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Can clinicians and scientists explain and prevent unexplained underperformance syndrome in elite athletes: an interdisciplinary perspective and 2016 update

Nathan A Lewis,1,2,3 Dave Collins,4,5 Charles R Pedlar,1,3 John P Rogers1,6

ABSTRACT

The coach and interdisciplinary sports science and medicine team strive to continually progress the athlete’s performance year on year. In structuring training programmes, coaches and scientists plan distinct periods of progressive overload coupled with recovery for anticipated performances to be delivered on fixed dates of competition in the calendar year. Peaking at major championships is a challenge, and training capacity highly individualised, with fine margins between the training dose necessary for adaptation and that which elicits maladaptation at the elite level. As such, optimising adaptation is key to effective preparation. Notably, however, many factors (eg, health, nutrition, sleep, training experience, psychosocial factors) play an essential part in moderating the processes of adaptation to exercise and environmental stressors, for example, heat, altitude; processes which can often fail or be limited. In the UK, the term unexplained underperformance syndrome (UUPS) has been adopted, in contrast to the more common referenced term overtraining syndrome (OTS). In the UK, the term unexplained underperformance syndrome (UUPS) has been adopted, in contrast to the more common referenced term overtraining syndrome (OTS). The use of the term overtraining syndrome (OTS), rather than UUPS, can be unhelpful when engaging with the athlete and coach. OTS can lead both to misapprehend training as the sole cause and the problem (which in itself can encourage blame), thus diverting attention and focus from what are often the key issue/s.

INTRODUCTION

Over the past decade an expert group from Europe and the USA has provided joint consensus statements on the topic of overtraining syndrome (OTS).1 2 In the UK, the term unexplained underperformance syndrome (UUPS) has been adopted.3 4 For the purpose of this review, UUPS will be used throughout as opposed to OTS. The term UUPS reflects the complex nature of the condition, the multifactorial aetiology, and that an imbalance between training load and recovery may not be the primary cause for underperformance. UUPS draws on the distinction that a decline in performance represents the universal feature, and that an imbalance between training load and recovery may not be the primary cause for underperformance. UUPS draws on the distinction that a decline in performance represents the universal feature and a prerequisite for the diagnosis of UUPS is an unexpected decline in performance2 7 despite a period
of extended rest, causation requires a broader perspective.

The wide variation in symptoms on presentation adds to the complexity of the condition, with no absolute consistencies, other than a heightened state of fatigue and increased perception of effort with training. Mood disturbances, decreased motivation, weight loss, loss of appetite, increased incidence of infection and sleep disturbances are all variously reported components of the condition.1 2 8-10 As part of the diagnostic work up, significant organic disease must be excluded (eg, cardiac or endocrine diseases), along with infectious diseases, allergies, eating disorders, depression and nutritional deficiencies such as energy, carbohydrate/protein intake, iron, vitamin D and magnesium.2-4 10 Indeed, the diagnosis of UUPS can only conclusively be made retrospectively, once the recovery time is known. Moreover, expert groups have stated that the symptoms and physiological responses are highly variable and in many instances, individual.2 5 10 The latter point emphasises the importance of longitudinal monitoring of individual athletes, thereby increasing the potential for early identification and signs of maladaptation. In fact, the individual presentation of the condition emphasises the challenges faced by researchers using conventional statistical approaches to identify significant differences between UUPS versus healthy athletes for a given physiological variable. In elites, an informed critical or biochemical variable. In elites, an informed...

Consequently, we propose that UUPS can only be prevented and causative factors identified, and thus understood and countered this condition.1 2 8-10 As part of the diagnostic work up, significant organic disease must be excluded (eg, cardiac or endocrine diseases), along with infectious diseases, allergies, eating disorders, depression and nutritional deficiencies such as energy, carbohydrate/protein intake, iron, vitamin D and magnesium.2-4 10 Indeed, the diagnosis of UUPS can only conclusively be made retrospectively, once the recovery time is known. Moreover, expert groups have stated that the symptoms and physiological responses are highly variable and in many instances, individual.2 5 10 The latter point emphasises the importance of longitudinal monitoring of individual athletes, thereby increasing the potential for early identification and signs of maladaptation. In fact, the individual presentation of the condition emphasises the challenges faced by researchers using conventional statistical approaches to identify significant differences between UUPS versus healthy athletes for a given physiological variable. In elites, an informed critical or biochemical variable. In elites, an informed critical or biochemical variable. In elite athletes, the prevalence of UUPS/OTS may be high,1 2 5 6 particularly if assessing across athletic careers. In elite middle distance runners, Morgan et al.8 found that 64% of male athletes and 60% of female athletes reported experiencing UUPS/OTS at some point in their athletic careers, while a recent study across 26 sports including 129 elite Swiss athletes estimated a career prevalence rate of 30%.6

