and W. K. Seow	A longitudinal case-control study of caries development from birth to 36 months	2013	Case-Control	3 YEARS	552	Australia	1	7
Seow, H.;Battistutta, D.;Morawska, A.;Holcombe, T.	Case-control study of early childhood caries in Australia	2009	Case-Control	0 to 4 years	617	Australia	1	7
Slade, A. E.;Bill, C. J.;Do, L. G.	Risk factors for dental caries in the five-year-old South Australian population	2006	Case-Control	5 years	1398	Australia	1	6
Marina de Deus Moura de Lima	Risk factors associated with early childhood caries-a case control study	2016	Case-Control	< 6 years	1016	Brazil	2	7
Tavares, S. T. Moysés, S. J. Moysés, J. C. Bisinelli, B. H. S. França and F. A. Ribeiro	Dental caries protection factors in 5-year-old Brazilian children	2008	Case-Control	5 years	966	Brazil	2	7
Tiberia, A. R. Milnes, R. J. Feigal, K. R. Morley, D. S. Richardson, W. G. Croft and W. S. Cheung	Risk factors for early childhood caries in Canadian preschool children seeking care	2007	Case-Control	1 to 5 years	132	Canada	1	2
Werneck, Herenia P.;Kulkarni, Gajanan V.;Locker, David	Early Childhood Caries and Access to Dental Care among Children of Portuguese-Speaking Immigrants in the City of Toronto	2008	Case-Control	≤ 4 years	104	Canada	1	7
Lulic-Dukic, H. Juric, W. Dukic and D. Glavina	Factors predisposing to early childhood caries (ECC) in children of pre-school age in the city of Zagreb, Croatia	2001	Case-Control	2 to 5 years	145	Croatia	1	7
Dabawala, B. S. Suprabha, R. Shenoy, A. Rao and N. Shah	Parenting style and oral health practices in early childhood caries: a case-control study	2016	Case-Control	3 to 5 years	422	India	3	8

Author	Title	Year	Type of study	Age	Sample size	Country	*HDI	NOS
Mahesh, M.;Rodrigues, S.	Risk factors for early childhood caries: a case-control study	2013	Case-Control	0.5 to 6 years	380	India	3	2
Menon, R. Nagarajappa, G. Ramesh and M. Tak	Parental stress as a predictor of early childhood caries among preschool children in India	2012	Case-Control	4 to 5 years	800	India	3	8
Sridevi, S. Pranoti, S. Anand, W. Umesh and G. Sachin	Factors associated with early childhood caries among 3 to 6 year old children in India: A case control study	2018	Case-Control	3-6 years	690	India	3	7
Ohsuka, N. Chino, H. Nakagaki, I. Kataoka, Y. Oshida, I. Ohsawa and Y. Sato	Analysis of risk factors for dental caries in infants: a comparison between urban and rural areas	2009	Case-Control	1.6 to 3 years	232	Japan	1	8
Huntington NL1, Kim IJ, Hughes CV.	Caries-risk factors for Hispanic children affected by early childhood caries.	2002	Case-Control	≤ 5 years	120	United States	1	6
Nunn, T. Dietrich, H. K. Singh, M. M. Henshaw and N. R. Kressin	Prevalence of Early Childhood Caries Among Very Young Urban Boston Children Compared with US Children	2009	Case-Control	1 to 3 years	4,431	United States	1	8
Smith, V. M. Badner, D. E. Morse and K. Freeman	Maternal risk indicators for childhood caries in an inner city population	2002	Case-Control	3 to 5 years	60	United States	1	6

***HDI 1 = Developed countries (HDI >0.8); *HDI 2 and 3 = Developing countries (HDI <0.8)**

Appendix 9: Characteristics and findings from studies exploring water fluoridation and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Sanders and Slade, 2010	Cohort	1398	Australia	1	8	Significant	Higher risk of caries was also associated with suboptimal fluoride concentration in drinking water relative to the reference groups; Prevalence Ratio: 1.36 (1.12, 1.66)
Hong et al 2014	Cohort	509	United States	1	8	Significant	Higher levels of fluoride in home tap water were significantly associated with lower caries experience in children in the first six months of life; (p=0.006)
Peltzer et al 2014	Cohort	597	Thailand	2	9	Significant	Odds Ratio of children living in areas with suboptimal water fluoridation is 1.99 (1.08–3.69), compared with children living in areas with optimal water fluoridation
Mahesh et al 2013	Case-control	380	India	3	2	Significant	Exposure to sub-optimal water fluoridation increases the risk of childhood caries; 4.58, 95 % CI 2.13–9.86; p=0.0001
Gussy, 2016	Cohort	467	Australia	1	7	Not Significant	Not Significant
Tiberia et al 2007	Case-control	132	Canada	1	3	Not Significant	Not Significant

Appendix 10: Characteristics and findings from the three studies exploring Geographic Place of residence and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Ju et al, 2016	Cohort	1720	Australia	1	8	Significant	Children residing in outer regional areas had the highest proportion of disease; Outer regional 1.30 (1.02–1.66)
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	hildren living in rural areas were at higher risk of developing caries than children living in urban areas; Odds Ratio = 2.4, CI= 1.2 – 4.5; p=0.014
Ohsuka et al., 2009	Case-control	232	Japan	1	8	Significant	Prevalence of caries in 3 year olds was 25.7% in urban children and 47.6% in rural children [p<0.05], therefore, significantly higher risk in the rural area than in the urban area.

Appendix 11: Characteristics and findings from the study exploring Community Level Deprivation and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Ismail, 2009	Cohort	788	United States	1	6	Significant	Living in relatively disadvantaged low-income neighbourhood is a risk factor of caries; (most disadvantaged); 1.43 (p=0.03)

Appendix 12: Characteristics and findings from the study exploring Access to Dentist and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Brickhouse et al 2008	Cohort	23936	United States	1	9	Significant	Living in relatively high dentist to population area is a protective factor of caries; Base: (< 5 dentists per 1,000 population); (5-10 dentists per 1,000 population) = - 0.16 (p≤0.001)
Wigen and Wang, 2011	Cohort	1348	Norway	1	9	Significant	Parents with poor access to dental care; 2.0 [1.2–3.3]
Rocha, 2017	Cohort	73	Brazil	2	7	Not significant	Not significant

Appendix 13: Characteristics and findings studies exploring race and ethnicity and the risk of ECC

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Schroth et al., 2014	Cohort	207	Canada	1	9	Significant	Mothers of infants with caries were significantly more likely to be Aboriginal
Brickhouse, 2008	Cohort	23,936	United States	1	9	Significant	Children of White origin more at risk White 1.00 Black 0.03 Hispanic 0.43 (p<0.001) American Indian 0.61 (p<0.001) Other 0.21 (p<0.001)
Wigen and Wang, 2014	Cohort	1,348	Norway	1	9	Significant	Having a Non-western parent poses a significant risk
Skafida et al, 2018	Cohort	3,770	UK	1	8	Significant	Non-white children have significantly higher risk of developing caries
Meurman and Pienihäkkinen 2010	Cohort	337	Finland	1	7	Significant	Non-Finnish children have significantly higher risk of developing caries
Gao et al 2010	Cohort	1,782	Singapore	1	7	Significant	Malays children are more susceptible to caries (β =0.087, $p < 0.05$)
Cabral et al, 2017	Cohort	495	Brazil	2	6	Significant	Being Black/Biracial was significantly related to caries presence
Lam et al, 2017	Cohort	543	Singapore	1	7	Significant	Chinese children are at higher risk. This is significant
Schluter et al 2007	Cohort	1,018	New Zealand	1	8	Significant	Children of Pacific mothers more likely to have caries than children of non-Pacific mothers
Van der Tas, 2016	cohort study	4306	NETHERLANDS	1	9	Significant	Ethnicity is a risk factor of developing caries: Dutch = Reference; Surinamese- Hindustani = 2.06 (1.46 – 2.89); Surinamese- Creole - 1.45 (1.02 – 2.07);

							Turkish = 4.48 (3.60 – 5.58); Dutch Antillean - 1.28 (0.90 – 1.83); Moroccan - 4.43 (3.46 – 5.67); Cape Verdean- 1.63 (1.17 – 2.26)
Nunn et al., 2009	Case-control	4,431	United States	1	8	Significant	Children from Hispanics and Asians race had greater risks of developing caries
Werneck, 2008	Case-control	104	Canada	1	7	Significant	Children with at least one non-European parents had greater risks of developing caries
Tiberia et al., 2007	Case-control	132	Canada	1	3	Significant	Being a Caucasian was significantly related to increased caries presence and severity
Slade, 2006	Case-control	1,398	Australia	1	6	Significant	Caries prevalence was significantly elevated among indigenous children
Smith et al., 2002	Case-control	60	United States	1	6	Not significant	Not significant

Appendix 14: Characteristics and findings from the studies exploring Country of Birth and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Julihn, et al, 2018	Cohort study	73,658	Sweden	1	8	Significant	Children of mothers with maternal country at birth (Underdeveloped) were 3.38 (3.13 to 3.63) times more likely to have caries
Grindefjord, 1996	Cohort	692	Sweden	1	8	Significant	Children of Immigrant background were 2.34 [1.33 – 4.33]; P=0.003 times more likely to have caries
Wigen and Wang, 2014	Cohort	1,348	Norway	1	9	Significant	Outcome: Children of parents of non-western background had 5.4 times higher probability of having caries at the age of 5 years
Wong et al., 2012	Cohort	358	Hong Kong	2	7	Significant	Percentage increase in caries is higher in children not born in Hong Kong, with a p-value =0.036, however, multivariate analysis not done
Nunn et al., 2009	Case-control	4,431	United States	1	8	Significant	US children of foreign-born parents almost three times the odds of having caries compared to US children of United States-born parents (OR = 2.7, P < 0.001)
Werneck, 2008	Case-control	104	Canada	1	7	Significant	Children of parents from non-European countries (Brazil and Angola) more likely to have caries: Mothers = [2.51 (1.11–5.71) 0.026], Fathers = [3.37 (1.49–7.58) 0.003]
Sanders and Slade, 2010	Cohort	1,398	Australia	1	8	Not Significant	Not significant
Fontana et al 2011	Cohort	329	United States	1	8	Not Significant	Not significant
Smith et al., 2002	Case-control	60	United States	1	6	Not Significant	Not significant

Wagner and Heinrich-Weltzien, 2017	Case-control	1,162	Germany	1	9	Not Significant	Not Significant

Appendix 15: Characteristics and findings from the studies exploring Language and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Meurman and Pienihäkkinen 2010	Cohort	337	Finland	1	7	Significant	Those whose language are non-Finnish were significant at univariate [OR=3.3; CI: 1.1–10.1; p=0.037]
Fontana et al 2011	Cohort	329	United States	1	8	Significant	Children with English not primary language at home were more at risk: OR > 2.0
Nunn et al., 2009	Case-control	4431	United States	1	8	Significant	Children whose primary language not English had greater risks (OR = 3.2, P < 0.001)
Seow, 2009	Case-control	617	Australia	1	7	Significant	Children who do not speak English had greater risk: OR: 5.62 (1.43–22.11) p=0.032

Appendix 16: Characteristics and findings from the studies exploring employment status and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Fontana et al 2011	Cohort	329	United States	1	8	Significant	Children with one or no adult with job were significantly at risk of developing caries [OR = 3.64; CI = 1.49-8.85].
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Significant	Both parents are jobless at 3years; Adjusted Relative risk increases (RRI) (%) = 11% (CI, 6–16, P < 0.001)
Mahesh, 2013	Case-control	380	India	3	2	Significant	Unemployment in mothers is a significant risk factor of caries in children [Risk Ratio: 3.45, CI = 1.70–6.99].
dos Santos Pinto et al., 2016	Cohort	538	Brazil	2	9	Not Significant	Not Significant
Pinto et al, 2016	Cohort	538	Brazil	2	7	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Tanaka et al 2013	Cohort	315	Japan	1	7	Not Significant	Not Significant

