Overtraining in Athletes
An Update

Rod W. Fry, Alan R. Morton and David Keast
Department of Human Movement and Recreation Studies, The University of Western Australia, Perth, Western Australia, and Department of Microbiology, Queen Elizabeth II Medical Centre, Perth, Western Australia, Australia

Overtraining appears to be caused by too much high intensity training and/or too little regeneration (recuperation) time often combined with other training and non-training stressors. There are a multitude of symptoms of overtraining, the expression of which vary depending upon the athlete’s physical and physiological makeup, type of exercise undertaken and other factors. The aetiology of overtraining may therefore be different in different people suggesting the need to be aware of a wide variety of parameters as markers of overtraining. At present there is no one single diagnostic test that can define overtraining. The recognition of overtraining requires the identification of stress indicators which do not return to baseline following a period of regeneration. Possible indicators include an imbalance of the neuroendocrine system, suppression of the immune system, indicators of muscle damage, depressed muscle glycogen reserves, deteriorating aerobic, ventilatory and cardiac efficiency, a depressed psychological profile, and poor perform-

1. Overtraining

The literature reveals evidence linking excessive exercise and physiological conditions and chronic fatigue which exists within time (reviewed by Keast et al. 1988). Examples of high intensity training has also been documented (Fi, 1985) that they have achieved in practice and in competition (Bompa 1983). Exposure to acute and chronic training loading of overtraining has been shown to experience overtraining athletes have failed to meet performance expectations (Smith et al. 1988). Athletes have often shown chronic lethargy characterized by general fatigue, inability to perform at maximum levels of exercise stress slightly to weeks of training and inability to race as a result of acute and chronic stressors. These athletes have often experienced overtraining, their careers (Morgan et al. 1990). The ‘staleness’ associated with overtraining increases in performance result training load to which the athlete is subjected but which he/she has previously endured. It is obligatory that train the athlete in order to provide adaptation to the stresses encountered (Bempe 1983). Exposures of exercise stress slightly to weeks of training and inability to race as a result of acute and chronic training loading. The recognition of overtraining requires the identification of stress indicators which do not return to baseline following a period of regeneration. Possible indicators include an imbalance of the neuroendocrine system, suppression of the immune system, indicators of muscle damage, depressed muscle glycogen reserves, deteriorating aerobic, ventilatory and cardiac efficiency, a depressed psychological profile, and poor perform-

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Summary

Overtraining appears to be caused by too much high intensity training and/or too little regeneration (recovery) time often combined with other training and nontraining stressors. There are a multitude of symptoms of overtraining, the expression of which vary depending upon the athlete’s physical and physiological makeup, type of exercise undertaken and other factors. The aetiology of overtraining may therefore be different in different people suggesting the need to be aware of a wide variety of parameters as markers of overtraining. At present there is no one single diagnostic test that can define overtraining. The recognition of overtraining requires the identification of stress indicators which do not return to baseline following a period of regeneration. Possible indicators include an imbalance of the neuroendocrine system, suppression of the immune system, indicators of muscle damage, depressed muscle glycogen reserves, deteriorating aerobic, ventilatory and cardiac efficiency, a depressed psychological profile, and poor perform-

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1. Overtraining

The literature reveals extensive anecdotal evidence linking excessive exercise stress with pathological conditions and chronically reduced work capacity which exists within the athlete from time to time (reviewed by Keast et al. 1988; Kuipers & Keizer 1988). Examples of high calibre athletes who have been forced to withdraw from major world events as a result of acute and chronic infections have also been documented (Fitzgerald 1988). Other athletes have failed to meet performance standards that they have achieved in previous years despite maintaining their high level training programmes. These athletes have often reported episodes of chronic lethargy characterised by symptoms of general fatigue, inability to perform consistent weeks of training and inability to recover after hard races (Smith et al. 1988). Athletes have often been shown to experience overtraining at some stage in their careers (Morgan et al. 1987a,b, 1988; Silva 1990). The 'staleness' associated with these decreases in performance results from an excessive training load to which the athlete fails to adapt.

It is obligatory that training programmes stress the athlete in order to provide the stimuli for adaptation to the stresses encountered during competition (Bompa 1983). Exposing the athlete to levels of exercise stress slightly greater than those to which he/she has previously encountered within the training programme, has been termed overload training (Kuipers & Keizer 1988) and is the mechanism by which the stimuli for adaptation is applied. In order for the athlete to adapt to overload training, adequate rest must be incorporated into the training programme and large immediate increments in training avoided (Bompa 1983). If inadequate rest and/or too great an increment in training load has been imposed, the athlete may enter a state of failing adaptation or overreaching, characterised by fatigue and nonrecovery from training sessions (Kuipers & Keizer 1988). If a heavy training load is continued while the athlete is overreaching, chronic fatigue and staleness may lead to deterioration in performance and the athlete will become more susceptible to minor illnesses and chronic infections.

Currently no model cited in the literature presents a reliable method for monitoring the training regimen in such a way that the overloading training stimulus is optimised, thus preventing overreaching. The development of valid, reliable and objective methods for the early detection of overreaching would allow a reduction in training load prior to the more serious symptoms of overtraining becoming apparent. Symptoms of overtraining have been well documented (reviewed by Hackney et al. 1990; Kuipers & Keizer 1988) [see table 1]. The table presents a selected list of symptoms which have been described in the literature as being the consequence of overtraining and illustrates the overwhelming complexity of the overtraining problem. An understanding of this complexity is necessary to gain insight into the scope of the overtraining problem. Recognition of a state of fatigue which renders the athlete susceptible to overtraining prior to the overt symptoms becoming apparent would be of obvious benefit to the athlete and coach.