A 2016 review of this topic from the point of view of the applied practitioner and clinician is both warranted and timely, building on the comprehensive consensus statements of Meeusen et al.5 because it is both realistic and practical to prevent the development of UUPS. Indeed, only the implementation of well-structured longitudinal monitoring programmes encompassing biomarkers, physiological and physical measures, in tandem with wellness and readiness to train tools that draw on an integrated interdisciplinary approach to data interpretation and athlete management can achieve this. Consequently, we propose that UUPS can only be prevented and causative factors identified, and thus understood and countered this condition.1 2 8-10 As part of the diagnostic work up, significant organic disease must be excluded (eg, cardiac or endocrine diseases), along with infectious diseases, allergies, eating disorders, depression and nutritional deficiencies such as energy, carbohydrate/protein intake, iron, vitamin D and magnesium.2-4 10 Indeed, the diagnosis of UUPS can only conclusively be made retrospectively, once the recovery time is known. Moreover, expert groups have stated that the symptoms and physiological responses are highly variable and in many instances, individual.2 5 10 The latter point emphasises the importance of longitudinal monitoring of individual athletes, thereby increasing the potential for early identification and signs of maladaptation. In fact, the individual presentation of the condition emphasises the challenges faced by researchers using conventional statistical approaches to identify significant differences between UUPS versus healthy athletes for a given physiological variable. In elites, an informed critical or biochemical variable. In elite athletes, the prevalence of UUPS/OTS may be high,1 2 5 6 particularly if assessing across athletic careers. In elite middle distance runners, Morgan et al.8 found that 64% of male athletes and 60% of female athletes reported experiencing UUPS/OTS at some point in their athletic careers, while a recent study across 26 sports including 129 elite Swiss athletes estimated a career prevalence rate of 30%.6

A sport physician’s perspective

Reduced performance and fatigue is a common presentation for sports physicians, particularly when working with endurance athletes.12 Notably, however, it can be diagnostically challenging to distinguish between the normal physiological spectrum of fatigue/functional over-reaching (OR) in an athlete with high training volumes, and the multitude of medical conditions that can present in a similar way. With persistent fatigue and underperformance in athletes lasting 2 weeks or more despite adequate rest and recovery, many different causes should be considered. Any significant medical problem can contribute to underperformance but the most common causes seen in a younger athletic population are outlined in box 1 along with less common causes in box 2.12

Once the underperformance has been established, athletes should be assessed by a sports physician in order to promptly diagnose and treat any underlying medical causes. Given the breadth of possibilities, the initial consultation may be lengthy, with many areas covered and initial investigations undertaken. There may be one specific trigger for fatigue and underperformance, but more

Box 1 Common causes for athlete underperformance

Musculoskeletal injuries, for example, trauma, tendinopathy, hamstring tears, bone stress

Physiological: functional over-reaching, jet lag, insufficient sleep, pregnancy, heat, cold, altitude, dehydration

Infection, for example, upper and lower respiratory tract infections, flu-like illness, traveller’s diarrhoea/gastroenteritis, glandular fever (Epstein-Barr virus)

Asthma

Allergies/hay fever

Nutritional deficiencies: protein, carbohydrate, minerals (eg, iron, magnesium), vitamins (eg, D), Relative Energy Deficiency in Sport20 21

Iron deficiency (+/- anaemia), for example, coeliac disease, vegetarians, altitude, menorrhagia, non-steroidal anti-inflammatory drug abuse

Concussion

Stress, anxiety, depression, eating disorders

Non-functional over-reaching and unexplained underperformance syndrome
commonly, there is a combination of several contributing factors, for example, high training volumes with inadequate recovery; environmental conditions (altitude, heat); external stresses related to work, studies or relationships; recent illness or viral infection; nutritional deficits. Clearly, significant medical problems need to be excluded through a detailed history with systematic questioning (box 3), a targeted physical examination (box 4) and appropriate baseline (box 5) and targeted investigations at the clinician’s discretion (box 6).

If significant medical causes have been excluded, a diagnosis of UUPS can be made if the athlete continues to underperform in conjunction with fatigue despite recovery and rest. There are often identifiable medical causes and thus the underperformance is explainable and treatable, and a diagnosis of UUPS is not appropriate. To complicate matters, in many cases, there are confounding factors such as recent viral illness, psychosocial factors, nutritional deficiencies, unfamiliar environmental conditions and sudden increases in training that contribute to the development of UUPS; part of the management will involve addressing these underlying issues. Accordingly, athletes with UUPS should be referred for assessment by a suitable qualified sports nutritionist/dietitian and an exercise physiologist.