Appendix 17: Characteristics and findings from the studies exploring number of hours spent at work and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Ismail, 2009	Cohort	788	United States	1	6	Not Significant	Not Significant
Tanaka et al 2013	Cohort	315	Japan	1	7	Not Significant	Not Significant
Schroth et al., 2014	Cohort	207	Canada	1	9	Not Significant	Not Significant

Appendix 18: Characteristics and findings from the studies exploring type of work and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Cabral et al, 2017	Cohort	495	Brazil	2	6	Yes	Low CBO* or unemployed is associated with the child's caries status :Mother: HR = 6.04 2.22–16.4; Father: HR = 3.49 1.75–6.95
Mattila et al., 2000	Cohort	828	Finland	1	7	Yes	Children of “blue collar” parents more likely to develop caries; p = 0.015. Odds/Risk ratio not presented.
Meurman and Pienihäkkinen 2010	Cohort	337	Finland	1	7	Yes	Children of parents with blue collar employment are at greater risk of developing caries, Odds Ratio: 1.9; CI = 1.0–3.4; p = 0.034
Skafida et al, 2018	Cohort	3770	UK	1	8	Yes	The mother's type of NS-SEC occupation is significantly associated with the child's dmft [Never worked OR = 3.47 (1.56-7.74); Semi-routine and routine OR = 1.95[1.44 -2.64]]
Tanaka et al 2013	Cohort	315	Japan	1	7	Not Significant	Not Significant

*CBO: Brazilian classification of occupations

Appendix 19: Characteristics and findings from the studies exploring gender and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Lulić-Dukić et al 2001	Case-control	145	Croatia	1	7	Significant	Males were more likely to develop caries than girls [p = 0.02]. Odds/Risk ratio not presented
Sridevi et al, 2018	Case-control	690	India	3	7	Significant	Males are at higher risk male: 1.28 [0.06]
Yokomichi, 2015	Cohort	117175	Japan	1	8	Significant	Males are more at risk; Adjusted Relative risk increases (RRI) (%) = Boys: 3% (CI, 2-5, P < 0.001)
Guedes, 2016	Cohort	469	Denmark	1	7	Significant	Males are more at risk. Being female is protective; Female 0.59 (0.39–0.90) p =0.015
Ismail et al 2009	Cohort	788	United States	1	6	Significant	Females have a higher risk of developing caries, when compared with male counterparts [*IRR = 1.3; CI = 1.0 – 1.7; p=0.02]
Ismail et al 2008	Cohort	788	United States	1	6	Significant	Females are more at risk of developing caries in children.
Seow, 2009	Case-control	617	Australia	1	7	Not Significant	Not Significant
Slade, 2006	Case-control	1398	Australia	1	6	Not Significant	Not Significant
Agarwal, 2011	Case-control	150	India	3	6	Not Significant	Not Significant
Mahesh, 2013	Case-control	380	India	3	2	Not Significant	Not Significant
Nunn et al., 2009	Case-control	4431	United States	1	8	Not Significant	Not Significant
Werneck, 2008	Case-control	104	Canada	1	7	Not Significant	Not Significant
Menon, 2013	Case-control	800	India	3	8	Not Significant	Not Significant
Schroth et al 2014	Cohort	207	Canada	1	9	Not Significant	Not Significant
Wigen et al 2011	Cohort	0	Norway	1	9	Not Significant	Not Significant
Peltzer et al 2014	Cohort	597	Thailand	2	9	Not Significant	Not Significant
Schluter et al 2007	Cohort	1018	New Zealand	1	8	Not Significant	Not Significant

Ju et al	Cohort	1720	Australia	1	8	Not Significant	Not Significant
Ohsuka et al	Case-control	232	Japan	1	8	Not Significant	Not Significant
Nishide et al, 2018	Cohort	463	Japan	1	6	Not Significant	Not Significant
Skafida et al, 2018	Cohort	3770	UK	1	8	Not Significant	Not Significant
Tanaka, 2013	Cohort	315	Japan	1	7	Not Significant	Not Significant
Wong et al., 2012	Cohort	358	Hong Kong	2	7	Not Significant	Not Significant
Warren, 2016	Cohort	232	United States	1	9	Not Significant	Not Significant
Meurman and Pienihäkkinen	Cohort	337	Finland	1	7	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Sanders and Slade, 2010	Cohort	1398	Australia	1	8	Not Significant	Not Significant

Appendix 20: Characteristics and findings from the studies exploring parental education and risk of caries

Author	Study design	Country	Sample Size	HDI	NOS (9)	Conclusion	
Ju et al, 2016	Cohort	Australia	1,720	1	7	Significant	Maternal education is a risk factor: Year 9 or below: 1.45 (1.20–1.76)
Leroy, 2012	Cohort	Belgium	1057	1	8	Significant	Low parental education is a risk of developing caries: Mother: 2.37 [1.02–5.52]; Father: 2.23 [1.01–4.91]
Cabral et al, 2017	Cohort	Brazil	495	2	6	Significant	Parental education is a risk factor: Parents with less than High school / University qualifications are at higher risks: Mother: 1.98 1.35–2.91; Father: 2.23 1.44–3.45
Goettems et al, 2018	Cohort	Brazil	1303	2	8	Significant	Lower number of years of maternal education is a risk factor: [Path Analysis = 0.16; p<0.001]
Peres et al, 2017	Cohort	Brazil	1129	2	9	Significant	Maternal education is a risk factor: ≤4 years of education: RR = 4.1 [2.1 to 8.0]
Ribeiro et al., 2017	Cohort	Brazil	388	2	7	Significant	Maternal education is a risk factor: Low maternal education level (number of years of study (e.g., >8 vs ≤8)) was associated with caries IRR = 1.43, CI: 1.09–1.88
Zhou et al., 2012	Cohort	China	225	2	7	Significant	Maternal education is a risk factor: Children of mother's with less than 12 years of schooling at greater risk.

							Incidence Density Ratio = 0.35 [0.15–0.83]
Mattila et al., 2000	Cohort	Finland	828	1	7	Significant	Maternal education is a risk factor: The higher the mother's basic educational level (higher than 9 years), the more frequently her child's dmft = 0 ($p < 0.001$). The mother's higher degree of vocational education demonstrated a significant ($p < 0.001$) association with dmft = 0.
Wong et al., 2012	Cohort	Hong Kong	358	2	7	Significant	Parental education >15 years is a protective factor: Both parents: IRR= 0.40, P = 0.002
Tanaka et al 2013	Cohort	Japan	315	1	7	Significant	Parental education >15 years is a protective factor: Mother: 0.34 (0.16-0.70); Father: 0.54 (0.30-0.98)
Grytten et al., 1988	Cohort	Norway	231	1	7	Significant	Children of mothers with low education were more likely to develop dental caries.
Wigen and Wang, 2011	Cohort	Norway	1348	1	9	Significant	Maternal education is a risk factor: Low maternal education level (≤12 years of schooling) was associated with caries at both bivariate and multivariate levels: 1.5 [1.1 – 2.3]
Wigen and Wang, 2014	Cohort	Norway	1348	1	9	Significant	Maternal education is a risk factor: Low maternal education level (≤12 years of schooling) was associated with caries at both bivariate and multivariate levels: 1.9 [1.3 – 2.8]
Wigen, 2011	Cohort	Norway	1348	1	9	Significant	Maternal education is a risk factor:

							Low maternal education level (≤12 years of schooling) was associated with caries at both bivariate and multivariate levels: 1.5 [1.1 – 2.3]
Gao et al 2010	Cohort	Singapore	1782	1	7	Significant	Low parental education is a risk factor: High caries incidence was identified among children of parents with low education attainment ($p < 0.05$)
Lam et al, 2017	Cohort	Singapore	543	1	7	Significant	Maternal education is a risk factor: Mother attending University is protective and significant: 0.40 (0.18-0.90)
Julihn, et al, 2018	Cohort	Sweden	73,658	1	8	Significant	Maternal education is a risk factor: Children of parents with < 9 years of education areat higher risk of ECC: AOR = 1.99 (1.79 to 2.22)
Grindefjord, 1996	Cohort	Sweden	692	1	8	Significant	Maternal education is a risk factor: children with maternal education <= 9 years at risk: 2.58 [1.66 – 4.01]
Östberg et al., 2017	Cohort	Sweden	243	1	8	Significant	Parental education: Mother: ≤9 years [OR = 3.02 (1.08–8.49)]; Father: ≤9 years [OR = 0.82 (0.37–2.19)]
Peltzer, 2014	Cohort	Thailand	597	2	9	Significant	Maternal education is a risk factor: High school: 2.13 (1.09–4.16) None 1.00 Primary 1.78 (0.92–3.45) High school 2.13 (1.09–4.16)

							Post-high school 1.14 (0.60–2.17)
Thitasomakul et al., 2009	Cohort	Thailand	495	2	7	Significant	Caries increment was significantly higher among children whose mothers had only primary school education. Maternal education is a risk factor: 1.1 (p<0.001).
Skafida et al, 2018	Cohort	United Kingdom	3770	1	8	Significant	Maternal education is a risk factor: No qualifications OR = 2.29*** [1.47,3.58]
Hong et al., 2014	Cohort	United States	509	1	8	Significant	Parental education is a protective risk factor: Parent with minimum education of graduate had an Odds ratio of 0.77; p= 0.008
Warren, 2016	Cohort	United States	232	1	9	Significant	Children of mothers with lower educational status ("Less than high school", "High school or GED", or "Attended college") have higher risks of developing caries. This is significant at bivariate level with a p-value of 0.0229.
Seow, 2009	Case-control	Australia	617	1	7	Significant	Maternal education >12 years is a protective factor: In Childcare setting: OR = 0.26 (0.08–0.80); In public clinic setting: OR = 0.69 (0.22–2.22)
Lima et al., 2016	Case-control	Brazil	1016	2	7	Significant	Parental education is a protective risk factor: mother p<0.002; father p<0.001

Tavares et al., 2008	Case-control	Brazil	966	2	7	Significant	Maternal education is a risk factor: 1.34; p=0.049
Agarwal, 2011	Case-control	India	150	3	6	Significant	Maternal education is a risk factor: P = 0.036
Nunn et al., 2009	Case-control	United States	4431	1	8	Significant	Parental education is a protective risk factor: College graduate and beyond: 0.22 (0.12, 0.41)
Pinto et al, 2017	Cohort	Brazil	538	2	7	Not significant	Not significant
Piva et al, 2017	Cohort	Brazil	163	2	8	Not significant	Not significant
Rocha, 2017	Cohort	Brazil	73	2	7	Not significant	Not significant
Schluter et al 2007	Cohort	New Zealand	1,018	1	8	Not significant	Not significant
Fontana et al 2011	Cohort	United States	329	1	8	Not significant	Not significant
Ismail, 2008	Cohort	United States	788	1	6	Not significant	Not significant
Ismail, 2009	Cohort	United States	788	1	6	Not significant	Not significant
Tiberia et al., 2007	Case-control	Canada	132	1	3	Not significant	Not significant
Mahesh, 2013	Case-control	India	380	3	2	Not significant	Not significant
Menon et al., 2013	Case-control	India	800	3	8	Not significant	Not significant
Huntington et al., 2002	Case-control	United States	120	1	6	Not significant	Not significant
Smith et al., 2002	Case-control	United States	60	1	6	Not significant	Not significant
dos Santos Pinto et al., 2016	Cross-sectional nested in a cohort study	Brazil	538	2	9	Not significant	Not significant
Nunes, 2014	Cross-sectional nested in a cohort study	Brazil	244	2	8	Not significant	Not significant

Pinto et al, 2016	Cross-sectional nested in a cohort study	Brazil	538	2	7	Not significant	Not significant

