A Cumulative Stress and Training Continuum Model: A Multidisciplinary Approach to Unexplained Underperformance Syndrome

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This article provides an understanding of the unexplained underperformance syndrome (UPS; Budgett, Newsholme, Lehmann, et al. 1999) and the problems surrounding the current presentation of it. This was done in terms of processes and outcomes. Recommendations for a more stringent usage of terms was provided. Based on Selye’s (1956) General Adaptation Theory and a model described by Kenttä and Hassmén (1998; 2002), a Cumulative Stress and Training Continuum Model was put forward that incorporates both the continuum paradigm and the supercompensation cycle model of training. These models, combined with a multidisciplinary approach, provide a useful framework for understanding the processes and outcomes of athletic performance in general and the unexplained underperformance syndrome in particular. The Cumulative Stress and Training Continuum Model emphasizes the complex interactions between the many factors affecting UPS and their weblike, cumulative, and synergistic relationships. Such an approach assists in explaining how seemingly nonsignificant factors in their own right could exert disproportionate importance and effect. Also, it emphasizes the individual variability and susceptibility to UPS as well as the fluctuations within a given individual. The unique contribution of the Cumulative Stress and Training Continuum Model put forward in the present article is that it is a truly inclusive and holistic model for addressing UPS.

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Introduction

One way in which athletes have tried to improve their performances is by engagement in more intensified physical conditioning programs. Such intensified training is not without risk, the outcome of training stress being positive or negative depending on the athlete’s ability to adapt to the imposed training load. The inability to deal with training stress has resulted in performance decrements among athletes of all descriptions and disciplines. Although this state has been given different names in the literature, in the present context it will be referred to as unexplained underperformance syndrome (UPS).

UPS has recently been defined by Budgett et al. (1999) as “a persistent unexplained performance deficit (recognised and agreed by coach and athlete) despite two weeks of relative rest” (p. 231).

The scientific literature relating to UPS is vast and covers a wide range of aspects: physiological, biochemical, haematological, immunological, nutritional, and psychosocial. Unfortunately, a significant amount of confusion surrounds the UPS phenomenon due to the failure to adopt any form of internationally agreed on or standardized terminology and definitions (Budget et al. 1999; Kellman 2002). The aims of this article are threefold: first, to provide an understanding of UPS and the problems surrounding its current presentation; secondly, the problems of terminology will be addressed and recommendations for a more stringent usage of terms will be provided; finally, a cumulative stress and training continuum model, which incorporates both the continuum paradigm and the supercompensation cycle model of training, will be presented. These models combined with a multidisciplinary approach provide a useful framework for understanding the processes, outcomes of athletic performance in general, and UPS in particular.

Terminology Issues

An examination of the literature indicates that UPS has been described by a variety of different terms. For example, burnout and staleness have been classified as being the same as UPS, while chronic fatigue syndrome was introduced as being the term used in American literature (Budget 1990; Owen 1994; Raglin 1993). The plethora of terms, many of which are often used synonymously, has contributed significantly to the confusion and uncertainty surrounding this subject. In particular the interchangeable usage of the terms overtraining and overreaching has been a frequent source of confusion.
The Processes

Budgett (1990, 1994) and others have made a clear distinction between the two processes of overreaching and overtraining. Overreaching is defined as “the normal process of hard training, which, when combined with adequate rest enables athletes to reach their full potential” (Budgett 1994, p. 65). Overtraining, on the other hand, was defined as “the process of excessive training, which, when combined with inadequate rest, leads to the pathological underperformance syndrome” (Budgett 1994, p. 65). Overreaching and overtraining, in this view, are the consequence of different processes with distinct outcomes. Such an interpretation is compatible with the supercompensation cycle model of training adaptation (Viru 2001). The supercompensation model relates successive periods of training and recovery, over a time scale, which involve the application of a training stimulus (stress) designed to cause a disturbed homeostasis combined with optimal recovery conditions, which will result in positive training adaptation or supercompensation (Bompa 1999; Viru 2001). Thus, inappropriate training loads and recovery periods both independently and in conjunction with each other can lead to performance decrements. It also considers and integrates nontraining stressors and thus is compatible with multidisciplinary models. Adopting this model, overreaching is synonymous with an optimal level of training overload that results in positive training adaptations.

Kuipers (1998), however, referred to overreaching as “the first phase of overtraining or incomplete recovery, and which is quickly reversible” (p. 1138). He continued by suggesting that overreaching is occasionally induced deliberately in an attempt to obtain an increased supercompensation. Furthermore, a gradual transition from overreaching to UPS or staleness will take place. This introduces the concept of a continuum in both process and outcome. It also indicates the inability to maintain a standardized and stringent use of terminology.