**Box 3** What to address within the athlete’s history

<table>
<thead>
<tr>
<th>Topic</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>Onset; duration; severity; possible triggers; recent travel</td>
</tr>
<tr>
<td>Underperformance</td>
<td>Quantify degree and duration of impairment</td>
</tr>
<tr>
<td>Recent training</td>
<td>Review any physiology data available; for example, training heart rate</td>
</tr>
<tr>
<td>Training</td>
<td>Type(s); volume; frequency; intensity; location; monotony; goals;</td>
</tr>
<tr>
<td>Sleep</td>
<td>Quantity; quality; insomnia; daytime naps</td>
</tr>
<tr>
<td>Psychological screen</td>
<td>Presence of mood disturbances and the duration; Stress; anxiety;</td>
</tr>
<tr>
<td>Medical history</td>
<td>Injuries; illness; surgery</td>
</tr>
<tr>
<td>Menstrual history</td>
<td>Menarche; contraception; last period; frequency of periods;</td>
</tr>
<tr>
<td>Drug history</td>
<td>Presence of menorrhagia</td>
</tr>
<tr>
<td>Nutrition screen</td>
<td>Typical daily food; fluid intake; timing; changes; dietary exclusions;</td>
</tr>
<tr>
<td>Family history</td>
<td>Sudden cardiac death; atopy; autoimmune or endocrine disorders</td>
</tr>
<tr>
<td>Social history</td>
<td>Work; examinations; financial pressures; support structure around</td>
</tr>
<tr>
<td>Systematic questioning</td>
<td>Cardiovascular, respiratory, gastrointestinal, sexual health,</td>
</tr>
<tr>
<td>Specifically on fever</td>
<td>Cough; wheeze; sore throat; swollen glands; muscle pain; postural</td>
</tr>
<tr>
<td>Blood count</td>
<td>White cell count; glucose; protein; C reactive protein</td>
</tr>
<tr>
<td>Vitamin D (D2 and D3)</td>
<td>Biochemistry; renal function and electrolytes; liver function tests;</td>
</tr>
<tr>
<td>Thyroid function tests</td>
<td>Red blood cell magnesium</td>
</tr>
<tr>
<td>Urine dipstick</td>
<td>Specific gravity; glucose; blood; protein; leucocytes</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
</tr>
</tbody>
</table>

**Box 4** Physical examination to be completed as part of the athlete consultation

<table>
<thead>
<tr>
<th>Topic</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>Body mass index; temperature; jaundice; anaemia; lymphadenopathy;</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>Heart rate and rhythm; blood pressure (lying and standing); heart sounds</td>
</tr>
<tr>
<td>Respiratory system</td>
<td>Respiratory rate; auscultation; peak flow rate; oxygen saturations</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Abdomen such as tenderness; masses; hepatosplenomegaly</td>
</tr>
<tr>
<td>Neurovascular</td>
<td>Muscle tone and strength; reflexes; sensation upper and lower limbs</td>
</tr>
<tr>
<td>Other targeted examination</td>
<td>As directed by the history</td>
</tr>
</tbody>
</table>

**Box 5** Routine baseline investigations for the athlete with underperformance and fatigue

<table>
<thead>
<tr>
<th>Topic</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full blood count</td>
<td>Differential white cell count</td>
</tr>
<tr>
<td>Serum ferritin</td>
<td>Vitamin D (D2 and D3)</td>
</tr>
<tr>
<td>Blood profile</td>
<td>Biochemistry; renal function and electrolytes; creatine kinase; glucose;</td>
</tr>
<tr>
<td>Thyroid function tests</td>
<td>C reactive protein</td>
</tr>
<tr>
<td>Red blood cell magnesium</td>
<td></td>
</tr>
<tr>
<td>Epstein-Barr virus serology</td>
<td></td>
</tr>
<tr>
<td>Urine dipstick</td>
<td>Specific gravity; glucose; blood; protein; leucocytes</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
</tr>
</tbody>
</table>
Where there are underlying psychological issues (and this should be the default rather than the exception position), the athlete should be referred early to a sport psychologist or other mental health professional for further assessment and treatment. After this return to training and competition, should be guided by the interdisciplinary team, with clear communication with the athlete and their coach on time scales, volume, intensity and frequency of training. Regular review and feedback using a variety of longitudinal monitoring tools is ideal in the progression back to full training.15 14

A performance nutrition scientist’s perspective

Meeusen et al2 highlight a number of nutritional considerations in the prevention of UUPS giving priority to the importance of carbohydrate, energy and fluid intake for the prevention of glycogen depletion, a negative energy balance (EB) and dehydration.2 They address the importance of adjusting carbohydrate intake for periods of intensified training to prevent glycogen depletion and the resulting metabolic, endocrine, mood and performance disturbances that can accrue and are observed in over-reached states.15 16 However, there are other nutritional factors which warrant consideration in the prevention and management of UUPS.