Appendix 21: Characteristics and findings from the studies exploring social class and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Peres et al, 2017	Cohort	1129	Brazil	2	9	Significant	**Family class** is significantly associated with the child's caries status • 5 : [highest] - Base • 4: RR = 1.4 [0.8 to 2.3] • 3: RR = 1.8 [1.1 to 2.8] • 2: RR = 2.1 [1.3 to 3.3] • 1 [lowest]: 2.6 [1.7 to 4.1]
Menon et al., 2013	Case-control	800	India	3	8	Significant	Participants under "Lower middle SES" are at three times more risk of developing caries
Sridevi et al, 2018	Case-control	690	India	3	7	Significant	Low socioeconomic status is a significant predictor of caries in children; p=0.02
Agarwal, 2011	Case-control	150	India	3	6	Significant	children from low socioeconomic class are likely to have more ECC at preschool age
Ribeiro et al., 2017	Cohort	388	Brazil	2	7	Not Significant	Not Significant
Wagner and Heinrich-Weltzien, 2017	Case-control	1162	Germany	1	9	Not Significant	Not Significant

Appendix 22: Characteristics and findings from the studies exploring household income and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Peres et al, 2017	Cohort	1129	Brazil	2	9	Significant	Lowest Income group: 2.6 [1.7 to 4.1]
Julihn et al, 2016	Cohort	73,658	Sweden	1	8	Significant	Lower Income associated with caries: 1.98 (1.71 to 2.38)
Sanders and Slade, 2010	Cohort	1398	Australia	1	8	Significant	Lower Income associated with caries: <= AUD$31,199 [PR = 1.38 (1.15 to 1.66)]
Cabral et al, 2017	Cohort	495	Brazil	2	6	Significant	Lower Income associated with caries: [Path Analysis = - 0.09; p<0.001]
Goettems et al, 2018	Cohort	1303	Brazil	2	8	Significant	Lower Income associated with caries: HR = 1.89 [1.24–2.90]
Piva et al, 2017	Cohort	163	Brazil	2	8	Significant	Lowest Income group: RR = 1.86 (1.25-2.76); p=0.002
Zhou et al., 2012	Cohort	225	China	2	7	Significant	HIGHER Family Income [≥450 USD] is a significant risk of caries; 2.40 (1.23–4.66); p=0.010
Peltzer, 2014	Cohort	597	Thailand	2	9	Significant	Higher Income protective: >200,000 Thai Bhat= 0.31 (0.12–0.83)
Fontana, 2011	Cohort	388	United States	1	8	Significant	Lower Income associated with caries: 1.21 (1.06-1.37)
Slade, 2006	Case-control	1398	Australia	1	6	Significant	Lowest Income group: 1.55 (1.21–1.98)
Lima et al., 2016a	Case-control	1016	Brazil	2	7	Significant	Family income was related to caries in children; P <0.001
Werneck, 2008	Case-control	104	Canada	1	7	Significant	Income group <$40,000 were almost 4 times more likely to have a child with ECC. OR = 3.73 (1.51–9.18); p=0.04
Nunn et al., 2009	Case-control	4431	United States	1	8	Significant	Annual income >$20,000 had a relatively lower odds ratio of 0.48 (0.34 to 0.68).

Nunes, 2014	Cohort	244	Brazil	2	8	Not Significant	Not Significant
dos Santos Pinto et al., 2016	Cohort	538	Brazil	2	9	Not Significant	Not Significant
Pinto et al, 2016	Cohort	538	Brazil	2	7	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Ismail et al., 2008	Cohort	788	United States	1	7	Not Significant	Not Significant
Ismail, 2009	Cohort	788	United States	1	6	Not Significant	Not Significant
Warren, 2016	Cohort	232	United States	1	9	Not Significant	Not Significant
Pinto et al, 2017	Cohort	538	Brazil	2	7	Not Significant	Not Significant
Schroth et al., 2014	Cohort	207	Canada	1	9	Not Significant	Not Significant
Wong et al., 2012	Cohort	358	Hong Kong	2	7	Not Significant	Not Significant
Tanaka, 2013	Cohort	315	Japan	1	7	Not Significant	Not Significant
Thitasomakul et al., 2009	Cohort	495	Thailand	2	7	Not Significant	Not Significant
Tavares et al., 2008	Case-control	966	Brazil	2	7	Not Significant	Not Significant
Huntington et al., 2002	Case-control	120	United States	1	6	Not Significant	Not Significant

Appendix 23: Characteristics and findings from the studies exploring Single-Parent Status and risk of caries in children under six

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Piva et al, 2017	Cohort	163	Brazil	2	8	Significant	Married/cohabiting is protective: 1.36 (1.02 - 1.85)
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	Children of mothers cohabiting 3.3 (1.5-7.6)
dos Santos Pinto et al., 2016	Cohort	538	Brazil	2	9	Significant	Single-parent children: 1.57 (1.05 – 2.35)
Julihn et al, 2016	Cohort	73,658	Sweden	1	8	Significant	Single-parent children: 1.55 (1.42 to 1.71)
Pinto et al, 2016	Cohort	538	Brazil	2	7	Significant	Single-parent children: 1.62 [1.04–2.52]
Fontana, 2011	Cohort	329	United States	1	8	Significant	Number of adults caring for child (per 1 less) increases risk of developing ECC; p<=0.05
Slade, 2006	Case-Control	1398	Australia	1	6	Significant	Single-parent children: 1.35 (1.10-1.65)
Lima et al., 2016	Case-control	1016	Brazil	2	7	Not significant	Not significant
Huntington et al., 2002	Case-control	120	United States	1	6	Not significant	Not significant
Pinto et al, 2017	Cohort	538	Brazil	2	7	Not significant	Not significant
Östberg et al., 2017	Cohort	243	Sweden	1	8	Not significant	Not significant
Peltzer, 2014	Cohort	579	Thailand	2	9	Not significant	Not significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not significant	Not significant
Schluter et al (2007)	Cohort	1,018	New Zealand	1	8	Not significant	Not significant

Appendix 24: Characteristics and findings from the studies exploring other personal relationships and risk of caries in children under six

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	Children spending a less time with other children on weekdays were more likely to have caries
Ohsuka et al., 2009	Case-control	232	Japan	1	8	Significant	Those living with grandparents had higher risk of developing caries [OR = 3.868; CI= 0.955–15.670]
Seow, 2009	Case-control	617	Australia	1	7	Significant	Those not living with original family more at risk of developing caries
Ismail et al., 2008	Cohort	788	United States	1	7	Not Significant	Not Significant

Appendix 25: Characteristics and findings from the study exploring community participation and risk of caries in children under six

Author	Study design	Age	Sample Size	Country	HDI	Risk definitions and outcome	NOS
(Tavares et al., 2008)	Case Control	5 years	966	Brazil	2	**Exposure:** Mother's participation in neighbourhood groups **Outcome definition:** Caries-free cases defined as 5-year-olds without any record of disease diagnosis and/or any curative treatment procedure until the data collection **Findings**: No significant difference seen between mothers who participate in community groups and mothers who do not. (p=0.36) **Conclusion**: Not significant	7

Appendix 26: Characteristics and findings from the studies exploring birthweight and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Kay et al, 2010	Cohort	985	UK	1	8	Significant	OR: 1.08 (95% CI: 1.03, 1.13) per 100 g increase, P = 0.002
Hong et al., 2014	Cohort	509	United States	1	8	Significant	Low birth weight: Odds Ratio is 1.70 [p=0 .04]
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Significant	Birth weight (g) <2500 is protective
Lima et al., 2016	Case-control	1016	Brazil	2	7	Not significant	Not significant
Slade, 2006	Case-control	1,398	Australia	1	6	Not Significant	Not Significant
Sridevi et al, 2018	Case-Control	690	India	3	7	Not Significant	Not Significant
Seow, 2009	Case-control	617	Australia	1	7	Not Significant	Not significant
Sanders and Slade	Cohort	1,058	Australia	1	8	Not Significant	Not Significant
Zhou et al., 2012	Cohort	225	China	2	7	Not Significant	Not Significant
Wigen and Wang, 2011	Cohort	1,348	Norway	1	9	Not Significant	Not Significant
Nirunsittirat, 2016	Cohort	860	Thailand	2	8	Not Significant	Not Significant
Peltzer, 2014	Cohort	597	Thailand	2	9	Not Significant	Not Significant
Thitasomakul et al., 2009	Cohort	495	Thailand	2	7	Not Significant	Not Significant

Fontana, 2011	Cohort	329	United States	1	8	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Ju et al, 2016	Cohort	1,720	Australia	1	7	Not Significant	Not significant

Appendix 27: Characteristics and findings from the studies exploring child's birth order and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS (9)	Conclusion	
Dabawala et al., 2017	Case-control	422	India	3	8	Significant	Higher birth order were associated with higher risk of developing caries
Mahesh, 2013	Case-control	380	India	3	2	Significant	First born children have higher risks: First born child [Risk Ratio = 4.182; p = 0.0001]
Tiberia et al., 2007	Case-control	132	Canada	1	3	Significant	Greater than second born more at risk
Sanders and Slade, 2010	Cohort	1,398	Australia	1	8	Significant	Higher risk of caries associated with second or subsequent birth order: >first born = 1.46 (1.17, 1.83)
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Significant	Adjusted Relative risk increases (RRI) (%)26% (CI, 24–29, P < 0.001) for non-firstborn babies
Huntington et al., 2002	Case-control	120	United States	1	6	Not Significant	Not Significant
Menon et al., 2013	Case-		India	3	8	Not Significant	Not Significant
Ohsuka et al., 2009	Case-		Japan	1	8	Not Significant	Not Significant
Werneck, 2008	Case-		Canada	1	7	Not Significant	Not Significant
Leroy, 2012	COHORT		Belgium	1	8	Not Significant	Not Significant
Nishide et al, 2018	Cohort		Japan	1	6	Not Significant	Not Significant
Peltzer, 2014	Cohort		Thailand	2	9	Not Significant	Not Significant
Wigen and Wang, 2011	Cohort		Norway	1	9	Not Significant	Not Significant

Appendix 28: Characteristics and findings from the studies exploring pre-term babies and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS (9)	Conclusion	Comments
Sridevi et al, 2018	Case-Control	690	India	3	7	Significant	Multivariate analysis showed participants with preterm birth are more prone (OR: 1.65) to ECC than full-term birth.
Nirunsittirat, 2016	Cohort	860	Thailand	2	8	Significant	Preterm babies: RR = 0.61 (0.43, 0.85); i.e., the mean dmfs score was 39% lower for preterm children than in children delivered full-term.
Seow, 2009	Case control	617	Australia	1	7	Not Significant	Not Significant
Lima et al., 2016a	Case-control	1,016	Brazil	2	7	Not Significant	Not Significant
Ju et al	Cohort	1,720	Australia	1	8	Not Significant	Not Significant
Schroth et al., 2014	Cohort	207	USA	1	9	Not Significant	Not Significant
Wigen and Wang, 2011	Cohort	1,348	Norway	1	9	Not Significant	Not Significant
Zhou et al., 2012	Cohort	225	China	2	7	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Not Significant	Not Significant

Appendix 29: Characteristics and findings from the studies exploring type of birth and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Boustedt, et al, 2018	Cohort	551	Sweden	1	7	Significant	There were higher caries occurrence among children delivered with C-section; AOR = 2.5 (1.7–4.0)
Fontana et al (2011)	Cohort	329	UNITED STATES	1	9	Significant	Children delivered through vaginal birth were more at approximately twice at risk of developing ECC. P<0.05
Sridevi et al, 2018	Case-control	690	India	3	7	Not Significant	Not Significant

Appendix 30: Characteristics and findings from the studies exploring maternal age at child birth and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS (9)	Conclusion	Details
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Significant	Children of younger mothers are more at risk of developing caries <25 years: 17% (CI, 14–20, P < 0.001) for a maternal age <25 years
Warren, 2016	Cohort	232	United States	1	9	Significant	"The study found that younger maternal age was associated with caries in this population": Maternal age [OR = 0.931 (0.871 - 0.996); p = 0.0381]
Wigen, 2011	Cohort	1348	Norway	1	9	Significant	Children of younger mothers are more at risk of developing caries <25years = 1.9 (1.2–3.2) (Bivariate analysis)
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	Children of younger mothers are more at risk of developing caries <20yrs = 5.0 (1.3-19.8)
Peres et al, 2017	Cohort	1129	Brazil	2	9	Significant	Age of mother at childbirth is significant risk: Older mothers are protective against ECC: 20–29 = 0.6 [0.4 to 0.9], 30–39 = 0.5 [0.3 to 0.7]
Lam et al, 2017	Cohort	543	Singapore	1	7	Significant	Children of older mothers are more at risk of developing caries ≥34 years 3.07 (1.15-8.20)
Julihn et al.,	Cohort	73,658	Sweden	1	8	Significant	Mothers >34 years = 1.26 (1.16 to 1.36); <25 = 2.06 (1.89 to 2.24)