Overtraining associated in studies which have carefully controlled the training stress imposed on athletes are scarce. The works of Costill et al. (1988),
Table I. The major symptoms of overtraining as indicated by their prevalence in the literature

<table>
<thead>
<tr>
<th>Physiological/performance</th>
<th>Psychological/Information processing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased performance</td>
<td>Feelings of depression</td>
</tr>
<tr>
<td>Inability to meet previously attained performance standards/criteria</td>
<td>General apathy</td>
</tr>
<tr>
<td>Recovery prolonged</td>
<td>Decreased self-esteem/worsening feelings of self</td>
</tr>
<tr>
<td>Reduced tolerance of loading</td>
<td>Emotional Instability</td>
</tr>
<tr>
<td>Decreased muscular strength</td>
<td>Difficulty in concentrating at work and training</td>
</tr>
<tr>
<td>Decreased maximum work capacity</td>
<td>Sensitive to environmental and emotional stress</td>
</tr>
<tr>
<td>Loss of coordination</td>
<td>Fear of competition</td>
</tr>
<tr>
<td>Decreased efficiency/decreased amplitude of movement</td>
<td>Changes in personality</td>
</tr>
<tr>
<td>Reappearance of mistakes already corrected</td>
<td>Decreased ability to narrow concentration</td>
</tr>
<tr>
<td>Reduced capacity of differentiation and correcting technical faults</td>
<td>Increased internal and external distractability</td>
</tr>
<tr>
<td>Increased difference between lying and standing heart rate</td>
<td>Decreased capacity to deal with large amounts of information</td>
</tr>
<tr>
<td>Abnormal T wave pattern in ECG</td>
<td>Gives up when the going gets tough</td>
</tr>
<tr>
<td>Heart discomfort on slight exertion</td>
<td>Immunochemical</td>
</tr>
<tr>
<td>Changes in blood pressure</td>
<td>Increased susceptibility to and severity of illness/colds/allergies</td>
</tr>
<tr>
<td>Changes in heart rate at rest, exercise and recovery</td>
<td>Flu-like illnesses</td>
</tr>
<tr>
<td>Increased frequency of respiration</td>
<td>Unconfirmed glandular fever</td>
</tr>
<tr>
<td>Perfuse respiration</td>
<td>Minor scratches heal slowly</td>
</tr>
<tr>
<td>Decreased body fat</td>
<td>Swelling of lymph glands</td>
</tr>
<tr>
<td>Increased oxygen consumption at submaximal workloads</td>
<td>One-day colds</td>
</tr>
<tr>
<td>Increased ventilation and heart rate at submaximal workloads</td>
<td>Decreased functional activity of neutrophils</td>
</tr>
<tr>
<td>Shift of the lactate curve towards the x axis</td>
<td>Decreased total lymphocyte counts</td>
</tr>
<tr>
<td>Decreased evening postworkout weight</td>
<td>Reduced response to mitogens</td>
</tr>
<tr>
<td>Elevated basal metabolic rate</td>
<td>Increased blood eosinophil count</td>
</tr>
<tr>
<td>Chronic fatigue</td>
<td>Decreased proportion of null (non-T, non-B lymphocytes)</td>
</tr>
<tr>
<td>Inomnia with and without night sweats</td>
<td>Bacterial infection</td>
</tr>
<tr>
<td>Feels thirsty</td>
<td>Reactivation of herpes viral infection</td>
</tr>
<tr>
<td>Anaemia nervous</td>
<td>Significant variations in CD4 : CD8 lymphocytes</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>Biochemical</td>
</tr>
<tr>
<td>Sublima</td>
<td>Negative nitrogen balance</td>
</tr>
<tr>
<td>Upper respiratory infections</td>
<td>Hypothalamic dysfunction</td>
</tr>
<tr>
<td>Headaches</td>
<td>Flat glucose tolerance curves</td>
</tr>
<tr>
<td>Increased aches and pains</td>
<td>Decreased muscle glycogen concentration</td>
</tr>
<tr>
<td>Gastrointestinal disturbances</td>
<td>Decreased bone mineral content</td>
</tr>
<tr>
<td>Muscle soreness/tenderness</td>
<td>Delayed menarche</td>
</tr>
<tr>
<td>Tendinopathic complaints</td>
<td>Decreased haemoglobin</td>
</tr>
<tr>
<td>Periodic complaints</td>
<td>Decreased serum ferritin</td>
</tr>
<tr>
<td>Nausea</td>
<td>Lowered TIBC</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Mineral depletion (Zn, Co, Al, Mn, Be, Cu, etc.)</td>
</tr>
<tr>
<td>Changes in appetite</td>
<td>Increased urea concentrations</td>
</tr>
<tr>
<td>Increased C-reactive protein</td>
<td>Elevated ketosteroids in urine</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
<td>Low free testosterone</td>
</tr>
<tr>
<td></td>
<td>Increased serum hormone binding globulin</td>
</tr>
<tr>
<td></td>
<td>Decreased ratio of free testosterone to cortisol of more than 30%</td>
</tr>
<tr>
<td></td>
<td>Increased uric acid production</td>
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</tbody>
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Kirwin et al. (1988), Morgan et al. (1990) are exceptions: carefully controlled training and monitoring of that stress using indices are required before thorough insight into overtraining can be achieved. Studies into regulation and the effect of training in response to controlled overtraining may be the key to the development of a monitoring training to prevent it.

2. Terminology

Overtraining results from between performance demand and available performance capacity (Kindermans & Keizer 1988; Noakes 1989; Re Gundersen et al. 1986), despite maintenance of training load. Kindermans' statement does not identify this with the statement that overtraining occurs in the absence of recovery. Kindermans's statement does not identify this with the statement that overtraining occurs in the absence of recovery. Kindermans's statement does not identify this with the statement that overtraining occurs in the absence of recovery.

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Fatigue resulting from placing stress on specific muscle groups. Morgan...
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Kirwan et al. (1988), Morgan et al. (1988) and Murphy et al. (1990) are exceptions. More studies with carefully controlled training stress and systematic monitoring of that stress using a variety of stress indices are required before scientists can gain a thorough insight into overtraining and confidently prescribe training to optimise performance and avoid overtraining. Studies investigating hormonal regulation and the effect of hormonal misregulation in response to controlled training situations may be the key to the development of a system for monitoring training to prevent overtraining.