For example, a negative EB is deemed to be a risk factor for UUPS. However, it is important to recognise that a negative EB per se is not detrimental to adaption and performance in athletes, although it is likely to increase fatigue and ratings of perceived exertion (RPE). Nor is it necessary for the athlete to be in a state of EB for improvements in athletic performance.2 17–19 Athletes will go through training periods with reduced energy availability (EA) and negative EB by a process of periodisation, with the manipulation of macronutrient intake catered for in order to optimise body composition and performance. Significantly, however, a sustained chronic lack of sufficient EA leads to a decline in bioenergetic hormones, poor recovery and maladaptation, placing the athlete at risk of both injury and UUPS.2 17 20 21 For example, Vanheest et al5 directly linked EA and performance in elite athletes. The athletes in whom performance declined across the 12 weeks by ~8% were in a negative EB for the entire 12-week period (low EA; 10–12 kcal/kg fat-free mass (FFM)). In contrast, the athletes in which performance improved by ~8% after 12 weeks, experienced ~8–10 weeks of a negative EB, and were in EB at weeks 2 and 4 only (sufficient EA; 30–37 kcal/kg FFM). It should also be noted that performance was only adversely affected in the low EA group after 6 weeks of sustained low EA. Critically, the training volume and intensity were increased after week 4. This unique study highlights the importance of sufficient EA and periodised nutrition around intensified training periods to facilitate recovery and adaptation, and the prevention of underperformance. Moreover, a sustained chronic period of low EA is sufficient to induce a state of explained underperformance in endurance athletes, and highlights the importance of well-structured and designed monitoring programmes in athletes to prevent unexplained underperformance (UUPS). It is our experience that the use of bioenergetics hormones (eg, free triiodothyronine) can aid the prescription of nutritional support around the training response (stimulus and recovery) in elite healthy athletes.

In this regard, there is increasing interest surrounding the athlete exercising with low carbohydrate availability on muscle physiology and indeed performance, referred to as train low, or fasted training and of late, sleep low.22 23 The latter approach may confer a greater risk for UUPS given the crucial role for sleep in athletic performance,24 dietary carbohydrates for sleep onset25 and calorie intake for sleep quality.24 However, increasing dietary protein may serve to attenuate the risk, given the capacity for protein to enhance sleep quality,24 and recovery.26 Although there are physiological benefits to the athlete performing structured training sessions in the presence of low carbohydrate availability,27–32 there are risks associated with such practices. Indeed, such sessions should be planned in to the athletes training cycle along with appropriately structured monitoring (physiological, biochemical, hormonal, immunological, psychological, performance), and be relevant to the athlete’s physiological development (eg, training age), training goals and performance requirements.32 The practitioner, or athlete self-imposing a restricted carbohydrate diet without monitoring and guidance, with a view to reducing fat mass or enhancing muscle oxidative capacity, for example, may cause chronic low EA and this carries a high risk of maladaptation, infection, reduced performance and UUPS.2 17 20 Exercising with low carbohydrate availability will increase inflammatory cytokine release,33 and it has been proposed that excessive cytokine release and inflammatory state during and post-exercise may lead to UUPS.34 The cytokine,

**Box 6** Further investigations to consider in cases of unexplained underperformance syndrome (UUPS)

| Infection: malaria screen, viral studies, for example, cytomegalovirus serology, hepatitis, HIV, lyme disease, stool samples for microscopy, culture, sensitivity, ova, cysts and parasites |
| Endocrine: Glycated haemoglobin, hormone studies, for example, follicle-stimulating hormone, luteinising hormone, oestadiol, free and total testosterone, serum hormone-binding globulin, prolactin, cortisol, ACTH/synacthen test, parathyroid hormone, triiodothyronine |
| Nutrition: vitamin B12, red cell folate, coeliac screen (if iron deficient), trace elements (ie, zinc, copper) |
| Chest X-ray, spirometry, allergy testing |
| Cardiopulmonary exercise testing and echocardiogram |
| Specialist referral where indicated, for example, endocrinology |