Tavares et al., 2008	Case-control	966	Brazil	2	7	Significant	Children of old mothers are more at risk of developing caries >26yrs =1.66 (1.19-2.32)
Ju et al, 2016	Cohort	1,720	Australia	1	8	Not Significant	Not Significant
Menon et al., 2013	Case-control	800	India	3	8	Not Significant	Not Significant
Peltzer, 2014	Cohort	365	Thailand	2	9	Not Significant	Not Significant
Piva et al, 2017	Cohort	163	Brazil	2	8	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
Sanders and Slade, 2010	Cohort	1398	Australia	1	8	Not Significant	Not Significant
Schluter et al 2007	Cohort	1,018	New Zealand	1	8	Not Significant	Not Significant
Skafida et al, 2018	Cohort	3770	UK	1	8	Not Significant	Not Significant
Smith et al., 2002	Case-control	60	United States	1	6	Not Significant	Not Significant
Zhou et al., 2012	Cohort	225	China	2	7	Not Significant	Not Significant

Appendix 31: Characteristics and findings from the studies exploring Maternal Oral Health Behaviour and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	Irregular tooth brushing pattern of parents is a risk factor of caries in children; Mothers: 2.2 (1.4-3.5); Fathers: OR 1.4, (1.1-1.7)
Grytten et al., 1988	Cohort	231	Norway	1	7	Significant	On bivariate analysis, there were more caries in children with mothers attending less than once a year (p=0.039)
Werneck, 2008	Case-control	104	Canada	1	7	Significant	Children of mothers who did not have dental visit in the past year were significantly at risk of developing caries (OR = 4.10 (1.78–9.43)
Agarwal, 2011	Case-control	150	India	3	6	Significant	Significant in bivariate analysis (p=0.038)
Lima et al., 2016	Case-control	1016	Brazil	2	7	Significant	There is a significant difference in mothers who had dental appointment during pregnancy and those who did not. P<0.001 (at univariate analysis)
Östberg et al., 2017	Cohort	243	Sweden	1	8	Not significant	Not significant
Smith et al., 2002	Case-control	60	United States	1	6	Not significant	Not significant
Tavares et al., 2008	Case-control	966	Brazil	2	7	Not significant	Not significant

Appendix 32: Characteristics and findings from the studies exploring age at start of tooth brushing and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Thitasomakul et al., 2009	Cohort	495	Thailand	2	7	Significant	Children who did not have their teeth brushed before 9 months had significantly higher risks of developing caries. Incidence Density ratio of 1.5; $p \leq 0.001$
Wong et al., 2012	Cohort	358	Hong Kong	2	7	Significant	Children who started later than 12 months had significant risk of developing caries compared to children who started brushing between 6-12 months: 6–12 months [Ref]; 13–18 months 2.46 [1.41–4.30]; 19–24 months 2.28 [1.27–4.07]; 24 months 2.60[1.50–4.51]
Lima et al., 2016a	Case-control	1016	Brazil	2	7	Significant	Children who started tooth brushing after the 1st tooth erupted are at higher risk of ECC; $p < 0.001$
Lulić-Dukić et al., 2001	Case-Control	145	Croatia	1	7	Significant	Children who started tooth brushing at 25 months or more are at higher risk of ECC; $p < 0.001$
Wagner and Heinrich-Weltzien, 2017	Case-control	1162	Germany	1	9	Significant	Brushing before 1st year of life was protective against ECC [OR = 0.2, CI =0.07–0.54; p=0.002]
Nunes, 2014	Cohort	244	Brazil	2	8	Not Significant	Not Significant
Dabawala et al., 2017	Case-control	422	India	3	8	Not Significant	Not Significant

Appendix 33: Characteristics and findings from the studies exploring parent OH support and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Nunes, 2014	Cohort	244	Brazil	2	8	Significant	hildren of mothers that supervise their children's toothbrushing were at lower risk of developing caries; 0.27 (0.11-0.66); p=0.004
Yokomichi, 2015	Cohort	117175	Japan	1	8	Significant	families where there is no support for child rearing are at higher risk of developing ECC; 17% (CI, 13–22, P < 0.001)
Huntington et al., 2002	Case-control	120	United States	1	6	Significant	Children from families affected by caries were less likely to have adult supervision during routine oral hygiene; No supervision group; p=0.048
Mahesh, 2013	Case-control	380	India	3	2	Significant	Brushing not supervised by an adult is a risk of developing ECC; p=0.0219
Wagner and Heinrich-Weltzien, 2017	Case-control	1162	Germany	1	9	Significant	Bivariate analysis found statistically significant associations between caries experience in children and supervision/regular second brush by parent (OR = 0.02, 95 % CI 0.01–0.05, p < 0.001).
Werneck, 2008	Case-control	104	Canada	1	7	Significant	Children who were never supported to visit a dentist had higher caries risk OR 2.86 (1.23–6.65)
Pinto et al, 2016	Cohort	538	Brazil	2	7	Not Significant	Not Significant
Nishide et al, 2018	Cohort	463	Japan	1	6	Not Significant	Not Significant
Schroth et al., 2014	Cohort	207	Canada	1	9	Not Significant	Not Significant
Dabawala et al., 2017	Case-control	422	India	3	8	Not Significant	Not Significant
Lima et al., 2016a	Case-control	1016	Brazil	2	7	Not Significant	Not Significant

Appendix 34: Characteristics and findings from the studies exploring family caries history and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS (9)	Conclusion	Summary
Fontana, 2011	Cohort	329	United States	1	8	Significant	Primary Caregivers' caries history is a risk factor of caries in children; p<0.05 Other children have cavities or fillings: OR = >2
Grytten et al., 1988	Cohort	231	Norway	1	7	Significant	Children of mothers with missing teeth were more likely to develop dental caries; p=0.002
Ismail, 2009	Cohort	788	United States	1	6	Significant	caregivers caries experience is a risk factor of developing caries in children; 1.4 [1.1–1.9]
K. Boustedt, et al, 2018	Cohort	551	Sweden	1	7	Significant	Siblings caries history is significant risk factor in children; OR = 2.1 (1.1–3.5)
Mattila et al., 2000	Cohort	828	Finland	1	7	Significant	Mothers caries history = [OR = 1.3; CI 1.0-1.6, p = 0.035]
Nishide et al, 2018	Cohort	463	Japan	1	6	Significant	Mothers caries history = OR = 1.84 (1.09–3.12)
Pinto et al, 2017	Cohort	538	Brazil	2	7	Significant	Mothers with caries: 1.67 [0.87-2.97]; Adjusted = 1.41 [0.78-2.57]
Warren, 2016	Cohort	232	United States	1	9	Significant	Mothers with caries:1.045 [1.018 - 1.072]
Lima et al., 2016a	Case-control	1016	Brazil	2	7	Significant	Significant: Mother's caries history: 2.61 [1.45-4.67; p=0.001]; Father's: 1.72 [1.02-2.89; p=0.04]
Plonka et al, 2013	Case-Control	552	Australia	1	7	Significant	Mothers with caries: OR 5.8 [2.1–15.9]
Peltzer, 2014	Cohort	365	Thailand	2	9	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant
dos Santos Pinto et al., 2016	Cohort	538	Brazil	2	9	Not Significant	Not Significant

Appendix 35: Characteristics and findings from the studies exploring Smoking and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS (9)	Conclusion	Summary
Wigen, 2011	Cohort	1348	Norway	1	9	Significant	Smoking mothers(yes): Bivariate = (OR = 1.9 [1.0 – 3.6]
Boustedt, et al, 2018	Cohort	551	Sweden	1	7	Significant	Smoking parents: Mother smoking (at birth): AOR = 2.6 (1.2–5.6); Father smoking (at birth): AOR = 3.0 1.7–4.9)
Ju et al	Cohort	1,720	Australia	1	8	Not Significant	Not Significant
Tanaka et al, 2015	Cohort	76920	Japan	1	9	Significant	Exposed to: only maternal smoking during pregnancy = 1.14 (1.00 to 1.30) 0.05; only household smoking at 4 months = 1.60 (1.52 to 1.69) <0.01; to second-hand smoke during pregnancy and at 4 months = 1.89 (1.77 to 2.02) <0.01
Yokomichi, 2015	Cohort	117,175	Japan	1	8	Significant	15% increase (CI, 13–17, P < 0.001) in caries seen in children where there is maternal or paternal smoking
Plonka et al, 2013	Case-Control	552	Australia	1	7	Significant	Mother smoked during pregnancy (p = 0.02) and smoker in household during pregnancy and at 6, 12, 18 months (p = 0.07 to p = 0.01)
Julihn	nested in cohort study	73,658	Sweden	1	8	Significant	Maternal smoker: 1.94 (1.75 to 2.16)
Leroy, 2012	Cohort	1057	Belgium	1	8	Not Significant	Not Significant
Peltzer et al 2014	Cohort	597	Thailand	2	9	Not Significant	Not Significant
Schluter et al 2007	Cohort	1,018	New Zealand	1	8	Not Significant	Not Significant
Warren, 2016	Cohort	232	United States	1	9	Not Significant	Not Significant
Rocha, 2017	Cohort	73	Brazil	2	7	Not Significant	Not Significant

Appendix 36: Characteristics and findings from the studies exploring financial support (access to health insurance) and risk of caries

Author	Study design	Sample Size	Country	HDI	NOS	Conclusion	Details
Brickhouse, 2008	Cohort	23936	UNITED STATES	1	9	Significant	Type of insurance is a risk factor of ECC; State Children's Health Insurance Program-enrolled children were significantly less likely to have untreated dental caries than were Medicaid-enrolled children (odds ratio [OR]=0.74; 95% CI=0.67, 0.82)
Fontana et al (2011)	Cohort	329	UNITED STATES	1	9	Significant	Children with no private health insurance (or have Medicaid or Hoosier Healthwise) were seen to be more at risk of developing ECC, with an Odds Ratio of approximately twice that of children with private Insurance
Huntington et al., 2002	Case-control	120	United States	1	6	Significant	Families affected by ECC were less likely to have private dental insurance and were more likely to be uninsured (P-value=0.005).
Nunn et al., 2009	Case-control	4431	United States	1	8	Significant	hildren without health insurance or who were on Medicaid were over three times more likely to have caries than children with private health insurance (OR = 3.3, P < 0.001).
Tiberia et al., 2007	Case-control	132	Canada	1	3	Significant	Children who had dental insurance were less likely to have decay (P=0.001), While children with government assistance were more likely to have decay (P=0.004)
Werneck, 2008	Case-control	104	Canada	1	7	Significant	children with caries were more likely than controls to be without dental insurance; Without dental insurance: OR = 6.75 (2.85–16.00); p= < 0.001

Appendix 37: Summary of rationale provided in review papers between SDH and Early Childhood Caries

Authors	Exposure	Influences caries	Rationale provided
Peltzer et al (2014)	Place	Yes	Directly through water Fluoridation
Hong et al (2014)	Place	Yes	Directly through water Fluoridation
Sanders and Slade 2010	Place	Yes	Directly through water Fluoridation
Tiberia et al (2007)	Place	No	N/A
Mahesh et al (2013)	Place	Yes	Water Fluoridation
Ohsuka et al-2009	Place	Yes	Children living in rural areas have increased risks of developing caries because of deficient infrastructures and because child care support usually provided by grandparents in rural areas
Mattila et al (2000)	Place	Yes	Rationale proposes was that rural children's teeth were brushed less frequently than those of urban children, and the rural populations value dental health less or consider them less important, than in urban population.
Ju et al-2016	Place	Yes	- Families residing in rural areas have limited access to fresh food produce and healthy food options. - Secondly, they lack nutritional knowledge and culturally appropriate information on healthy food types. - Thirdly, residents have limited access to toothbrushes and fluoridated toothpastes - Lastly, people who live in rural areas also have fewer job opportunities à Low SES
Ismail et al (2009)	Place	Yes	Mechanism not discussed
Brickhouse et al (2008)	Place	Yes	Mechanism not discussed