2. Terminology

Overtraining results from a discrepancy between performance demand and functional capacity (Arnold 1980; Mellerowicz & Barron 1971). Overtraining is defined principally as a decreased performance capacity (Kuipers & Keizer 1988; Noakes 1989; Reidman 1985; Stray-Gundersen et al. 1986), despite increased or maintenance of training load. Kuipers (1986) qualifies this with the statement that depressed performance occurs in the absence of organic illness. Kindermann's statement does not appear to be correct, however, as evidence presented in this review suggests that the immune system can be depressed in overtraining, leading to infectious disease accompanying overtraining.

Historically no uniform terminology has been used to define overtraining (Callister et al. 1990; Kuipers & Keizer 1988). Standard terminology must be encouraged in the international literature and the state of the art paper presented by Kuipers and Keizer (1988) provides a sound starting point. Several aspects of that terminology are developed further in this paper. Kuipers and Keizer (1988) state that confusion arises because most investigators do not discriminate between the process of overload training (stimulus for adaptation) and the outcome, which may be overtraining (short term overtraining), overtraining syndrome (chronic overtraining) or muscular overstrain, which is local fatigue resulting from placing too much strain on specific muscle groups. Morgan et al. (1987) used the term 'overtraining' to mean overload training, and stated that overtraining is approached as a deliberately planned and appropriate feature of training microcycles. Coaches and sport scientists in the United States (Griffith 1926; Morehouse & Rasch 1958; Morgan 1985b; Wolf 1961) have historically labelled a state of failing adaptation as 'staleness', whereas European workers have tended to use the term 'overtraining' (Rompa 1983; Harre 1982; Kurety 1971; Mellerowicz & Barron 1971). The terms 'overfatigue' and 'overstrain' have also been used synonymously with overtraining and staleness (Craikowski 1982), while 'chronic fatigue' has been used to mean overtraining (Kurety 1971; Wolf 1961).

The term which has emerged in the scientific literature since Falsetti's (1983) and Brown's (1983) statements about short term overtraining is 'overreaching'. Falsetti (1983) states that overreaching is used by some coaches as a form of training using a large stimulus for adaptation to promote a fitness peak prior to a major competition. This results in decreased performance for a short period of time but following an extended recovery period results in supercompensation and increased performance capacity (Fleck 1988; Kuipers & Keizer 1988). Overreaching is, however, the first stage of overtraining which if disregarded develops into the more severe overtraining syndrome. Complete recovery from overtraining may require weeks or months (Kuipers & Keizer 1988).

It now appears that some redefinition of terms is warranted:

1. The process of stressing an individual to provide a stimulus for adaptation and supercompensation is overload training.
2. Training fatigue/stress is the normal fatigue that is experienced following several days of heavy training associated with an overload training stimulus. This fatigue is reversed and supercompensation occurs by the last few days of a period of reduced training load (regeneration microcycle).
3. Overtraining is the general term which indicates that the individual has been stressed by training and extraneous stressors to the extent that he/she can not perform at an optimum level following...
an appropriate regeneration period. A drop in performance is necessary for overtraining diagnosis.

4. Overreaching follows the intentional or unintentional induction of short term overtraining. The symptoms of overreaching can be reversed by a longer than normal regeneration period.

5. The overtraining syndrome is the state of chronically depressed performance accompanied by one or more of the more serious symptoms. Overtraining syndrome and staleness will be considered synonymous, as discussed by Kuipers and Keizer (1988). Recovery from the overtraining syndrome requires a significantly longer time than that required in overreaching.

Overstrain follows acute tissue damage induced by isolated intensive training sessions which exceed the muscular stress tolerance. This generally occurs after single or repeated bouts of excessive exercise which result in damage to muscle fibres. Muscular overstrain may or may not accompany overreaching or overtraining syndrome (Kuipers and Keizer 1988).

Training stressors are those resulting from the physical, physiological, and psychological stress induced by the training workloads administered during overload training. Extraneous stressors are those resulting from activities and psychological forces related to lifestyle. Supercompensation is a state of heightened work capacity, above that of which the athlete has recently been capable. This has been characterised as a state of balanced homeostasis with homeostatic markers reflecting either baseline values or improvements, depending upon the nature of the variable.

3. The Basic Theory of Athletic Improvement - Overload Training

Celest Ullrich states 'Whenever the homeostatic balance of the body is upset, the human organism attempts to adjust in such a way that the balance is restored. Until the balance is restored, a state of stress exists' (Counsilman 1968). Improvements in an athlete's ability to tolerate the demands of competition and training are achieved through adaptation to the stressors applied in the training programme (Bompa 1983, 1989; Counsilman 1968; Harre 1982; Kukushkin 1983; Matveyev 1981; Selye 1957). Thus the aim of the training process is to apply a series of stimuli which will displace the homeostasis of the individual's functional systems and therefore provide the stimulus for adaptation (Matveyev 1981).

Following overload it has been shown that there is a period where metabolism re-establishes homeostasis, the length of this period depends upon the degree to which homeostasis has been disrupted (Bompa 1983; Harre 1982). In addition to regaining homeostasis, the organism adapts to the stressor such that, if the same stressor were imposed again, the homeostatic mechanisms would not be displaced to the same extent (Bompa 1983; Harre 1982; Kukushkin 1983; Selye 1957). Following this adaptation an individual has been shown to be capable of doing more work for an equivalent homeostatic displacement.

For this adaptation and supercompensation to be of benefit, a period where no further homeostatic displacement occurs is necessary to provide adaptational processes time to become reorganised (Harre 1982). For this reason, rest has been regarded as an organic component of the training process (Counsilman 1968; Harre 1982; Matveyev 1981). However if no secondary adaptational stimulus is applied during the phase of supercompensation it has been shown that involution in the degree of adaptation occurs and the training effect is lost (Bompa 1983, 1989; Harre 1982; Matveyev 1981).