If UUPS diagnosed, then a comprehensive nutritional and physiology assessment by sports nutritionist and physiologist

interleukin-6 (IL-6), previously referred to as a ‘fatigoe- 
gen’, increases under conditions of low carbohydrate 
availability.33 35 Indeed, IL-6 has been implicated in 
fatigue and UUPS.33 with elevated concentrations 
reported to impair exercise performance.36 
Furthermore, altered cytokine responses to exercise (eg, 
elevated IL-6) have been observed in illness prone ath-
letes,37 and athletes presenting with UUPS frequently 
report infective illnesses.34 Nutritional support for 
the immune system should be prioritised through intensified 
training and competition periods, especially in those 
athletes with a history, or at an increased risk of UUPS, 
for example, known psychological stress, illness prone 
athletes and junior athletes. It is beyond the scope of 
this paper to address the immune support of the elite 
athlete, which is comprehensively reviewed elsewhere.38 
Finally, exercising with low muscle glycogen availability 
increases skeletal muscle breakdown, amino acid oxidation, 
sweat and urine nitrogen losses39 40 and lowers 
plasma glutamine concentrations41 the latter an observ-
ation reported in chronically fatigued elite athletes.42 43 

Adequate dietary protein is integral for health, 
immunity, adaptation and athletic performance.26 44 45 
with inadequate dietary protein reported in chronically 
fatigued athletes44 and significant alterations in plasma 
amino acids observed in fatigued versus healthy athletes, 
that is, glutamine and glutamate.42 43 46–49 In addition, 
increased dietary protein (3 g/kg/day) intake during a 
period of overload training in endurance athletes has 
been shown to attenuate the decline in performance 
and increase in symptoms of stress.36 Furthermore, 
increasing dietary protein (3 g/kg/day) during a 2-week 
block of high-intensity training in cyclists restored the 
exercise-induced leucocyte trafficking impairment, with 
fewer symptoms of upper respiratory tract infections.50 

In accordance with the aforementioned studies in 
endurance training models of OR, additional protein in 
the form of amino acids served to attenuate decrements 
in performance and increases in muscle damage 
observed in high volume resistance training OR.51 
Periodised adjustments to the athlete’s protein intake 
may serve to modify the impact of intensified periods of 
training on measures of fatigue and immunity, and 
enhance performance outcomes. Indeed, it should be 
emphasised that the periodisation of nutritional 
support, consisting of adjustments to energy and macro-
nutrients (eg, carbohydrate and protein) and micronu-
trient intakes around training phases (eg, iron, 
magnesium, vitamin D), and most crucially intensified 
training blocks, are a critical element of the successful 
optimisation of performance, and prevention of UUPS. 

Inflammation, oxidative stress (OS) and alterations in 
redox homoeostasis (ARH) have been implicated in the 
pathophysiology of OTS,52–54 with ARH reported in ath-
letes diagnosed with OTS.35 In chronic fatigue syndrome 
(CFS; a conditioned likened to OTS), OS is associated 
with the degree of fatigue at rest and postexercise,35–37 
with several studies demonstrating increased OS in CFS 
versus healthy controls.56 58–62 CFS can be said to repre-
sent the extreme end of the OTS continuum. In well-
trained athletes, the short-term (2 weeks) restriction of 
fruit and vegetable intake (ie, low antioxidant diet), to 
just one-two servings per day, results in increased OS, 
exercise RPE and inflammation, albeit with no change in 
performance.63 64 However, the lack of a significant 
change in performance may merely reflect the short-
term nature of the studies. Polyphenols, constituents of 
fruit and vegetables, have been shown to attenuate OS 
in athletes and reduce the inflammatory response to 
exercise, including IL-6, improve sleep duration and 
quality, and enhance skeletal muscle regeneration.65–69 

It is interesting to speculate that a chronic lack of suffi-
cient dietary fruits and vegetables (recognising the vast 
array of different chemicals present70) may lead to 
slower rates of skeletal muscle regeneration in the 
athlete, thus placing the athlete at greater risk of UUPS. 
It is known that the skeletal muscles of healthy endur-
ance athletes display evidence of structural damage, 
while those endurance athletes diagnosed with acquired 
training intolerance (ie, UUPS) display significantly 
greater evidence still of skeletal muscle pathology.71 

Vitamin E (α-tocopherol) is integral to skeletal muscle 
membrane repair72 with serum vitamin E inversely asso-
ciated with muscle pain thresholds generated via elec-
trical stimulation and fatigue in CFS and healthy 
controls.55 Low dietary intakes of vitamin E are reported 
in some athletes73 74 but not others,75 with increased 
postexercise red blood cell haemolysis observed in 
female athletes with low fat intakes and reduced serum 
vitamin E concentrations.74 A relationship with red 

blood cell fragility and serum vitamin E was also 
reported in elite male swimmers.76 Increased skeletal 
muscle uptake of vitamin E and the carotenoid 
β-carotene occurs with exercise,77 and there is evidence 
for specific training methodologies (ie, 18 days of 
live-high-train-low) impacting negatively on elite athletes 
antioxidant status (eg, serum vitamin E and carotenoids) 
in some studies,78 79 but not others;80 for a review see 
Lewis et al.81 