A valuable insight into where much of the misunderstanding might lie was provided by Foster and Lehmann (1997). They divided the training process into four specific levels: undertraining, optimal training, overreaching, and overtraining. Thus a distinct differentiation was made between the level of training stimulus that would result in keeping fatigue at a moderate level and a training stimulus that would cause significant depression of performance capacity. However, the excessive level of training to which they refer is actually the product of an extended duration of engagement in an overreaching stimulus (a few days to a couple of weeks) set within cycles of regeneration, and to which they ascribed the title periodization. Periodization, therefore, would be the extensive or prolonged usage of an overreaching stimulus that results in very severe training accompanied by significant depression of performance capacity, and not the overreaching. The latter should simply be used to address the exercise intensity of an individual training session, and which if not used over an extensive period
of time would not result in the consequences described. This misunderstanding was further corroborated by Raglin (1993), who clearly defined the process of stressing the athlete to the point whereby incomplete recovery occurs between training sessions as overtraining, not overreaching, and the entire process of systematically increasing and decreasing training as periodization. A further complication was introduced by defining positive adaptation as being the result of overtraining, and maladaptation the result of overwork.

Lehmann, Foster, and Keul (1993) introduced the terms short-term overtraining (STOT) and long-term overtraining (LTOT). STOT was characterized by training fatigue, reduction or stagnation of performance capacity, transient incompetence, and usually lasting only a matter of days to 2 weeks. Having introduced this additional term to add further potential confusion, Lehman et al. (1993) suggested that overreaching might be a useful alternative to use in its place. It was stressed, however, that it should be differentiated from LTOT. STOT could progress smoothly to LTOT but, most importantly, that supercompensation appeared possible only after STOT and not LTOT. This is an indicator that Lehman et al. (1993) viewed STOT as similar to overreaching. A similar type of reasoning was adopted by Kenttä and Hassmén (1998; 2002). They introduced the terms positive overtraining and negative overtraining to describe the processes. It was argued that these could be differentiated by the outcomes of the training process. We would argue that these authors were simply referring to overreaching and overtraining, respectively.

Overreaching should only be used to describe training that produced short-term (a few days) symptoms of a disrupted homeostasis; in contrast, overtraining should be used to describe the process of excessive training by which the phenomenon of UPS occurs (Hooper and Mackinnon 1995). To complete the picture, Budgett (1994) also used the term undertraining. This refers to a level of training overload or stimulus that fails to exceed the current adaptation threshold to disrupt homeostasis and thus fails to facilitate supercompensation. The relationship of these different elements, in the context of the training process, is best depicted graphically (see Figure 1). The adoption of the proposed training cycle terminology by researchers could take away some of the confusion that currently exists within the literature.

The Outcomes

The outcomes attributed to UPS are abundant (see Table 1). Probably the most important outcome is fatigue. There is clear distinction between UPS fatigue and physiological fatigue. Training stress appears to be a major contributory factor to UPS fatigue. A distinction has also been made between sympathetic and parasympathetic outcomes of overtraining (Kellmann 2002; Kereszty 1971; Raglin 1993; see Table 1). It has been suggested that the sympathetic form is less frequent in modern-day sports (Lehmann, Lormes, Opitz-Gress, et al. 1997). Although
this is not the place to elaborate on this distinction, it should be stressed that, in the context of a training/stress continuum model, the differences observed in the symptoms of this subdivision may simply be differing temporal manifestations of the same condition. Alternatively, they reflect the variability of the individual response to this condition (Foster and Lehmann 1997; Raglin 1993).

As pointed out earlier, the terms staleness and burnout have often been used to describe UPS. However, both terms should actually be considered as distinct syndromes. Staleness is a psychobiological phenomenon in which physiological changes will have a profound effect on psychological aspects and vice versa. In general, staleness is seen as a precursor of burnout. Being a psychological term, in burnout the emotional and mental demands are well acknowledged (Maslach 1976; Smith 1986). Burnout, in this respect, has a gradual onset and consists of physical, emotional, and psychological components and is a result of a chronic imbalance between perceived demands and perceived response capabilities (stress). Symptoms identified from the adult and youth worlds of athletics as well as the helping professions and business included lack of energy, exhaustion, sleeplessness, depression, tension, irritability, anger, headaches, other physical ailments, decreased performance, a tendency to internalize all failures, disillusionment with sport, a loss of confidence, and withdrawal from participation (Cohn 1990; Feighley 1984; Freudenberger 1980; Henschen 1998; Pate, McClenaghan, and Rotella 1984; Smith 1986).