The degree of supercompensation achieved depends upon the size of stimulus for adaptation and, therefore, the degree of imbalance in homeostasis induced. For this reason, a series of fatigue inducing stimuli may be applied in order to create a 'valley of fatigue' (Counsilman 1968). This fatigue valley occurs because full recovery is not permitted between training sessions, creating a progressively greater imbalance in homeostatic mechanisms and, consequently, a more powerful stimulus for regeneration and supercompensation.

In general, physiological and biochemical research have supported the concept of fatigue-induced supercompensation compensation has been demonstrated 2 or 3 day stimulus. Other studies have followed intensive training and staleness (Adams 1966), swimmer's fatigue (Adams et al. 1988) and arm stroke (Stern et al. 1985).

4. Consequences of F. Symptoms of Overtraining

There is no evidence it provides clear guidelines for from the symptoms of the. There is a continuum of symptoms (see fig. 1), th those that reflect the im
with the definition of overtraining may be separating the normal fatigue which results from a series of high intensity training sessions and is necessary to promote a training effect from the underlying fatigue associated with overtraining. The problem arises because we do not yet have a clear definition for the point at which training fatigue finishes and overtraining begins. In addition, the symptoms of training fatigue will be superimposed on those of overtraining, further complicating the picture. The key to separating overtraining from the normal training response may lie in the structure of the training programme through the definition of the time point in each block of training when full regeneration should be complete. Poor performance or indicators of an imbalance in homeostatic mechanisms at this stage would indicate that residual fatigue is still present and that subsequent heavy training should not be initiated.

It is likely that everyone responds to stress in different ways and, therefore, the large variety of symptoms presented in Table I represents individual variation in the expression of overtraining symptoms. In addition, different types of activity (e.g. predominantly anaerobic vs predominantly aerobic) may result in different symptoms.

At the outset of overtraining, increased fatigue and decreased performance may be the only symptoms, but with accumulation of fatigue other objective and subjective symptoms have been shown to appear progressively (Kuipers & Keizer 1983). Falsetti (1983) states that there is a constellation of symptoms. However, short and long term overtraining may differ only in the length of time that the symptoms have existed and the time it takes for recovery (Henschen 1986; Nideffer 1988).

More recently it has been suggested that some of the symptoms associated with the overtraining syndrome may be associated with excessive production of cytokines (Lloyd et al. 1989; McDonald et al. 1987; Wakefield & Lloyd 1987) due to an unbalanced immune system. The role of stress proteins in perpetuating the symptoms of overtraining syndrome is also worthy of research (Burdon 1987; Khamarsi et al. 1990; Lazlo 1988; Lindquist & Craig 1988; Locke et al. 1990; Polla 1988; Polla & Young 1989; Schlesinger 1986; Welch 1987; Young & Elliot 1989). It is probable that continued training while under the stress of overreaching stimulates the transcription, translation, and subsequent expression of stress proteins resulting in their associated symptoms.

5. Who is Susceptible?

Athletes at all levels of performance are at risk of overtraining, but it is the most highly motivated athletes who are most prone (Costill 1986; Karpovich 1965; O'Brien 1988; Town 1985). This is probably regardless of the athlete's level of performance (Town 1985; Stray-Gundersen et al. 1986) and it may be overtraining that prevents many sports people from becoming elite.

Emulating the training of elite performers may be a particular problem for the inexperienced (Falsetti 1983). Less experienced athletes and those who train themselves may be particularly prone to overtraining due to inexperience at recognising symptoms and naivety with regards to the relative roles of overload and regeneration in the training process (Falsetti 1983; Noakes 1989; O'Brien 1988). These athletes are more likely to be subjected to amateur coaching techniques.

Wolf, in 1961, studied 95 athletes (aged 15 to 34) who had exhibited symptoms of overtraining. He found that 73 out of the 95 cases occurred in sports where strength, speed and coordination were essential. Athletes who are going through a bad patch or attempting to 'break a plateau', may begin to train incessantly and, instead of improving, performances become worse (Karpovich 1965). This may lead to overtraining becoming an epidemic in a whole squad if it is experienced by a particularly difficult period of training and competition (Wolf 1961). Finally, those athletes who have been absent from a squad can not rejoin at the same level of intensity of training as when they left, as they will have lost condition and preparedness to train (O'Brien 1988).

The most outstanding training-related factor leading to overtraining is reported to be the failure to include enough regeneration units in the training programme (Bompa 1983; Erick 1983; O'Brien 1988; there are many other training-related factors as well as nontraining shown to contribute to overtraining (Keizer 1988)).

6. Recognition of Overtraining

It can be seen from this that overtraining will be represented by various homeostatic balance, while being recognised by enhanced performance on test criteria at previous levels. Ideally, subjective symptoms should take place until supercompensation (Bompa 1983; Harre 1982;1.

The athlete's own pre-existing physical and psychological state may be a particular problem for the inexperienced (Falsetti 1983). Less experienced athletes and those who train themselves may be particularly prone to overtraining due to inexperience at recognising symptoms and naivety with regards to the relative roles of overload and regeneration in the training process (Falsetti 1983; Noakes 1989; O'Brien 1988). These athletes are more likely to be subjected to amateur coaching techniques.

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6. Recognition of Overtraining

It can be seen from this review that overtraining will be represented by variables not achieving homeostatic balance, while supercompensation will be recognised by enhanced, or at least equal, performance on test criteria compared with the previous levels. Ideally, subsequent training should not take place until supercompensation has occurred (Bompa 1983; Harre 1982; Kuipers & Keizer 1988).

The athlete's own previous results (as opposed to population or group norms) must be used as his/her own standard (Bompa 1983; Brown 1983; Burke 1982; Halpern 1986; O'Brien 1988). Relating results to normal population ranges may not be sensitive enough to identify the subtle changes that may be indicative of the onset of overtraining. Results may have to move markedly before they may be considered to be clinically significant in terms of the normal population; however, the degree of movement relative to the athlete's own scores may be relatively minor at the onset of overtraining. Gross overtraining and staleness may occur well before an athlete's results move out of the normal range. One-off testing merely characterises athletes relative to a group and does not indicate a direction of movement in variables (Van Handel et al. 1988a). Therefore, before data can be used to define overtraining, baseline data must be generated when the athlete is in a normally trained state (Brown 1983; Nideffer 1988; Van Handel et al. 1988a).