Environmental factors (eg, overseas training camp, 
altitude, heat stress) may influence the risk of UUPS 
through (1) enforced dietary changes coupled with 
added physiological stress such as increased training 
volume or competition; recognising short-term dietary 
omissions influence recovery and fatigue, and (2) com-
bination of heat stress, dehydration and high-intensity 
exercise resulting in mucosal injury, gut permeability, 
and increased bacterial and inflammatory challenge and 
OS.82 83 Notably, oxidants are implicated in 
ischaemia-induced gut injury and associated bacterial 
translocation, with antioxidants (ie, ascorbic acid) pre-
venting exercise-induced endotoxaemia resulting from 
maximal exercise.83 Through our own observations 
(unpublished data), biomarkers of redox status should 
be assessed in athletes with UUPS. Clearly, the micronu-
trient status of an athlete will deteriorate with a
reduction in fruits, vegetables or the omission of any food group. Nutritional deficiencies are a cause of fatigue and should be considered as part of the examination of the athlete presenting with fatigue and UUPS. To our knowledge, no published data exist on the micronutrient status of athletes diagnosed with UUPS.

To summarise, the quality of the diet is important for short-term and long-term health of the athlete, with a diet lacking in the basic components comprising recovery and constituting an increased risk for UUPS, particularly when significant ‘stressors’ are present or increased. A failure to appropriately periodise and individualise the nutritional support, and monitor the athlete effectively throughout the season further increases the risk of UUPS. Finally, it is noteworthy that young athletes with a history of UUPS report loss of appetite with periods of heavy training; emphasising the importance of periodised nutrition plans, monitoring, education and guidance in prevention.

An exercise physiologist’s perspective

Typically, the literature around the physiology of OR, NFOR and OTS/UUPS begins with an explanation of the stress-recovery-adaptation curve, first described in the seminal work of Seyle, with many variations on this fundamental principle since then. Meeusen et al bring together a summary of aspects of the literature, system by system, alluding to the fact that the stress-recovery-adaptation curve is too general and the diagnosis of UUPS requires a process of exclusion, beginning in the clinic and extending to the sports science team to take into account non-clinical aspects including training volume, EB and nutrition, recovery habits, and psychology.

A major ‘input’ into the athlete’s capacity to recover and adapt is the dose of training, taking into account intensity, duration and frequency. Anecdotally, high performance athletes with a detailed and individualised training plan and regular indicators of progress to inform the training and recovery process may be protected to some degree from UUPS. Unpublished data from British Cycling (Leeder) indicates that UUPS is generally absent from the GB Olympic squad athletes in recent years (presented at the British Association of Sport and Exercise Medicine Spring Conference 2014: http://blogs.bmj.com/bjsm/2014/04/16/the-fatigued-athlete-and-red-s-lessons-from-the-field-and-the-basem-spring-conference-2/); however, those without the support of an effective sports science and medicine team are vulnerable.

Meeusen’s position stand recognises that the intensification of training is often concurrent with the development of fatigue. The contrasting metabolism of low-intensity vs high-intensity exercise is fundamental to this. It is well described that fat oxidation rates peak at about 60% to 65% of maximal oxygen consumption (VO2max) and once blood lactate is accumulating, fat metabolism dramatically diminishes and may be negligible above 90% of VO2max in favour of carbohydrate to fuel a high ATP turnover and maintain workload. This has significant implications for glycogen use, recovery duration and, therefore, EB. Furthermore, training at altitude or in the heat will further increase glycogen turnover and decrease exercise efficiency, as will training in a fatigued or depleted state. Accordingly, consideration should be given to the contrasting metabolic demands of training sessions when designing a training programme. Assisting coaches and athletes to progress training appropriately to avoid UUPS by managing periods of OR is a key role of the sports physiologist.

Training and redox homoeostasis may be an important feature of the stress-adaptation response through the process of hormesis, although again few data relating to redox homoeostasis exist in elite athletes with UUPS. It is possible that there is an optimum ‘dose’ of exercise and associated production of reactive oxygen and nitrogen species that results in adaptation (see Vollard et al). A recent study of OR over 6 weeks demonstrated an increase in OS with cumulative over-load, apparently overwhelming antioxidant defence system by week 6. Additionally, in functionally over-reached state (F-OR), a taper will result in supercompensation and an expected level of performance.