Karpovich (1965) linked fatigue to staleness and alluded to the concept of a continuum of outcomes. Boredom was presented as an early manifestation on this continuum and was seen as a feeling of disinclination to continue a given
Table 1
Characteristics of the Unexplained Underperformance Syndrome (UPS) and the Sympathetic and Parasympathetic Outcomes of Overtraining (Budgett et al. 1999; Kellmann 2002; Lehmann et al. 1998)

<table>
<thead>
<tr>
<th>UPS</th>
<th>Sympathetic</th>
<th>Parasympathetic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>Impaired performance</td>
<td>Impaired performance</td>
</tr>
<tr>
<td>Frequent minor infections</td>
<td>No supercompensation</td>
<td>No supercompensation</td>
</tr>
<tr>
<td>Unexplained/usually</td>
<td>Restlessness, irritability</td>
<td>Fatigue, apathy, depression</td>
</tr>
<tr>
<td>heavy, stiff and/or sore</td>
<td>Disturbed sleep</td>
<td>NOT sleep disturbed</td>
</tr>
<tr>
<td>muscles</td>
<td>Weight loss</td>
<td>Constant weight</td>
</tr>
<tr>
<td>Mood disturbance</td>
<td>Increased resting heart rate</td>
<td>Low resting heart rate</td>
</tr>
<tr>
<td>Change in expected sleep quality</td>
<td>Increased resting blood pressure</td>
<td>Low resting blood pressure</td>
</tr>
<tr>
<td>Loss of energy</td>
<td>Retarded recovery after exercise</td>
<td>Suppressed</td>
</tr>
<tr>
<td>Loss of competitive drive</td>
<td></td>
<td>heart rate exercise profile</td>
</tr>
<tr>
<td>Loss of libido</td>
<td></td>
<td>glucose exercise profile</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td></td>
<td>lactate exercise profile</td>
</tr>
<tr>
<td>Excessive sweating</td>
<td></td>
<td>Neuromuscular excitability</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catecholamine sensitivity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Altered hypthalomo, pituitary,</td>
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<td></td>
<td></td>
<td>adrenal function</td>
</tr>
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activity, resulting from the enforced participation in an activity—physically, mentally, or socially—without adequate motivation and, therefore, without any interest. This is an important concept as it provides a link with the view that boring or dull training is a potential causal factor in UPS. Karpovich (1965) also highlighted the role of unsympathetic coaches as a potential source of aggravation of the problem.

Hooper, Mackinnon, Howard, et al. (1995), while acknowledging the fact that the outcome of staleness was on a continuum, nevertheless failed to distinguish between staleness and burnout. Raglin (1993) even described an intermediate stage in the development of staleness, which was termed distress. This state was characterized as having several of the symptoms of staleness without a resultant performance decrement. It also has been suggested that UPS is closely related to Seyle’s third stage of exhaustion (Ryan 1983).

Kenttä and Hassmén (1998; 2002) have described this phenomenon as the overtraining–response continuum, and have provided a time frame of the processes and outcomes involved. They suggested that the overreached state and staleness were to be considered as appearing at the opposite ends of the continuum, but they used staleness and burnout as synonyms for UPS. Failure to recover within 72 hours after training was used as the criterion to denote short-term (acute), negative overtraining, resulting in the overreached state (distress). Staleness was considered a long-term effect (chronic), the product of more severe overtraining, and appears at the other end of the continuum (Kenttä and Hassmén 1998). The overreached state was selected as the starting point on the overtraining–response continuum because it is the point beyond which maladaptation occurs. Moreover, sympathetic staleness was viewed as an intermediate stage leading to parasympathetic staleness. An important contribution of the work of Kenttä and Hassmén (1998) was the recognition of the concepts of cumulative stressors and a stress threshold. The overtraining–response continuum can best be understood as being synonymous with UPS, and along which there are various identifiable stages, the boundaries between which are fluid and display individual variability. This concept is depicted graphically in Figure 2.

Models of Burnout

General Adaptation Theory was outlined as part of Selye’s (1965) discourse on the stress syndrome. It was hypothesized that stress affect individuals in both specific and general ways. Specific reactions are said to be different and dependent on the nature of the stressor, whereas general stress reactions are similar for all living organisms. The most salient feature of this model is the emphasis placed on the interaction of stressors and their cumulative and synergistic effects. Any specific stressors not only affect a given physiological system, they affect or interact with every other system. This is a central feature, which is missing in
some of the models that will be discussed, but which forms the core of the Cumulative Stress and Training Continuum Model proposed later.