To date there has been no single definitive test that can show overtraining. Due to the interrelationships between variables which may be associated with overtraining or overreaching, it is probable that there will never be one definitive parameter diagnostic of the overtrained state, because overtraining will affect different bodily systems in different ways depending upon the nature of the sport. The approach to be recommended, therefore, is the development of an integrated set of tests based on several parameters known to be associated with unbalanced homeostasis. The ideal screening test battery would be designed to be incorporated within the periodised training programme and include components useful for monitoring improvement, establishing training workloads, and screening for overtraining.

The time required for the restoration of homeostasis will vary depending upon the functional systems being stressed. As a consequence the decision of which tests to include in a test battery must be based upon the identification of parameters which reflect the homeostatic systems identified in the literature as those most likely to be disrupted as overtraining develops. A discussion of such systems and parameters follows.

7. The Parasympathetic vs Sympathetic Systems

Israel (1976) identified 2 types of overtraining one as the result of a sympathetic nervous system dominance (bodysmoid) and the other characterised by parasympathetic nervous system dominance (addisonoid). The difference in the differentiation between the 2 types of overtraining is carried further in the works of several other authors (Bompa 1983; Kerezty 1971; Kindermann 1986; Kuipers & Keizer 1988). Kindermann (1986) lists symptoms of sympathetic overtraining as increased pulse rate at rest, decreased body mass, disturbed sleep, decreased pulse recovery after load, decreased appetite, emotional instability. Bompa (1983) adds sweating to this list and goes into more detail with regards to psychological symptoms. Kerezty (1971) states that elevated basal metabolic rate, negative nitrogen balance and ECG abnormalities are also symptoms of sympathetic overtraining.

Symptoms of parasympathetic overtraining are reported by Bompa (1983) to include progressive anaemia, low blood pressure and digestive disturbances. Kerezty (1971) states that parasympathetic
dominance may be characterised by a diminished counterregulatory capacity against hypoglycaemia. The existence of such a diminished capacity is supported by the research findings of Barron et al. (1985) and Kuipers and Keizer (1988), although these may also be evident in sympathetic overtraining as Barron et al. (1985) reported elevated basal cortisol levels in their study. Kuipers and Keizer (1988) add early fatigue, low resting pulse, a fast return of heart rate to basal levels following exercise as other important factors associated with parasympathetic overtraining. The authors also state that parasympathetic overtraining may be more difficult to define as the symptoms are less alarming and, in the initial stages, are more like the effects of training improvements.

Symptoms in table I generally reflect a predominance of the sympathetic form of overtraining in the literature, and this is supported by statements cited in Kerezty (1971) and Kuipers and Keizer (1988). An outstanding factor for the diagnosis of overtraining is that both types will be reflected by a decreased work capacity (Kerezty 1971). It is clear that overtraining represents a perturbation of nervous regulation (Bompa 1983; Michael 1961).

It has been proposed that the parasympathetic form of overtraining may be a reflection of an advanced state of overtraining closely associated with exhaustion of the neuroendocrine system, while the sympathetic type may reflect a prolonged stress response preceding exhaustion (Kindermann 1986; Kuipers & Keizer 1988). This fits nicely into the general adaptation model proposed by Selye (1957). Alternatively, sympathetic overtraining may predominantly affect speed and power athletes, while endurance athletes are more prone to parasympathetic signs of overtraining. Individual differences in the nervous system will also predispose to either sympathetic or parasympathetic overtraining (Harre 1982).

The existence of both types of overtraining indicates that the physiologist must be alert to both down-regulation and up-regulation of the neuroendocrine homoeostasis when attempting to screen for and diagnose overtraining in athletes. It is apparent that the differences in the neuroendocrine response may be due to either progression through different stages of the overtraining response, individual variation or differences in the stress imposed by the sport undertaken. Certainly, however, there are many reports of sympathetic symptoms in endurance athletes.

8. The Neuroendocrine System

Training may be able to facilitate an adaptation to stress because gradually increasing training loads have been shown to increase stability of the pituitary-adrenocortical system as indicated by lower basal levels of stress hormones like prolactin and adrenocorticotropic hormone (ACTH) (Keizer et al. 1987a,b; Viru 1985a,b). However, excessive stress leading to imbalances in the neuroendocrine axis may also contribute to the symptoms of overtraining (Israel 1976). Repeated exercise when hormonal balance is deranged may further stress the hormonal system, thus compromising recovery (Kuipers & Keizer 1988).

Barron et al. (1985) presented evidence for adrenocortical insufficiency in athletes suffering from overtraining, as suggested by previous authors (Kerezty 1971; Mellerowicz & Barron 1971; Prokop 1963). The authors found that the hypothalamus was less sensitive to the stress of hypoglycaemia in overtrained subjects and attributed this to hypothalamic exhaustion. The data revealed impaired growth hormone, ACTH, cortisol and prolactin responses to insulin-induced hypoglycaemia. The sample group (n = 4) used in the Barron et al. (1985) study is small and it would be of benefit to overtraining research to have the study repeated with a larger number of subjects, although the difficulties of such a task are appreciated. The hypothalamus is known to be the coordinating centre between the hormonal system, autonomous nervous system and behaviour (Selye 1957). Insulin-induced hypoglycaemia acts by altering the secretion of hypothalamic factors which have been shown to stimulate the r hormone and prolactin (Barron et al. 1985). Barron pituitary dysfunction as suggested that the dysfunction they do state that pituitary insufficiency, but that this could their study due to the later stage. The concept of altered hormone levels being elevated (Barron et al. 1985) has the potential to insomnain symptoms reported in overtraining.