Gao et al (2010)	Race	Yes	- Ethnic minority families (Malays) have tendencies to adopt dentally unhealthy oral health behaviors, such as frequent intake of sweet desserts. - Ethnicity is also said to exert its effect on dental attendance, through cultural norms and beliefs (e.g., fatalism), faith in other cures, and ethnic beliefs about disease causation and prevention
Nunn (2009)	Race	Yes	Parents of ethnic minority children tended more to visit the dentist for treatment purposes, as opposed to parents seeking preventive care for their children.
Tiberia et al (2007)	Race	Yes	Parents of children with different ethnicities were seen to be mostly uneducated and poor
Werneck et al (2008)	Race	Yes	Immigrants find it difficult to access preventive care owing to a lack of financial resources, dental insurance, language and economic limitations. They also suggested that dentists are also unable to engage with immigrants due to language barrier.
Wigen et al (2010)	Race	Yes	Lower educational attainment of immigrant parents, with reduced ability to adapt to health promoting behaviour There is also 'cultural' differences in child rearing and immigrant parents' beliefs and attitudes towards dental health
Seow et al (2009)	Race	Yes	Not discussed
Wigen et al (2014)	Race	Yes	Not discussed
Wagner et al (2017)	Race	No	N/A
Meurman and Pienihäkkinen	Race	No	N/A
Smith et al	Race	No	N/A

Fontana et al (2011)	Occupation	Yes	Not discussed
Mahesh et al (2013)	Occupation	Yes	Authors proposed that employed mothers are forced to put children in nursery, however, these nurseries are more involved in children's daily diet, general hygiene and oral health care
Mattila et al (2000)	Occupation	Yes	Not discussed
Meurman and Pienihäkkinen (2010)	Occupation	Yes	Not discussed
dos Santos Pinto et al (2017)	Occupation	No	N/A
Ismail et all (2009)	Occupation	No	N/A
Tanaka et al (2013)	Occupation	No	N/A
Schroth (2014)	Occupation	No	N/A
(Agarwal, 2011)	Education	Yes	Uneducated mothers lack the knowledge and awareness of dental health, oral hygiene practices, and feeding habits.
(Zhou et al., 2012)	Education	Yes	Mothers with a lower schooling may not know how to obtain the relevant information with regard to feeding, diet or dental care.
(Gao et al., 2010)	Education	Yes	Education is a component of socio-economic circumstances, which influences dental attendance, which, in turn, affects oral health.
(Grytten et al., 1988)	Education	Yes	Suggested educational attainment influences oral health behaviour
(Lima et al., 2016)	Education	Yes	Education level of parents was considered an important socioeconomic indicator
(Mattila et al., 2000)	Education	Yes	Parents' educational levels was an important social background factor in pre-school children's dental health, and their study showed that children from disadvantaged groups have the lowest levels of dental health
(Ostberg et al., 2017)	Education	Yes	Educated parents are able to promote health better within the family than non educated parents

(Peltzer, 2014)	Education	Yes	Contrary to some previous studies this study found that higher levels of education of the mother were associated with caries increment. Explanation provided was that higher education of the mother means she is usually away from the home and that possibly a grandmother is taking care of the child.
(Ribeiro et al., 2017)	Education	Yes	Parents with lower educational levels are more likely to have reduced access to healthy food or have inadequate choices
(Tanaka, 2013)	Education	Yes	Parents completing higher levels of education more interested in health, and this is likely to influence oral hygiene behaviors
(Tiberia et al., 2007)	Education	Yes	Parents with a higher level of education tended to have insurance from work, which improves access to care and affordability
(Wigen and Wang, 2011)	Education		Low educational level associated with reduced ability to adapt to health promoting behavior, and that mothers do not initiate oral hygiene practices early in their preschool child
(Wigen and Wang, 2014)	Education	Yes	Not discussed
(Wigen, 2011)	Education	Yes	Not discussed
(Nunn et al., 2009)	Education	Yes	Not discussed
(dos Santos Pinto et al., 2016)	Education	Yes	Not discussed
(Seow, 2009)	Education	Yes	Not discussed
(Hong et al., 2014)	Education	Yes	Not discussed
(Huntington et al., 2002)	Education	Yes	Not discussed
(Tavares et al., 2008)	Education	Yes	Not discussed
(Thitasomakul et al., 2009)	Education	Yes	Not discussed
(Warren, 2016)	Education	Yes	Not discussed
(Wong et al., 2012)	Education	Yes	Not discussed

(Nunes, 2014)	Education	No	N/A
(Ismail, 2009)	Education	No	N/A
(Smith et al., 2002)	Education	No	N/A
(Mahesh, 2013)	Education	No	N/A
(Menon et al., 2013)	Education	No	N/A

Appendix 38: Demographic Characteristics of study participants of Child Dental Health Survey 2013

PROGRESS-PLUS Domain	Variable Category	Caries	Caries ($d_3$mft) %	p-Value
Place	1st quintile (most deprived)	457	61%	**<0.001**
	2nd	314	55%	
	3rd	254	50%	
	4th	211	50%	
	5th quintile (least deprived)	120	41%	
	South East	94	45%	**<0.001**
	North West	131	66%	
	Yorkshire	89	53%	
	West Midlands	86	50%	
	South West	75	48%	
	North East	80	49%	
	Wales	289	59%	
	London	80	46%	
	East of England	66	48%	
	East Midlands	88	60%	
	Northern Ireland	278	53%	
	Urban	1044	55%	**0.001**
	Rural	312	48%	
	England	737	48%	**0.026**
	Wales	204	41%	
	Northern Ireland	252	48%	

PROGRESS-PLUS Domain	Variable Category	Caries	Caries ($d_3$mft) %	p-Value
Race	White	1096	53%	**<0.001**
	Asian	131	64%	
	Black	58	46%	
	Mixed	49	47%	
	Other ethnicities	22	82%	
	White	1096	53%	0.158
	Non-White	260	56%	
Occupation	Higher managerial, admin, professional occupations	771	52%	**0.1688**
	Intermediate occupations	223	52%	
	Routine and manual occupations	279	56%	
	Never worked and long-term unemployed	36	65%	
	Not Classified	47	50%	
Gender	Males	684	54%	0.358
	Females	672	52%	
Education	Degree level or above	569	50%	**0.0006**
	Another type of qualification	671	55%	
	No qualifications	116	63%	
Socioeconomic Status	Ineligible for free school meal	970	50%	**<0.001**
	Eligible for free school meal	223	63%	
Social Capital	Two parent	707	49%	**<0.001**
	Single parent	649	58%	

Appendix 39: Mapping of socio-demographic variables identified in CDHS 2013

Variable Description in CDHS 2013	**PROGRESS-PLUS class**	**Multilevel**
Country	Place	Area Level
English region / Wales / Northern Ireland	Place	Area Level
Public Health England region 2013 (with PHEREG13CDH)	Place	Area Level
2011 Output Area Classification Supergroup (home postcode)	Place	Area Level
England and Wales Urban/Rural Classification (home postcode)	Place	Area Level
England Index of Multiple Deprivation quintile (home postcode)	Place	Area Level
HMRC Children in Low Income Families Measure quintiles (home postcode)	Place	Area Level
5 category ethnicity of child (Combined)	Race/Ethnicity	At Birth
2 category ethnicity of child (combined)	Race/Ethnicity	At Birth
NSSEC 8 category analytical classes	Occupation	Parent Level
NS-SEC 5 category	Occupation	Parent Level
NS-SEC 3 categories	Occupation	Parent Level
Parent taken time off work in last 6 months because of child's dental	Occupation	Parent Level
Time off work because of child's dental health in last 6 months	Occupation	Parent Level
Employment status	Occupation	Parent Level
Whose work details recorded (FRP)	Occupation	Parent Level
Sex	Gender	At Birth
Sex of responding adult	Gender	Parent Level
Highest educational qualification of responding adult	Education	Parent Level
Partner's highest educational qualification	Education	Parent Level

Mapping of socio-demographic variables identified in CDHS 2013 - contd

Variable Description in CDHS 2013	PROGRESS-PLUS class	Multilevel
School type	Socioeconomic Status	Parent Level
Survey school deprivation status	Socioeconomic Status	Parent Level
Free school meal eligibility	Socioeconomic Status	Parent Level
Financial difficulties in last 6 months because of child's dental heal	Socioeconomic Status	Parent Level
Financial difficulties because of child's dental health in last 6 months	Socioeconomic Status	Parent Level
Who owns or rents accommodation	Socioeconomic Status	Parent Level
Relationship of responding adult to child	Social Capital	Parent Level
Living with partner	Social Capital	Parent Level
Partner's relationship to child	Social Capital	Parent Level
Ever had difficulty finding NHS dentist to treat child	PLUS: Local Health System	Area Level
Whether had problems accessing an NHS dentist	PLUS: Local Health System	Area Level
NHS problem: local dentists not taking on NHS patients	PLUS: Local Health System	Area Level
NHS problem: child on NHS only if parents private	PLUS: Local Health System	Area Level
NHS problem: no local school or community dental service	PLUS: Local Health System	Area Level
NHS problem: other	PLUS: Local Health System	Area Level
When last experienced difficulty finding NHS dentist	PLUS: Local Health System	Area Level
Age in years of responding adult	PLUS: Maternal Age	Parent Level
Responding adult's usual dental attendance	PLUS: OHB of Parents	Parent Level
Dental attendance of responding adult - trouble/never grouped	PLUS: OHB of Parents	Parent Level
Who usually brushes child's teeth	PLUS: Support	Parent Level

Caries experience measures in five-year olds in CDHS 2013

Code	Description
ddcyany2ac	(D) Any clinical decay experience in primary teeth (including cavitated enamel caries)

Appendix 40: Factors assessed in each caries risk assessment tool

Factors Yes	**NUS-CRA**	**Cariogram**	**CAT**	**CAMBRA**	**SDH-Based**
Socio-demographic					Yes
Age	Yes				Yes
Ethnicity	Yes				Yes
Family Socioeconomic Status	Yes		Yes	Yes	Yes
Place of residence					Yes
Occupation of parents					Yes
Parental Education					Yes
Single parenthood family					Yes
Difficulty finding dentist					Yes
Behavioural					
Infant feeding history	Yes			Yes	
Diet	Yes	Yes	Yes	Yes	
Fluoride	Yes	Yes	Yes	Yes	
Dental attendance of child			Yes	Yes	
Dental attendance of main carer					Yes
Child brushing habits					Yes
Clinical					
Oral Hygiene	Yes	Yes	Yes	Yes	
Past Caries History	Yes	Yes	Yes	Yes	
White Spot Lesions			Yes	Yes	
Enamel Defects			Yes		
Dental Appliance			Yes	Yes	
Systemic Health	Yes	Yes	Yes	Yes	
Medication				Yes	
Salivary and microbiological (for comprehensive assessment)**					
Saliva flow rate		Yes	Yes	Yes	
Saliva buffering capacity		Yes			
Mutans Streptococci	Yes	Yes	Yes	Yes	
Lactobacilli	Yes	Yes		Yes	

Appendix 41: Summary of number of studies per SDH factor

Progress Class	**Total Papers**
Maternal education	42
Socio-economic	36
Gender	25
Maternal age at birth	21
Birthweight	18
Race	17
Maternal occupation	16
Paternal education	16
Maternal caries history	16
Marital status	14
Pre-term birth	11
Maternal OHB	11
Parent supports child with OHB	11
Smoking	11
Parent place of birth	10
Birth order	10
Number of siblings	10
Paternal occupation	7
Water fluoridation	6
Local health system	6
Child's place of birth	6
Dental insurance	6
Primary language spoken at home	4
Paternal caries history	4
Urban-rural	3
Religion	3
Siblings with caries	3
Environmental smoking	3
Caesarean section delivery	3
Area-level deprivation	2