The processes operative in this model (i.e., the resources for dealing with stress) are said to be dependent on a reservoir of general adaptation energy that is finite and determined by heredity. General adaptation energy could be depleted, or almost depleted, due to the excessive demands of a single activity, the combined effects of a number of stressors, or the failure of replenishment resulting from inadequate diet or rest. As such, general adaptation energy can be regarded as synonymous with an adaptation threshold, which when exceeded begins to produce the continuum of processes and outcomes depicted in Figures 1 and 2. It is, therefore, compatible with the supercompensation cycle model.

The model is consistent with observations of the pathogenesis of UPS in athletes, which is often seen to be triggered by sudden and dramatic increases in training volume/intensity or from similar increases in nontraining stressors. It thus can explain how previously tolerable levels of physical training for an individual can evolve to become intolerable, and also provides a rationale as to why principles of treatment would be successful. The importance of this model for the athletic

![Diagram of the overtraining response continuum or the overtraining syndrome.](image)

**Figure 2.** The overtraining response continuum or the overtraining syndrome.
setting is that it does not overemphasize the role of training, and by not doing so provides a framework for understanding the interactions and combined effects of stressors that contribute to UPS.

The Cognitive–Affective Stress Model (Smith 1986) provides a stress-based interpretation of burnout in sport which, it is hypothesized, occurs via a four stage process (initial situational demand placed on the athlete, cognitive appraisal demands, physiological response, and coping behavior). The model also proposes that personality and motivational factors influence or impact all four stages of the process, that the process is both circular and continuous, and that a reciprocal relationships exist between all four stages of the model. This model, first and foremost, is dependent on the cognitive appraisal being the spark that ignites or sets into motion the entire chain of events; without this, subsequent events cannot occur. This places, however, an unjustifiable emphasis on the contribution of this factor, which cannot be substantiated and which also contradicts the vast body of evidence supporting a much stronger contribution from physical or training stresses. This model, therefore, fails to accommodate the possibility of other factors as having this capacity to trigger events—and, as such, on this basis is flawed. Any model that seeks to represent and provide an adequate framework to understand UPS must incorporate and integrate the potential for various different factors to act as this trigger, either independently or in conjunction with others. The exact contributions of this trigger would be the result of the individual’s capacity for coping with the diverse stresses produced by those different factors and the interaction of these with the prevailing situational conditions at a given time.

The basis of the Negative Training Stress Response Model is the argument that training results in physical and psychological stresses, which can, in turn, result in both positive and negative effects (Silva 1990). This model incorporates the concept of the supercompensation cycle of training, the well-documented evidence for positive and negative training adaptations, and the previously described continua of both the processes and outcomes identified as being operative in the pathogenesis of UPS. A major criticism of this model is that it emphasizes the effects of training stressors to the exclusion of the possibility that the effects of nontraining or psychosocial stresses could provide the stimulus for the process. In this sense, therefore, and from a multidisciplinary paradigm, it fails to provide a complete model for understanding UPS per se, and also the interdependent nature of and potential synergistic effects of all of the potential stressors involved.

The Empowerment Model (Coakley 1992) regards stress as the symptom of burnout, and not the cause. It is proposed that the cause is linked to the social organization of high-performance sport and its effects on identity and control issues in young athletes. These are extremely powerful concepts that should certainly be incorporated into, or facilitated by, any model of burnout. Notwithstanding this, it is clear that the overwhelming evidence from the scientific literature does not support Coakley’s assertion that this is the primary cause of
UPS or burnout. At most, the model has identified significant and additional sources of stress.

In the Training/Recovery Model put forward by Kenttä and Hassmén (1998), the supercompensation principle is described as comprising two processes: breakdown (training) and recovery (rest). It is suggested that the quantity and quality of recovery are directly proportional to the intensity of the applied training stress and that full recovery is the essential prerequisite that allows supercompensation to occur. It is also proposed that, when breakdown and recovery are initiated by a single training bout (overload stimulus), they should be regarded as a training and recovery unit, of which the most important part is the recovery process. As such, it includes many of the salient features of both Selye’s (1965) model and the supercompensation cycle.

The major emphasis in the study by Kenttä and Hassmén (1998) was the introduction of the concept of measuring the recovery process, for which the authors correctly assert that there has been a lack of emphasis that has in turn resulted in a lack of available monitoring protocols. A key feature of this understanding is the recommendation that the optimum recovery process should be considered in a proactive role or context (i.e., that of preventing the syndrome from occurring) rather than reactive (i.e., in terms of rehabilitation). The authors correctly point out that there are significant benefits in prevention rather than cure. They also infer that monitoring both training and recovery provides the optimal method for tailoring the “perfect” training program.