Further support for hypofunction in overtrained athletes comes from reports of menstrual dysfunction in female athletes who have amenorrhoea through exercise or stress (Dale et al. 1979; De Souza 1978; Frisch et al. 1980; Israel 1980; Ruffin et al. 1980). Impaired ovarian function may be related to diminished pituitary control (Feicht et al. 1978) of hypothalamic control of pituitary function in overtrained athletes (McArthur et al. 1980). Exercise changes appear similar to severe illness, major weight loss, environmental stress, or other stressful events (Prior 1982, 1987). The composure to be the hypothalamic-luteotropic axis (Prior 1987). The hypothalamic sensitisation and producing region stimulates the pituitary to produce luteinising hormone (Pituitary) stimulating hormone (FSH) responsible for the regulation in this context, much of the reproductive-system is involved in this process. Other changes include changes in the pulsatile release of FSH, luteinising hormone (Pituitary) stimulating hormone (FSH) and others.
Overtraining in Athletes

1. up-regulation of the neuroendocrine system when attempting to screen entraining in athletes. It is apparent in the neuroendocrine system to either a progression through the overtraining response; individual differences in the stress experienced are evident. Certainly, however, the role of sympathetic symptoms is significant.

**Neuroendocrine System**

- able to facilitate an adaptation to the dually increasing training loads
- increase stability of the anterior pituitary, its hormones like prolactin and cortisol (ACTH) (Keizer et al. 1985a,b). However, excessive stressors in the neuroendocrine system may further stress on the body, leading to the symptoms of overtraining (see fig. 2).

Further support for the hypothesis that overtrained athletes are prone to the stress of hypoglycaemia was provided through female athletes who have been shown to develop amenorrhea through exercise (Cohen et al. 1982; Prior 1982, 1987). The common link has been hypothalamic-pituitary dysfunction as a cause of overtraining and the inability to regulate blood sugar levels have been reduced in overtraining (Barron et al. 1985; Kuipers & Keizer 1988). Hypothalamic-pituitary dysfunction has the potential to induce a range of symptoms in overtrained athletes, as reported in overtraining (see fig. 2).

Although the incidence of menstrual disorders appears to be high in runners and ballet dancers (Loucks & Horvath 1985), swimmers, rows, fencers, volleyball players, tennis players, skiers, gymnasts, cyclists, field athletes and aerobic dance instructors are susceptible (Shangold 1985). Shangold states that menstrual dysfunction is most likely to occur in sports where loss of body fat is combined with excessive levels of exercise, these are mainly endurance activities by nature. Other studies have supported this statement (Frisch et al. 1980; Lutter & Cushman 1982; Speroff & Levene 1982; Speroff & Redwine 1980) and have suggested that amenorrheic athletes lose more weight in training than cyclic athletes. Frisch et al. (1980) found that athletes who began training before menarche experienced their first period at an average age of 15 years, whereas those who began training after menarche experienced theirs at an average age of 12.7 years, which was similar to the general population. Warren (1980) found that there was a prompt onset of amenorrhea in male dancers during injury- or vacation-related suspensions of exercise of at least 2 months. Lutter and Cushman (1982) and Sanborn et al. (1982) demonstrated abnormal luteal functions and low LH surge and found that these disturbances correlated with the distance run by athletes each week, but others have found no correlation between reproductive function and training load. The consequences of disturbed hormonal regulation associated with chronic repro-
Fig. 2. The hypothalamic-pituitary axis and overtraining. Stress generates many of the symptoms of overtraining through the response of the hypothalamic-pituitary axis. Note that this figure does not attempt to portray the complexity of hormonal regulation but rather portray the effects that misregulation may have on several bodily systems.

Productive dysfunction may include endometrial hyperplasia, adenocarcinoma, atopic endometrial hyperplasia (Coulam et al. 1983), vaginitis, urethritis, low bone density, osteopenia (Dalsky 1990; Drinkwater 1984; Lindberg et al. 1984; Marcus et al. 1985) and a higher incidence of stress fractures than cyclic athletes (Lindberg et al. 1984; Marcus et al. 1985). Reproductive dysfunction in males is less obvious and the most overt symptoms may be a decreased libido and sperm count. Similar hormonal mechanisms are anabolic-pituitary dysfunction to be the mediating factor (1989).

Severely overtrained European countries who o months and possibly years develop Addison’s disease explains the cause of Addison’s disease of the adrenal gland to monal concentrations, in p

Changes in circulatory fections have been shown to as and the duration of the re exercise (Hickson et al. 1990; A neuroendocrine stress response the recovery phase may ha state that a changed balance anabolic hormones may aff Intense physical exercise has increased cortisol, decre and an increased sex horm (SHBG) which contributes toerone levels by increasing the capacity of serum (Aldcreutz & Aldercreutz 1985; Kurowt & Viru 1982) as is the eleva Decreased testosterone levels l direct inhibition of testicular through elevated cortisol leve 1985; Cumming et al. 19 anisms (Cumming et al. 1989) be slow to return to normal lev exercise (Cumming et al. 1989; Griffith et al. 1990; Harkeet al. 1986; Kuoppasalmi et al. 1984) and may take several da 1989). Testosterone may be elem exercise (for reviews see ming et al. 1989); however, Ald have suggested that a decrease in free testosterone to cortisone
Overtraining in Athletes

Severely overtrained athletes from Eastern European countries who continue to overtrain for months and possibly years have been reported to develop Addison's disease (Noakes 1989). Noakes explains the cause of Addison's disease as the failure of the adrenal gland to properly regulate hormonal concentrations, in particular cortisol.