UUPS also has implications for the taper. In an elegant study, Aubry et al demonstrated that athletes in a F-OR, performed worse after a 2-week taper than a similar group defined as acutely fatigued from training. This highlights the importance of training management, and adds weight to the notion that 2 weeks should be adequate for a positive improvement in performance. Even in the F-OR group, there was a small increase in performance with a taper that would be absent in UUPS.

The assessment of heart rate variability may be an attractive option for preventing UUPS in endurance athletes; however, the data in this area are sparse. A recent study of trained triathletes demonstrated that in F-OR, a progressive tendency towards parasympathetic modulation of heart rate was evident. The study concluded that the heart rate variability should be assessed on a daily basis to account for noise in the signal. Together with many other considerations, the appropriate management and monitoring of workload and recovery is fundamental to the avoidance of UUPS. Various software exist that track training volume, and there are examples that use algorithms to yield an index of overall recovery; however, to our knowledge, none of these have been validated. However, a battery of monitoring tools could be effective in avoiding UUPS, including the measurement of physiological and psychological variables, together with logs of dietary and training practices.

It is recognised that key to the training and adaptation cycle is energy homoeostasis. Indeed the IOC has recently introduced a new term: Relative Energy Deficiency in Sport (RED-S) that implicates energy...
deficiency in a host of pathologies, underperformance problems and conditions, including the Female Athlete Triad, but not restricted to female athletes. Further, RED-S is implicated in a range of symptoms associated with the diagnosis of UUPS including decreased endurance performance and decreased training response. Energy homeostasis can be assessed globally through changes in body weight and body composition; however, biomarkers of energy homeostasis may also be of use including hormones, peptides and cytokines, comprehensively reviewed by Jürimäe et al.93

The other major component of the stress-recovery-adaptation is of course recovery and sports physiologists have strived to offer techniques of promoting or enhancing recovery in athletes, including (in addition to nutritional interventions described above) compression,94 cold water immersion,95 and sleep.24 Evidence that athletes with UUPS are failing to achieve sufficient sleep (quality and quantity) is absent from the literature. However, sleep remains a cause for concern, particularly given the clear and growing association between sleep deprivation and a range of negative associations including compromised immune function,96 compromised daytime performance;97 and generally lower quality and quantity of sleep in elite athletes compared with healthy non-athlete controls.98 Furthermore, one recent study using wristwatch actigraphy assessed sleep in the F-OR athlete, observing a significant decrease in sleep duration, sleep efficiency and immobile time during sleep (−7.9%, −1.6% and −7.6%, respectively,99), implying that chronic sleep deprivation could be a critical factor in the aetiology of UUPS.

A sports psychologist’s perspective

Building from Meeusen et al.,2 and from a psychological perspective, the first thing to notice is the recovery time factor which seems to be inherent in distinguishing between F-OR, NFOR and OTS; most particularly the stagnation factor associated with NFOR and therefore, presumably, OTS. Indeed, this led Meeusen and colleagues to use the term ‘prolonged maladaptation’ as a distinguishing characteristic between these various forms. For practitioners, this emphasises the importance of athlete perception as an important discriminating factor, perhaps even more important than actual physical training load (cf. Birrer et al.), between who does or does not suffer with these problems. Certainly, in the absence of the clear test for UUPS which has been suggested, perception seems to be one of several factors which, rather than discriminating by degree, may be either cause or symptom, depending on the individual.

The other factor worthy of mention from Meeusen et al. is the coaction and impact of psychology, over and above the well-examined mood state profiling which has dominated the literature. Meeusen et al.8,9 provide a good coverage of this literature, together with the pitfalls of mood as an OTS indicator (eg, need for consistent protocols, faking by athletes, consistency of self-report, etc). What is less apparent, however, is the effect that this state (however protracted) will have on other markers, including hormonal levels, performance tests, etc. As such, psychology is clearly more than just mood and has a plethora of interactive effects; indeed, it is this that underpins the promise of perceived measures (eg, RPE, muscle soreness100 101) as preventative tests.

Indeed, recent work (eg, Cook and Beaven102) supports this contention on the role of perception as a cause of incidence or prevention, particularly on the delicate edge where the system can go either way. In any case, it seems fair to suggest that perception of the challenge and how close it is to your limits is an important concomitant which practitioners should monitor and, where possible, employ. Other studies have highlighted psychological traits (eg, optimism,103 self-determined motivation104) and states (eg, the switch in perception to external factors when fatigued,104 self-esteem105), which also seem to mediate the impact of physical challenges.