While Kenttä and Hassmén’s (1998) model also acknowledges the contributions of both training and nontraining stressors to a cumulative level of stress resulting in UPS, it unfortunately lays claim to insufficient recovery after heavy physical training as being the main factor responsible for its cause. In this respect, it elevates the contribution of training stressors above that of nontraining stressors.

In the model by Kenttä and Hassmén (1998), the purpose of monitoring training and recovery is to enable the elite athlete to reach a balance in the zone where training yields optimal performance increments: the adaptation threshold. This adaptation threshold is directly related to three variables—recovery, stress, and capacity—and must be identified as it comprises a fundamental part of the monitoring of training and recovery. The evaluation of the effectiveness of training regimen must include the characteristics of the individual, the recovery process, and the psychosocial stressors. Significantly, a given training stimulus may have differential effects on an individual athlete’s performance over a time course; this is an important feature, which any model of the syndrome should incorporate.

Kenttä and Hassmén (1998) have proposed a comprehensive, proactive model that introduces a healthy emphasis on the role of recovery and includes many of the desirable features of the models of Selye and the supercompensation cycle. Where it possibly shows an element of weakness is in its failure to accommodate the input of the social stressors identified by Coakley (1992). It also retains the emphasis on the pathological condition, an issue that will be raised in the next section.
The Cumulative Stress and Training Continuum Model:--A Multidisciplinary Approach

For the elite athlete, the pursuit of excellence in a chosen field is at the very core of his or her existence—“life itself”—and “the training grounds and stadia are the workplace.” At the very most, UPS should be considered as a special subdivision of the wider burnout process and outcome. Indeed, it can be strongly argued that the term UPS confuses those outside the sport science community and, more importantly, places an erroneous emphasis on excessive training loads as being its principal or even sole causal factor. It is a misnomer, therefore, that serves to mask the true nature of the condition and possibly hinders the research process designed to elucidate its very nature and the mechanisms involved in its perpetration.

The model of Selye (1956) has provided an excellent basis for understanding the problem, while the extremely comprehensive model proposed by Kenttä and Hassmén (1998; 2002) has provided an extensive description of the components of the various processes involved. These authors also have suggested that their model permits the effects of the various problems (e.g., biochemical, physiological, nutritional, etc.) to be studied independently, but they acknowledge that it is only by adopting a holistic perspective that it can be fully comprehended. Thus, this model, while acknowledging the interactive and cumulative nature of potential stressors, fails to adequately convey the complexity of these interactions. A graphic representation of these interactions have been depicted in Figure 3, for which no apology is offered for the somewhat chaotic and confusing picture presented. In much of the research relating to this topic, and specifically that which involves biomedical systems (e.g., the immune system), even a regard for the complexity of these systems in their own right would often appear to be absent. The problems associated with the in vivo study of these systems are immense, and they present a major difficulty for interpreting isolated events and making generalizations. In fact, it is these very difficulties that have made the search for diagnostic markers such a morass.

The interactions are complex, weblike, cumulative, and synergistic. It is proposed that the Kenttä and Hassmén (1998; 2002) model needs to be expanded and revised to include a much stronger emphasis on the complexity of these interactions (see Figures 3, 4, and 5). It also must be acknowledged that many of the symptoms of the condition can result without the effects of excessive training stress at all, and that in some cases it may be increases in nontraining stressors that act as the catalyst causing the athlete to fail to cope with previously acceptable levels of training load. An example of a set of interactions (i.e., those occurring between, training load, fatigue, biochemistry, diet, and the immune system) is presented in Figure 5 to illustrate the nature of this problem.
Figure 3. A multicause diagram of the effects of various stressors on sports performance.
Conclusion

It is clear that UPS is complex in nature and has a multifactorial aetiology. Synergistic interactions assist in explaining how seemingly insignificant factors in their own right exert disproportionate importance and effect. It might, for example, explain why previously acceptable training stress levels in conjunction with other stressors contribute to the pathogenesis. It also highlights the individual variability in susceptibility to UPS and the fluctuations within a given individual (different

**Figure 4.** A simplified model of overtraining syndrome demonstrating the independent effects of training stressors and combined effects of training and nontraining stressors.
temporal expressions). It recognizes that at different times any one of the individual aetiological factors could become the predominant one, and thus act as the triggering mechanism. This insight greatly assists in explaining the insidious and pernicious nature of UPS. The unique contribution of the Cumulative Stress and Training Continuum Model put forward in the present article is that it addresses the interactive and cumulative nature of all potential stressors, and emphasizes the complexity of these interactions. To this end, it presents a truly inclusive and holistic model of UPS.

References