Changes in circulatory hormonal concentrations have been shown to affect the rate of recovery and the duration of the recovery phase after exercise (Hickson et al. 1990; Kuipers & Keizer 1988). A neuroendocrine stress response which persists in the recovery phase may have important implications, because Kuopiasalmi and Aldercreutz (1985) state that a changed balance between catabolic and anabolic hormones may affect recovery processes. Intense physical exercise has been shown to lead to increased cortisol, decreased free testosterone, and an increased sex hormone-binding globulin (SHBG) which contributes to the low free testosterone levels by increasing the testosterone binding capacity of serum (Aldercureutz et al. 1986). It has been shown that the down-regulation of cortisol production is of prime importance for the onset of anabolic processes (Kelly et al. 1986; Kuopiasalmi & Aldercreutz 1983; Kurkowski et al. 1984; Seene & Viru 1982) as is the elevation of testosterone. Decreased testosterone levels have been caused by direct inhibition of testicular secretion, possibly through elevated cortisol levels (Aakvaag & Opstad 1985; Cumming et al. 1989) or other mechanisms (Cumming et al. 1989). Testosterone may be slow to return to normal levels following severe exercise (Cumming et al. 1989; Dufauz et al. 1979; Griffith et al. 1990; Hakkinen et al. 1987; Jansson et al. 1986; Kuopiasalmi et al. 1980; Webb et al. 1984) and may take several days (Cumming et al. 1989). Testosterone may be elevated following short term exercise (for reviews see Bunt 1986; Cumming et al. 1989); however, Aldercreutz et al. (1986) have suggested that a decrease in the ratio of plasma free testosterone to cortisone of more than 30%, combined with a high level of SHBG capacity, may be a useful indicator of overtraining.

Many authors have reported elevated blood cortisol following exercise (Bunt 1986; Dessypiris et al. 1976; Kuopiasalmi et al. 1980; Kuopiasalmi & Aldercreutz 1985), while others have reported decrements following prolonged exercise (Bunt 1986; Viru 1985b). Maron et al. (1977) have reported that cortisol returns to pre-exercise levels within 2 to 3 hours of exercise cessation. However, with long-lasting intense exercise, cortisol and catecholamine levels may decrease below pre-exercise levels for several days (Viru 1985a). Ryan (1983) has reported that there is great individual variability in levels of hormones after exercise, which makes generalisation difficult.

Since the testosterone/cortisol ratio has implications for the switching on of anabolic processes in recovery, and as this may require more than 1 day to occur, the ratio may be associated with the reported catabolic state reported in overtraining (Kuipers & Keizer 1988). Increased binding of cortisol in muscle tissue, combined with the high levels of plasma cortisol, may lead to greater protein catabolism than anabolism inside the muscle cells. This would explain the increased urea and loss of body mass in overtrained athletes (Kindermann 1986). Kindermann (1986) has suggested a control value of 8.3 mmol/L urea in serum should not be exceeded during training. Monitoring urea levels during the regeneration phase may thus be useful in indicating the return to baseline of the nitrogen balance, and as this is hormonally driven, may be representative of restoration in hormonal mechanisms.

It is assumed that the strong activation of the pituitary-adrenocortical system is related to the sympathetic type of overtraining while strong inhibition or exhaustion of the sympathetic nervous system relates to the parasympathetic type of overtraining. Stress fractures may be caused by a reduction in capacity of the body to recover and repair tissue and may be related to changes in levels of hormones in the body (Frederick 1983). Alterations in caloric intake may reflect neurophysiological changes, since it has been shown that the
hypothalamus is important in regulating food intake (Keesey & Powley 1986).

Since hormone levels may be altered in states of physical fatigue (Aakvaag & Opstad 1985; Viru 1985a) monitoring hormones may be useful for the indication of overtraining. Measurement of the ACTH and cortisol response to insulin-induced hypoglycaemia could be a useful diagnostic test for overtraining in athletes and for monitoring recovery. Details of methods are described in Barron et al. (1985). Regulation of blood glucose levels may be inadequate resulting in the depression of blood glucose following a standard exercise protocol, measurement of pre- and postexercise glucose levels may be an indication.

One of the most serious consequences of overtraining may be a suppression of immune function such that the athlete becomes subject to short or long term reduced performance due to the contraction of infectious disease. It is known that participation in exercise during the incubation period of a viral illness can induce more serious symptoms and cause death from complications (Ames 1989). The first documented association between overtraining and increased incidence of infection was recognised in 1928 following the St Moritz Winter Olympics (Heis 1971). Since then, a substantial body of evidence has accumulated which supports such an association. The athlete may become progressively more susceptible to infection as training progresses at an intensive level through the course of the season (Asgeirsson & Bellanti 1987; Jokl 1984; Salo 1989; Tomasi et al. 1982). The training process has also been shown to be associated with considerable psychological stress, particularly during periods of competition and this may be an important contributor to immune suppression and increased morbidity in athletes under stressful training and competition conditions (Khansari et al. 1990; Morgan et al. 1987). It is essential, however, to separate the acute immune responses to exercise and responses due to failing adaptation. Some of the immunological changes associated with training may be a tuning of the immune system and not overtraining responses. Keast et al. (1988) and Cameron (1988) have reviewed the acute responses of the immune system to exercise. The response of natural killer cells, immunoglobulins and the leucocytosis of exercise have also been reviewed (Mackinnon 1989; McCarthy & Dale 1988).

There is no doubt that exercise has a major impact on components of the immune system, but whether the immunomodulation which occurs in association with acute exercise is clinically significant or not has yet to be determined. There is no evidence, anecdotal, epidemiological or otherwise that is able to associate increased incidence of infection directly with acute exercise. There is, however, considerable evidence to suggest that acute exercise can exacerbate current infection. Evidence suggesting that exercise training, as opposed to one-off intensive exercise, can lead to an immunosuppression does exist.

According to Surkina (1982) the immune system plays an essential role in maintaining the body's health and homeostatic state. Surkina states that, during participation in sports, the immune system is subjected to the training effects of physical and emotional loads which expand its functional limits. Ivanova and Tolkko (1981) have shown that acute intensive exercise induces a disruption of immunological homeostasis and that this provides a stimulus for immunological compensation and supercompensation following a regeneration period. Their study demonstrated that training improved baseline values of a number of immunological parameters. T-lymphocyte blast transformation in response to mitogens was significantly higher than values prior to the training period. However, Surkina (1982) has shown that when workloads were excessive, the immune system homeostasis was displaced to the extent that the athletes became increasingly susceptible to opportunistic infection. This may result from extremely sharp stress (2 marathons over 2 days) or from the accumulation of training and competition stress due to insufficient or no recovery time (chronic stress).