Appendix 42: Spread of local authority (LA) area across English Regions, Deprivation, and Fluoridation

Row Labels	Total LAs	Total LAs in most deprived quintile	Total LAs in least deprived quintile	Water Fluoridation (Yes)	Water Fluoridation (No)
North East	12	5	0	2	10
Yorkshire and the Humber	21	5	3	1	20
West Midlands	30	6	2	20	10
London	32	16	1	0	32
South West	36	2	4	0	36
North West	39	17	1	2	37
East Midlands	40	9	7	8	32
East of England	47	2	15	0	47
South East	67	3	33	0	67
Grand Total	324	65	66	33	291

Appendix 43: Demographic Characteristics of study participants

PROGRESS-PLUS Domain	Variable Category	Caries	Caries ($d_3$mft) %	p-Value
Place	1st quintile (most deprived)	457	61%	**<0.001**
	2nd	314	55%	
	3rd	254	50%	
	4th	211	50%	
	5th quintile (least deprived)	120	41%	
	South East	94	45%	**<0.001**
	North West	131	66%	
	Yorkshire	89	53%	
	West Midlands	86	50%	
	South West	75	48%	
	North East	80	49%	
	Wales	289	59%	
	London	80	46%	
	East of England	66	48%	
	East Midlands	88	60%	
	Northern Ireland	278	53%	
	Urban	1044	55%	**0.001**
	Rural	312	48%	
	England	737	48%	**0.026**
	Wales	204	41%	
	Northern Ireland	252	48%	

Appendix 44: Mapping of SDH variables to PROGRESS PLUS Domains

DOMAIN	Variable	Number (n)	Minimum	Maximum	Mean	Std. Error	Std. Deviation
OUTCOME	Caries prevalence	324	4.4	49.4	21.8	0.5	8.6
PLACE	IMD 2011	324	4.5	43.4	19.2	0.5	8.4
PLACE	IMD 2015	324	5	42	19.5	0.4	8
PLACE	RuralUrban.1	324	0	1	0.2	0	0.4
PLACE	RuralUrban.2	324	0	1	0.1	0	0.3
PLACE	RuralUrban.3	324	0	1	0.2	0	0.4
PLACE	RuralUrban.4	324	0	1	0.3	0	0.5
PLACE	RuralUrban.5	324	0	1	0	0	0.2
PLACE	RuralUrban.6	324	0	1	0.2	0	0.4
RACE	White	324	29	98.9	89.3	0.7	12.9
RACE	Mixed	324	0.4	7.6	1.9	0.1	1.4
RACE	Asian/Asian British: Indian	324	0.1	28.3	2	0.2	3.6
RACE	Asian/Asian British: Pakistani	324	0	20.4	1.4	0.2	2.9
RACE	Asian/Asian British: Bangladeshi	324	0	32	0.6	0.1	2.1
RACE	Asian/Asian British: Chinese	324	0.1	3.6	0.6	0	0.6
RACE	Asian/Asian British: Other Asian	324	0.1	11.3	1.2	0.1	1.7
RACE	Asians	324	0.4	43.5	5.7	0.4	7.7
RACE	Black African	324	0	16.4	1.3	0.1	2.4
RACE	Black Caribbean	324	0	11.2	0.7	0.1	1.5
RACE	Black Other Black	324	0	4.8	0.3	0	0.7
RACE	Blacks	324	0	27.2	2.3	0.2	4.4
RACE	Others	324	0	11.1	0.7	0.1	1.2
RACE	Country of birth:UK	324	44.9	97.7	88.5	0.6	10.4
RACE	Country of Birth: EU	324	57.6	98.9	92.6	0.4	7.8
RACE	Country of Birth:Other countries	324	1.1	42.4	7.4	0.4	7.8

RACE	All people aged 16 and over in household have English as a main language (English or Welsh in Wales)	324	52.6	98.9	92.7	0.5	8.1
RACE	At least one but not all people aged 16 and over in household have English as a main language (English or Welsh in Wales)	324	0.6	18	3.2	0.2	3.2
RACE	No people aged 16 and over in household but at least one person aged 3 to 15 has English as a main language (English or Welsh in Wales)	324	0	5.1	0.6	0	0.8
RACE	No people in household have English as a main language (English or Welsh in Wales)	324	0.3	24.3	3.5	0.2	4.2
RACE	Born in the UK	324	44.9	97.8	88.5	0.6	10.4
RACE	Resident in UK: Less than 2 years	324	0.1	10.3	1.4	0.1	1.6
RACE	Resident in UK: 2 years or more but less than 5 years	324	0.2	9.8	1.8	0.1	1.8
RACE	Resident in UK: 5 years or more but less than 10 years	324	0.3	11.9	2.3	0.1	2.2
RACE	Resident in UK: 10 years or more	324	1.3	29.3	5.9	0.3	5.2
OCCUPATION	1. Managers, directors and senior officials	324	6.6	23.3	11.2	0.1	2.4
OCCUPATION	2. Professional occupations	324	9	38	16.9	0.2	4.4
OCCUPATION	3. Associate professional and technical occupations	324	6.9	24.9	12.6	0.1	2.7
OCCUPATION	4. Administrative and secretarial occupations	324	7.7	17.6	11.4	0.1	1.5
OCCUPATION	5. Skilled trades occupations	324	3.6	21.6	11.9	0.1	2.6
OCCUPATION	6. Caring, leisure and other service occupations	324	5.4	13.5	9.5	0.1	1.4
OCCUPATION	7. Sales and customer service occupations	324	4.3	12.8	8.2	0.1	1.5
OCCUPATION	8. Process, plant and machine operatives	324	1.8	16.9	7.3	0.1	2.5
OCCUPATION	9. Elementary occupations	324	4.5	21.1	11	0.1	2.5

OCCUPATION	1. Higher managerial, administrative and professional occupations	324	4.4	22.3	10.6	0.2	3.6
OCCUPATION	1.1 Large employers and higher managerial and administrative occupations	324	0.9	6.1	2.5	0	0.8
OCCUPATION	1.2 Higher professional occupations	324	3.3	17.5	8.1	0.2	3
OCCUPATION	2. Lower managerial, administrative and professional occupations	324	12.9	32.4	21.4	0.2	3.4
OCCUPATION	3. Intermediate occupations	324	7.6	19.4	13	0.1	1.9
OCCUPATION	4. Small employers and own account workers	324	5	18	10	0.1	2.4
OCCUPATION	5. Lower supervisory and technical occupations	324	2.9	11.5	7.1	0.1	1.4
OCCUPATION	6. Semi-routine occupations	324	6.4	22.2	14.1	0.2	2.8
OCCUPATION	7. Routine occupations	324	4	22.4	11	0.2	3.3
OCCUPATION	L15 Full-time students	324	4.2	27.7	8	0.2	3.7
OCCUPATION	Economically active: Employee: Part-time	324	6.8	17.1	14.1	0.1	1.6
OCCUPATION	Economically active: Employee: Full-time	324	29.3	50.9	38.9	0.2	4
OCCUPATION	Economically active: Self-employed	324	5.1	17.4	10.3	0.1	2.6
RELIGION	Christian	324	27.1	80.9	61.4	0.5	8.1
RELIGION	Buddhist	324	0.1	3.3	0.4	0	0.3
RELIGION	Hindu	324	0	25.3	1.2	0.1	2.4
RELIGION	Jewish	324	0	15.2	0.4	0.1	1.3
RELIGION	Muslim	324	0.1	34.5	3.3	0.3	5.5
RELIGION	Sikh	324	0	10.6	0.6	0.1	1.5
RELIGION	Other religion	324	0.1	2.5	0.4	0	0.2
RELIGION	No religion	324	9.5	42.5	25.1	0.3	5.1
RELIGION	Religion not stated	324	5.4	20.5	7.2	0.1	1.3
EDUCATION	No qualifications	324	10.1	35.2	22.3	0.3	4.9
EDUCATION	Highest level of qualification: Level 1 qualifications	324	5.8	18.8	13.6	0.1	2.1

EDUCATION	Highest level of qualification: Level 2 qualifications	324	7.8	18.6	15.7	0.1	2
EDUCATION	Highest level of qualification: Apprenticeship	324	0.7	9.2	3.8	0.1	1.1
EDUCATION	Highest level of qualification: Level 3 qualifications	324	9.2	18.6	12.2	0.1	1.6
EDUCATION	Highest level of qualification: Level 4 qualifications and above	324	14.2	53.6	27.1	0.4	7.6
EDUCATION	Highest level of qualification: Other qualifications	324	3.4	15.7	5.4	0.1	2.1
SOCIOECONOMIC STATUS	Annual survey of hours and earnings - resident analysis	324	19254	41815	26805.4	236.7	4260.2
SOCIOECONOMIC STATUS	Average Job Seekers Jan - Dec 2011	324	1.5	11.8	4.1	0.1	2
SOCIOECONOMIC STATUS	5yr Average Job Seekers Jan07 - Dec 2012	324	1.2	9.6	3.5	0.1	1.7
SOCIOECONOMIC STATUS	8. Never worked and long-term unemployed	324	1.8	14.3	4.8	0.1	2.3
SOCIOECONOMIC STATUS	L14.1 Never worked	324	1.1	11.8	3.2	0.1	1.9
SOCIOECONOMIC STATUS	L14.2 Long-term unemployed	324	0.7	3.7	1.6	0	0.5
SOCIOECONOMIC STATUS	Not classified	324	4.2	27.7	8	0.2	3.7
SOCIOECONOMIC STATUS	Economically active: Unemployed	324	2	8	4	0.1	1.2
SOCIOECONOMIC STATUS	Unemployed: Never worked	324	0.1	2	0.6	0	0.3
SOCIOECONOMIC STATUS	Long-term unemployed	324	0.7	3.7	1.6	0	0.5
SOCIOECONOMIC STATUS	No adults in employment in household: With dependent children	324	1.3	10.4	3.7	0.1	1.6
SOCIOECONOMIC STATUS	GDHI per Head -2011	324	10310	47603	17825.7	239.4	4308.9
SOCIAL CAPITAL	Percentage of respondents who successfully obtained an NHS appointment in the last two years (2011)	324	86.6	99.1	94.6	0.1	2.3
SOCIAL CAPITAL	Proportion of respondents who tried to get an NHS dental appointment in the last two years (2011)	324	38.5	76.5	59.5	0.4	6.9
SOCIAL CAPITAL	2014-17 average - Percentage of respondents who successfully obtained an NHS	324	88.4	98.9	95	0.1	1.9

	appointment in the last two years						
SOCIAL CAPITAL	2014-17 av - Proportion of respondents who tried to get an NHS dental appointment in the last two years	324	38.2	74.5	59.3	0.4	6.4
SOCIAL CAPITAL	2016 Percentage of respondents who successfully obtained an NHS appointment in the last two years	324	86.5	99.1	94.8	0.1	2.1
SOCIAL CAPITAL	2016 Proportion of respondents who tried to get an NHS dental appointment in the last two years	324	37.2	74.4	58.6	0.4	6.4
SOCIAL CAPITAL	One family only: Married or same-sex civil partnership couple: Dependent children	324	8.3	23.5	15.7	0.2	2.8
SOCIAL CAPITAL	One family only: Cohabiting couple: Dependent children	324	1.1	6.1	4.1	0	0.8
SOCIAL CAPITAL	One family only: Lone parent: Dependent children	324	3.9	14.4	6.6	0.1	1.7
SOCIAL CAPITAL	Other household types: With dependent children	324	1.3	10.5	2.4	0.1	1.2
SOCIAL CAPITAL	Married	324	24.8	59.2	48.4	0.4	6.3
SOCIAL CAPITAL	One person in household with a long-term health problem or disability: With dependent children	324	2.6	7.5	4.4	0	0.8
SOCIAL CAPITAL	Lone parent in part-time employment: Total	324	22	49.6	35.5	0.3	4.7
SOCIAL CAPITAL	Lone parent in full-time employment: Total	324	15.7	42.7	27.6	0.3	4.5
SOCIAL CAPITAL	Lone parent not in employment: Total	324	20.1	62.3	37	0.4	7.3
SOCIAL CAPITAL	Day-to-day activities limited a lot: Age 16 to 64	324	1.6	7.2	3.4	0.1	1.1
SOCIAL CAPITAL	Day-to-day activities limited a little: Age 16 to 64	324	2.9	6.4	4.5	0	0.7
SOCIAL CAPITAL	Day-to-day activities not limited: Age 16 to 64	324	46.9	68.5	56	0.2	3.6