Certainly, as our understanding of stress extends from the homeostasis position of Selye towards the new allostatic perspective (eg, McEwen106), that the importance of such complex interactions between psychological and physiological factors becomes clear as they can account for the varied responses which characterise UUPS and similar conditions.107 Certainly, the integrated model suggested by Grove et al.108 in dance provides a good basis for applied work across other performance domains. A list of presetting conditions (both environmental and personality), antecedents and a mechanistic interaction of factors provides the structure by which practitioners can address the issue. In this regard, it is worth highlighting that non-training factors seem to have a clear association with UUPS, albeit (unsurprisingly) only in some cases.109 The ideas presented in this model on entrapment and restraining factors (relating, perhaps to a perceived lack of control) also hold some promise although, clearly and commonly, more research is needed. In simple terms, we know lots but discerning the exact blend to apply to any individual case remains a complex challenge,4 despite high-quality medical research, which is uncovering some of the mechanisms that seem to occur in some of the cases.110

In fact, the clearest and most significant point is the highly individualised nature of these complaints. In short, and as with so many human factors,111 a particular intra-individual blend of biopsychosocial elements is associated with the susceptibility to, and incidence of, UUPS.6 This inherent variability may well explain the use of OTS as a ‘diagnostic dustbin’ and the extreme variation in precursors and outcomes seem, to us, a likely part of the reconsideration of OTS as UUPS.3 4

Thus, these varied precursors, concomitants and outcomes point to the need for interdisciplinary and personalised support. Perhaps the best preventative strategy lies with coaches and support staff, who have good knowledge of the athletes, employing a variety of
different and personalised markers, as is now often the case with daily monitoring for training readiness.\(^{48,112}\)

In simple terms, the initial use of a broad testing spectrum, followed by a personalised refinement based on which markers appear most reactive to each individual, would seem the best general rule for progress. Further reflecting the psychological dimension, a focus on athlete perception as a sound general approach is to be encouraged; ensuring that athletes know what, why and when they are doing things and that they have confidence in the plan. Increased coach–athlete discussion, surely a sensible procedure across the development spectrum, is a major tool in this approach.

Finally, it is of utmost importance to ‘know your athlete!’ What s/he thinks of the specific training, why s/he thinks that and, more broadly, their general disposition (eg, optimistic/pessimistic, high/low trait anxious, etc). The bottom line being, changing perceptions (although often easier said than done) can have a significant effect.

**CONCLUSION**

The coach and interdisciplinary team strive to continually progress the athlete’s performance year on year. In structuring the annual training programme, coaches and scientists are planning distinct periods of progressive overload coupled with recovery for anticipated performances to be delivered on fixed dates of competition in the calendar year, for example, World Championships and Olympic Games. Should the devised period of overload training be too great for a given athlete leading into a precompetition tape,\(^9\) or tapering insufficient for super compensation, then the window of opportunity to perform optimally has passed, or worse, a period of UUPS ensues. Peaking at major championships is a challenge, and training capacity is highly individual, with fine margins between the training dose necessary for adaptation and that which elicits maladaptation at the elite level. Thus, monitoring of the athlete’s progression through periods of physical preparation (ie, microcycles and macrocycles) is crucial to understanding whether s/he is on plan (ie, performing and fatigued states managed). Ensuring that training and environmental stresses are appropriate is also crucial, recognising these will change year on year. Indeed, across the training cycle, there will be periods when the athletes training load, nutritional and psychological support require necessary adjustment (ie, optimisation) to stay on track. Such an approach requires a multidisciplinary structured monitoring programme providing knowledge of which variables to monitor (biochemical, psychological, physiological and non-exhausting physical tests, eg, five-bound test,\(^{113}\)), and when to test the athlete and capture data (eg, point in the training cycle; at rest; during or after controlled exercise; in the field or laboratory), thus directing the scientist and practitioner as to when to intervene to stay on plan. An additional benefit to regular monitoring is an increase in communications between the athlete, coach and sports science team, with the data driving purposeful conversations. That said, although a proactive and preventative approach is clearly favoured and feasible with monitoring and collection of athlete data, a retrospective analysis can still inform future athlete management and refinement of the monitoring programme can be used to explain episodes of underperformance. Furthermore, obtaining a well-controlled baseline for all monitoring variables is recommended, supporting the establishment of individual athlete reference data and quantification of intrindividual variation and meaningful changes. Finally, a spectrum of monitoring variables are necessary in the athletes programme, on the basis that no single variable has proven effective at identifying UUPS in all studies\(^2,14\) (ie, athletes and sports), with the utility of certain monitoring variables resulting from differences in (but not exclusive to) biochemical individuality, gender, sport, training age, training status, and the reliability and reproducibility of the analytical approaches.

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BMJ Open Sport Exerc Med 2015 1:
doi: 10.1136/bmjsem-2015-000063

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