SOCIAL CAPITAL	Very good health	324	38.2	57.8	47.1	0.2	3.7
SOCIAL CAPITAL	Good health	324	28.5	37.8	34.5	0.1	1.4
SOCIAL CAPITAL	Bad health	324	2.1	7.3	4.1	0.1	1.1
SOCIAL CAPITAL	Very bad health	324	0.6	2.2	1.2	0	0.3
SOCIAL CAPITAL	Provides no unpaid care	324	86.7	93.5	89.5	0.1	1.3
SOCIAL CAPITAL	Provides 1 to 19 hours unpaid care a week	324	4.3	9.2	6.8	0.1	0.9
SOCIAL CAPITAL	Provides 20 to 49 hours unpaid care a week	324	0.8	2.2	1.3	0	0.3
SOCIAL CAPITAL	Provides 50 or more hours unpaid care a week	324	1.2	4.1	2.4	0	0.6
SOCIAL CAPITAL	Medical and care establishment: NHS: General hospital	324	0	7.2	0.2	0	0.7
SOCIAL CAPITAL	Medical and care establishment: Registered Social Landlord/Housing Association	324	0	9.4	0.8	0.1	1.2
PLUS - Access to unhealthy diets	NCMP - Obesity 2008/09 to 2010/11	324	5.1	14.1	9.2	0.1	1.6
PLUS - Access to unhealthy diets	NCMP - Obesity 2009/10 to 2011/12	324	5.3	14.1	9.1	0.1	1.6
PLUS - Access to unhealthy diets	NCMP - Obesity 2010/11 to 2012/13	324	5.2	13.5	9	0.1	1.6
PLUS - Access to unhealthy diets	NCMP - Obesity 2011/12 to 2013/14	324	5.3	13.6	9	0.1	1.6
PLUS - Access to unhealthy diets	NCMP - Obesity 2011 average	324	5.6	13.7	9.1	0.1	1.6

Appendix 45: Ranking of caries prevalence by local authority

Local Authority	Region	Water Fluoridation	Caries Prevalence
Pendle	North West	No	49.4
Rochdale	North West	No	47.1
Burnley	North West	No	46.5
Hyndburn	North West	No	45.8
Salford	North West	No	44.6
Manchester	North West	No	43.0
Blackburn with Darwen	North West	No	42.6
Knowsley	North West	No	42.3
Slough	South East	No	41.5
Boston	East Midlands	No	41.0
Bradford	Yorkshire and the Humber	No	39.8
Harrow	London	No	39.6
Preston	North West	No	39.6
Leicester	East Midlands	No	38.7
St. Helens	North West	No	38.2
Allerdale	North West	Yes	37.9
Bolton	North West	No	37.8
Luton	East of England	No	37.6
Wigan	North West	No	37.6
Chorley	North West	No	36.4
Barrow-in-Furness	North West	No	36.1
Bury	North West	No	35.2
Copeland	North West	Yes	35.1
Bolsover	East Midlands	Yes	34.9
Oldham	North West	No	34.8
Torbay	South West	No	34.7
Liverpool	North West	No	34.6
Brent	London	No	34.6
Corby	East Midlands	No	34.6
Barnsley	Yorkshire and the Humber	No	34.5
Tameside	North West	No	34.1
Rotherham	Yorkshire and the Humber	No	33.0
Waltham Forest	London	No	32.9
Kingston upon Hull, City of	Yorkshire and the Humber	No	32.8
Stoke-on-Trent	West Midlands	No	32.6
East Hampshire	South East	No	32.6
Kirklees	Yorkshire and the Humber	No	32.5
Hillingdon	London	No	32.5
Peterborough	East of England	No	32.4
Middlesbrough	North East	No	32.1
Bedford	East of England	No	31.3
Wirral	North West	No	31.2

Leeds	Yorkshire and the Humber	No	31.1
Tower Hamlets	London	No	31.1
Coventry	West Midlands	Yes	30.7
Ealing	London	No	30.7
Enfield	London	No	30.5
Herefordshire, County of	West Midlands	No	30.5
Doncaster	Yorkshire and the Humber	No	30.4
Halton	North West	No	30.4
Westminster	London	No	30.3
Camden	London	No	30.0
Worcester	West Midlands	No	29.9
Oadby and Wigston	East Midlands	No	29.8
Gloucester	South West	No	29.8
Sefton	North West	No	29.6
South Holland	East Midlands	No	29.6
Rossendale	North West	No	29.5
Southampton	South East	No	29.4
Scarborough	Yorkshire and the Humber	No	29.4
Calderdale	Yorkshire and the Humber	No	29.3
Selby	Yorkshire and the Humber	No	29.3
Wyre Forest	West Midlands	No	29.3
Telford and Wrekin	West Midlands	No	29.1
Newham	London	No	29.0
Haringey	London	No	29.0
Lancaster	North West	No	28.9
Wakefield	Yorkshire and the Humber	No	28.8
Sheffield	Yorkshire and the Humber	No	28.6
Barking and Dagenham	London	No	28.6
West Lancashire	North West	No	28.6
Croydon	London	No	28.5
Wolverhampton	West Midlands	Yes	28.4
Sunderland	North East	No	28.4
Gravesham	South East	No	28.3
Nuneaton and Bedworth	West Midlands	Yes	28.2
Wellingborough	East Midlands	No	28.0
South Ribble	North West	No	27.5
Wycombe	South East	No	27.3
Stockport	North West	No	27.2
East Lindsey	East Midlands	No	26.8
Kensington and Chelsea	London	No	26.6
Darlington	North East	No	26.4
Watford	East of England	No	26.2
Birmingham	West Midlands	Yes	26.1
Nottingham	East Midlands	No	25.9

Bournemouth	South West	No	25.9
Wandsworth	London	No	25.8
County Durham	North East	No	25.8
Bath and North East Somerset	South West	No	25.8
Hounslow	London	No	25.7
Sutton	London	No	25.6
Weymouth and Portland	South West	No	25.6
Maidstone	South East	No	25.5
High Peak	East Midlands	No	25.5
Sandwell	West Midlands	Yes	25.4
Northampton	East Midlands	No	25.4
North West Leicestershire	East Midlands	No	25.2
Stevenage	East of England	No	25.2
Ashfield	East Midlands	Yes	25.1
Redcar and Cleveland	North East	No	24.9
Blackpool	North West	No	24.9
Adur	South East	No	24.9
Erewash	East Midlands	No	24.8
Kettering	East Midlands	No	24.7
Wyre	North West	No	24.7
Chesterfield	East Midlands	No	24.6
Hammersmith and Fulham	London	No	24.2
Barnet	London	No	24.1
Forest Heath	East of England	No	24.0
Derby	East Midlands	No	24.0
Mansfield	East Midlands	Yes	24.0
Eden	North West	No	23.9
Carlisle	North West	No	23.6
Oxford	South East	No	23.5
Walsall	West Midlands	Yes	23.4
Teignbridge	South West	No	23.3
Chichester	South East	No	23.3
Staffordshire Moorlands	West Midlands	No	23.2
Gateshead	North East	No	23.2
North East Lincolnshire	Yorkshire and the Humber	No	22.9
Hackney (including City of London)	London	No	22.9
East Riding of Yorkshire	Yorkshire and the Humber	No	22.9
Dudley	West Midlands	Yes	22.6
Northumberland	North East	No	22.6
Merton	London	No	22.5
Ipswich	East of England	No	22.5
Bristol, City of	South West	No	22.5
Islington	London	No	22.5
Hambleton	Yorkshire and the Humber	No	22.5

Great Yarmouth	East of England	No	22.4
Newark and Sherwood	East Midlands	No	22.3
Richmondshire	Yorkshire and the Humber	No	22.3
Greenwich	London	No	22.2
North Lincolnshire	Yorkshire and the Humber	Yes	22.2
Reading	South East	No	22.2
Hastings	South East	No	22.2
Charnwood	East Midlands	No	22.1
Wiltshire	South West	No	22.1
Gedling	East Midlands	No	22.0
Rugby	West Midlands	Yes	21.9
Exeter	South West	No	21.9
North Devon	South West	No	21.8
Poole	South West	No	21.8
Lambeth	London	No	21.7
South Tyneside	North East	No	21.7
Forest of Dean	South West	No	21.7
Melton	East Midlands	No	21.7
Medway	South East	No	21.7
Ribble Valley	North West	No	21.7
Blaby	East Midlands	No	21.6
Tewkesbury	South West	No	21.6
Tendring	East of England	No	21.5
Dartford	South East	No	21.5
Kingston upon Thames	London	No	21.5
Fylde	North West	No	21.5
Plymouth	South West	No	21.4
Craven	Yorkshire and the Humber	No	21.4
Milton Keynes	South East	No	21.3
North Dorset	South West	No	21.2
Redbridge	London	No	20.9
Daventry	East Midlands	No	20.8
Cheshire West and Chester	North West	No	20.7
South Kesteven	East Midlands	Yes	20.7
Mendip	South West	No	20.7
North Warwickshire	West Midlands	Yes	20.7
Stockton-on-Tees	North East	No	20.6
West Somerset	South West	No	20.6
Thurrock	East of England	No	20.5
Arun	South East	No	20.5
Havering	London	No	20.5
Hartlepool	North East	No	20.5
Fenland	East of England	No	20.4
Redditch	West Midlands	Yes	20.3

Warrington	North West	No	20.3
South Somerset	South West	No	20.3
Wychavon	West Midlands	Yes	20.2
Stafford	West Midlands	No	20.2
Colchester	East of England	No	20.1
Bassetlaw	East Midlands	Yes	20.0
North Tyneside	North East	Yes	20.0
Portsmouth	South East	No	20.0
Newcastle-under-Lyme	West Midlands	No	19.9
Torridge	South West	No	19.8
Vale of White Horse	South East	No	19.7
King's Lynn and West Norfolk	East of England	No	19.7
Christchurch	South West	No	19.7
Cheshire East	North West	No	19.6
Southend-on-Sea	East of England	No	19.6
Cherwell	South East	No	19.6
Lewisham	London	No	19.4
Hinckley and Bosworth	East Midlands	No	19.4
Newcastle upon Tyne	North East	Yes	19.3
Lincoln	East Midlands	Yes	19.3
Malvern Hills	West Midlands	No	19.3
Rushmoor	South East	No	19.2
East Northamptonshire	East Midlands	No	19.1
Warwick	West Midlands	Yes	19.1
Harrogate	Yorkshire and the Humber	No	19.1
Cornwall (including Isles of Scilly)	South West	No	19.1
Trafford	North West	No	19.0
Shropshire	West Midlands	No	18.8
Ryedale	Yorkshire and the Humber	No	18.6
Tunbridge Wells	South East	No	18.5
Sedgemoor	South West	No	18.4
Cheltenham	South West	No	18.3
Isle of Wight	South East	No	18.2
Purbeck	South West	No	18.1
Crawley	South East	No	18.0
Taunton Deane	South West	No	18.0
South Gloucestershire	South West	No	17.8
West Lindsey	East Midlands	Yes	17.7
Central Bedfordshire	East of England	No	17.7
Norwich	East of England	No	17.7
Basildon	East of England	No	17.7
West Oxfordshire	South East	No	17.6
Sevenoaks	South East	No	17.5
Brighton and Hove	South East	No	17.4

Bromley	London	No	17.4
Stratford-on-Avon	West Midlands	Yes	17.4
West Dorset	South West	No	17.4
Reigate and Banstead	South East	No	17.4
Swindon	South West	No	17.3
West Berkshire	South East	No	17.3
Runnymede	South East	No	17.1
Brentwood	East of England	No	17.1
Harborough	East Midlands	No	16.6
Broxbourne	East of England	No	16.5
Mid Devon	South West	No	16.5
Swale	South East	No	16.4
Richmond upon Thames	London	No	16.4
Woking	South East	No	16.3
Solihull	West Midlands	Yes	16.3
East Staffordshire	West Midlands	Yes	16.2
Hertsmere	East of England	No	16.2
Suffolk Coastal	East of England	No	16.2
Rushcliffe	East Midlands	No	15.9
Southwark	London	No	15.9
York	Yorkshire and the Humber	No	15.9
Spelthorne	South East	No	15.8
South Bucks	South East	No	15.7
Eastbourne	South East	No	15.7
Three Rivers	East of England	No	15.6
Rutland	East Midlands	No	15.6
Castle Point	East of England	No	15.4
South Oxfordshire	South East	No	15.3
Waveney	East of England	No	15.2
Mid Suffolk	East of England	No	15.1
Bracknell Forest	South East	No	15.0
Windsor and Maidenhead	South East	No	14.8
North Somerset	South West	No	14.8
Amber Valley	East Midlands	No	14.7
Huntingdonshire	East of England	No	14.6
Cannock Chase	West Midlands	Yes	14.5
Bexley	London	No	14.4
Hart	South East	No	14.4
Tandridge	South East	No	14.2
South Lakeland	North West	No	14.2
Stroud	South West	No	14.2
Braintree	East of England	No	14.1
Surrey Heath	South East	No	14.1
North East Derbyshire	East Midlands	No	14.0

East Dorset	South West	No	13.9
St Edmundsbury	East of England	No	13.8
Breckland	East of England	No	13.6
Chelmsford	East of England	No	13.5
Worthing	South East	No	13.4
South Derbyshire	East Midlands	No	13.4
Canterbury	South East	No	13.4
Welwyn Hatfield	East of England	No	13.4
Dacorum	East of England	No	13.3
North Norfolk	East of England	No	13.2
Rother	South East	No	13.2
Broxtowe	East Midlands	No	13.2
East Devon	South West	No	13.1
Aylesbury Vale	South East	No	13.0
Havant	South East	No	12.7
Winchester	South East	No	12.6
Tamworth	West Midlands	Yes	12.4
Cambridge	East of England	No	12.1
Babergh	East of England	No	12.0
West Devon	South West	No	12.0
Elmbridge	South East	No	11.8
Epsom and Ewell	South East	No	11.7
Dover	South East	No	11.7
East Cambridgeshire	East of England	No	11.5
Lichfield	West Midlands	Yes	11.4
Maldon	East of England	No	11.4
Cotswold	South West	No	11.4
New Forest	South East	No	11.4
Bromsgrove	West Midlands	Yes	11.4
North Hertfordshire	East of England	No	11.4
Broadland	East of England	No	11.2
South Hams	South West	No	11.1
South Staffordshire	West Midlands	Yes	11.1
Fareham	South East	No	11.0
Shepway	South East	No	10.9
Harlow	East of England	No	10.9
Derbyshire Dales	East Midlands	No	10.8
Wokingham	South East	No	10.8
Epping Forest	East of England	No	10.8
Mid Sussex	South East	No	10.6
Tonbridge and Malling	South East	No	10.4
South Northamptonshire	East Midlands	No	10.4
North Kesteven	East Midlands	Yes	10.1
East Hertfordshire	East of England	No	10.1

St Albans	East of England	No	9.9
Ashford	South East	No	9.8
Rochford	East of England	No	9.7
Lewes	South East	No	9.5
Gosport	South East	No	9.5
South Norfolk	East of England	No	9.5
Thanet	South East	No	9.3
Chiltern	South East	No	9.0
Eastleigh	South East	No	8.7
Wealden	South East	No	8.3
South Cambridgeshire	East of England	No	8.1
Mole Valley	South East	No	8.0
Test Valley	South East	No	7.9
Uttlesford	East of England	No	7.4
Basingstoke and Deane	South East	No	5.7
Guildford	South East	No	5.5
Waverley	South East	No	5.1
Horsham	South East	No	4.4

Appendix 46: Table demonstrating predictability of caries risk model

Rsquared	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Partial Least Squares	0.44	0.65	0.71	0.70	0.78	0.88
Random Forest	0.15	0.50	0.61	0.59	0.69	0.83
Gradient Boosting Machine	0.22	0.53	0.62	0.59	0.68	0.85
Linear Regression	0.34	0.65	0.73	0.71	0.79	0.87
Stepwise Regression	0.24	0.63	0.71	0.70	0.78	0.89
CART	0.10	0.36	0.43	0.43	0.51	0.69
Boostrap Aggregation (Bagging)	0.18	0.48	0.58	0.56	0.64	0.84
Support Vector Machine	0.31	0.59	0.67	0.64	0.73	0.83
Extreme Gradient Boosting	0.23	0.48	0.60	0.58	0.69	0.86
Penalised: Ridge Regression	0.32	0.66	0.74	0.71	0.80	0.87

Mean Absolute Error

	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Partial Least Squares	2.50	3.15	3.48	3.50	3.86	4.94
Random Forest	2.77	3.81	4.23	4.20	4.67	5.50
Gradient Boosting Machine	2.76	3.69	4.05	4.16	4.66	5.68
Linear Regression	2.22	3.04	3.44	3.42	3.75	4.76
Stepwise Regression	2.41	3.17	3.46	3.52	3.95	4.69
CART	3.38	4.59	4.95	4.95	5.24	6.27
Boostrap Aggregation (Bagging)	2.62	3.98	4.35	4.37	4.70	6.30
Support Vector Machine	2.51	3.46	3.84	3.84	4.18	5.60
Extreme Gradient Boosting	2.74	3.84	4.23	4.19	4.55	5.71
Penalised: Ridge Regression	2.34	3.05	3.41	3.42	3.75	4.79

Root Mean Square Error

	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Partial Least Squares	3.12	3.96	4.39	4.46	4.92	6.37
Random Forest	3.53	4.67	5.28	5.30	5.96	7.12
Gradient Boosting Machine	3.73	4.59	5.10	5.28	5.96	7.23
Linear Regression	2.78	3.96	4.30	4.39	4.72	6.36
Stepwise Regression	3.34	4.06	4.34	4.51	5.01	6.68
CART	4.37	5.76	6.28	6.29	6.71	9.11
Boostrap Aggregation (Bagging)	3.08	4.85	5.29	5.47	6.03	8.23
Support Vector Machine	3.30	4.34	4.84	4.89	5.25	7.57
Extreme Gradient Boosting	3.41	4.78	5.16	5.30	5.79	7.68
Penalised: Ridge Regression	3.09	3.85	4.29	4.39	4.84	6.12

Appendix 47: Qualifications Levels in England, Wales and Northern Ireland

Level 1	FIRST CERTIFICATE
	GCSE - GRADES 3, 2, 1 OR GRADES D, E, F, G
	LEVEL 1 AWARD
	LEVEL 1 CERTIFICATE
	LEVEL 1 DIPLOMA
	LEVEL 1 ESOL
	LEVEL 1 ESSENTIAL SKILLS
	LEVEL 1 FUNCTIONAL SKILLS
	LEVEL 1 NATIONAL VOCATIONAL QUALIFICATION (NVQ)
	MUSIC GRADES 1, 2 AND 3
Level 2	CSE - GRADE 1
	GCSE - GRADES 9, 8, 7, 6, 5, 4 OR GRADES A*, A, B, C
	INTERMEDIATE APPRENTICESHIP
	LEVEL 2 AWARD
	LEVEL 2 CERTIFICATE
	LEVEL 2 DIPLOMA
	LEVEL 2 ESOL
	LEVEL 2 ESSENTIAL SKILLS
	LEVEL 2 FUNCTIONAL SKILLS
	LEVEL 2 NATIONAL CERTIFICATE
	LEVEL 2 NATIONAL DIPLOMA
	LEVEL 2 NVQ
	MUSIC GRADES 4 AND 5
	O LEVEL - GRADE A, B OR C
Level 3	A LEVEL
	ACCESS TO HIGHER EDUCATION DIPLOMA
	ADVANCED APPRENTICESHIP
	APPLIED GENERAL
	AS LEVEL
	INTERNATIONAL BACCALAUREATE DIPLOMA
	LEVEL 3 AWARD
	LEVEL 3 CERTIFICATE
	LEVEL 3 DIPLOMA
	LEVEL 3 ESOL
	LEVEL 3 NATIONAL CERTIFICATE
	LEVEL 3 NATIONAL DIPLOMA
	LEVEL 3 NVQ
	MUSIC GRADES 6, 7 AND 8
	TECH LEVEL
Level 4	CERTIFICATE OF HIGHER EDUCATION (CERTHE)
	HIGHER APPRENTICESHIP
	HIGHER NATIONAL CERTIFICATE (HNC)
	LEVEL 4 AWARD
	LEVEL 4 CERTIFICATE

	LEVEL 4 DIPLOMA
	LEVEL 4 NVQ
Level 5	DIPLOMA OF HIGHER EDUCATION (DIPHE)
	FOUNDATION DEGREE
	HIGHER NATIONAL DIPLOMA (HND)
	LEVEL 5 AWARD
	LEVEL 5 CERTIFICATE
	LEVEL 5 DIPLOMA
	LEVEL 5 NVQ
Level 6	DEGREE APPRENTICESHIP
	DEGREE WITH HONOURS - FOR EXAMPLE BACHELOR OF THE ARTS (BA) HONS, BACHELOR OF SCIENCE (BSC) HONS
	GRADUATE CERTIFICATE
	GRADUATE DIPLOMA
	LEVEL 6 AWARD
	LEVEL 6 CERTIFICATE
	LEVEL 6 DIPLOMA
	LEVEL 6 NVQ
	ORDINARY DEGREE WITHOUT HONOURS
Level 7	INTEGRATED MASTER'S DEGREE, FOR EXAMPLE MASTER OF ENGINEERING (MENG)
	LEVEL 7 AWARD
	LEVEL 7 CERTIFICATE
	LEVEL 7 DIPLOMA
	LEVEL 7 NVQ
	MASTER'S DEGREE, FOR EXAMPLE MASTER OF ARTS (MA), MASTER OF SCIENCE (MSC)
	POSTGRADUATE CERTIFICATE
	POSTGRADUATE CERTIFICATE IN EDUCATION (PGCE)
	POSTGRADUATE DIPLOMA
Level 8	DOCTORATE, FOR EXAMPLE DOCTOR OF PHILOSOPHY (PHD OR DPHIL)
	LEVEL 8 AWARD
	LEVEL 8 CERTIFICATE
	LEVEL 8 DIPLOMA

Appendix 48: Mapping of CDHS variables to conceptual model

SDH from systematic review	**Variable in CDHS 2013**
Access to care	Ever had difficulty finding NHS dentist to treat child
Age child start tooth brushing	Age in years when started brushing teeth
Area-Deprivation	Deprivation quintiles (home postcode)
Child living with family	Relationship of responding adult to child
Gender	Gender
Parental Education	Highest educational qualification of responding adult
Employment Status	Employment status
Employment type	NSSEC category analytical classes
Maternal age at child birth	Age in years of responding adult
Parent's Oral Health Behaviour	Responding adult's usual dental attendance
Race/Ethnicity	Ethnicity of child
Single Parenthood	Living with partner
Socioeconomic Status	Free school meal eligibility
Support of routine oral hygiene	Who usually brushes child's teeth
Urban-Rural	Urban-Rural
Water Fluoridation	-
Child's birth order	-
Household family size	-
Family Caries History	-
Parental Smoking History	-
Access to dental insurance	-
Mode of delivery	-
Prematurity	-
Birth Weight	-