There are many reports of athletes who perceive themselves as being more susceptible to illness during periods of heavy training (Gerald 1988; Koch 1988; Salo 1989; Schouten et al. 1989) and in the immune system m human performance (F. letes have indicated tht to be enhanced with p (Anderson 1989; Koch 1 have demonstrated that programmes are capable of (Good & Ferrandes 1989).

There is now a pletcating that immunity can be enhanced with periodic illness. When physically monitored over a 9-week period athletes monitored over a 9-week period athletes have been shown to have increased incidence of infections and experienced symptoms of colds caused more abs did injuries (Johannson 1 studies of elite sportswomen skiers (Anderson 1989; Rl al. 1982) and an epidemian and Bateman (1983), whenpared with 15.3% of coniptory infections in the skiers. They also demonstrate that highly trained runners suf of infection with almost h experiencing symptoms. I increased susceptibility to sps recorded regularly over t has been associated with reactivation of latent herp cort-Glaser & Glaser 1987; borsky et al. 1976). Fol herpes virus, individuals for life (Kiecolt-Glia) hepatitis outbreak at the United States (Fox 1985; Morse et al. 1972) cause
with training may be a tuning
that exercise has a major im-
exercise induces a disruption
and a half months later he became ill. Surkina
slowed to the extent that the 
T-lymphocyte blast transfor-
to mitogens was significantly 
to prior to the training period.
(1982) has shown that when 
that is may result from extremely 
ons over 2 days) or from the 
and competition stress due to 
water, 90 out of 97 members of a college football 
t own as being more prone to infection during 
periods of heavy training (Anderson 1989; Fitz-
gerall 1988; Koch 1988; Reilly & Rothwell 1987; 
Schouten et al. 1988), suggesting that 
immune system may be a limiting factor for 
human performance (Fitzgerald 1988). Some ath-
ists have indicated that their immunity appears 
to be enhanced with moderate levels of training 
(Anderson 1989; Koch 1988). Experiments in mice 
have demonstrated that carefully designed training 
programmes are capable of enhancing immunity 

There is now a plethora of information indi-
cating that immunity can be adversely affected by 
poor training programmes. Reilly and Rothwell 
(1987) report that elite athletes had more days off 
training through illness than club or recreational 
racers. When physically conditioned rowers were 
monitored over a 9-week period, they were found 
more frequent and severe upper respiratory tract infections than control subjects (Douglas & Hansen 1978). In a 1-year prospective study of 
orienters, it was noted that minor diseases such as 
colds caused more absences from training than 
did injuries (Johansson 1986). This is supported in 
reviews of elite sportsmen, marathon runners and 
skiers (Anderson 1989; Reid et al. 1989; Tomasi et 
al. 1982) and an epidemiological survey by Peters 
and Bateman (1983), where 33.3% of runners com-
pared with 15.3% of controls suffered upper respir-
atory infections in the 2 weeks following a mara-
thon run. They also demonstrated that the more 
highly trained runners suffered a greater incidence 
of infection with almost half of the faster runners 
experiencing symptoms. Further evidence of in-
creased susceptibility to specific infections has been 
recorded regularly over the past 20 years. Stress 
has been associated with the development of and 
reactivation of latent herpes virus infection (Kiec-
coll-Glaser & Glaser 1987; Kosi et al. 1979; Lu-
borsky et al. 1976). Following infection with a 
herpes virus, individuals will remain latently in-
fected for life (Kiecoll-Glaser & Glaser 1988). In a 
hepatitis outbreak at the Holy Cross College in the 
United States (Fox 1985; Friedman et al. 1985; 
Morse et al. 1972) caused by polluted drinking
had low leucocyte counts and that these counts became lower as training progressed. On the other hand, a characteristic of promising athletes has been found to be a high comparative leucocyte count which persists throughout their career. Matvienko encountered cases that resembled leucocytosis; however, the athletes displayed good performances. He states that care must be taken in the assessment of haematological data since athletes could be wrongly excluded from a team when they have high leucocyte counts as these may be indicative of infection in some athletes. The importance of comparing an athlete’s own data with his/her own previous baseline data is highlighted by such examples. Umarova (1982) has shown that special care must be taken when administering heavy training loads to children as greater depressions of immunological parameters occur following training in children than with adults.

Skin sepsis and other bacterial infections may be more frequent in athletes (Asgeirsson & Bellanti 1987). Although it has often been difficult to differentiate between physical exertion and physical factors, such as trauma or crowding, as the cause of clinical symptoms, skin infections are commonly caused by staphylococci, streptococci or herpes simplex virus, and there is evidence to suggest that these occur predominantly through skin to skin contact (Dorman 1981; Ludlum & Cookson 1986; Selling & Kibrick 1964; Wheeler & Cabaniss 1965; White & Grant-Kels 1984). Other common skin infections among athletes are groin infection and athlete’s foot (Asgeirsson & Bellanti 1987).

What is not yet clear is the relative importance of training-induced immune suppression and the close proximity of athletes in many of the above situations as the predominant aetiological factor for the abnormally high incidence of infection.

9.1 Regulation of Immunity

The regulation of the immune response is very complex and involves a number of functional body systems, complex feedback mechanisms, an enormous range of messenger molecules and an as yet undetermined range of leucocyte populations and subpopulations. More detailed discussions of factors involved in immunoregulation can be found in the immunology literature (Khanzadi et al. 1990; Male et al. 1987; Roitt et al. 1989) [see fig. 3]. There are as a consequence a large number of possible mechanisms by which overtraining may affect the immune response.

It is now becoming apparent that the immune system relies exclusively on glutamine for cell replication (Ardawi & Newsholme 1985). Muscle may be the main source of this glutamine as cells of the immune system cannot synthesise these glutamine requirements de novo. The rate of glutamine utilisation of quiescent and dividing lymphocytes has been shown to be high (Newsholme 1990) and these cells cannot replicate in its absence (Griffiths & Keast 1990; Szondy & Newsholme 1989). Because glutamine synthesis and/or release across the muscle cell membrane appears to be the flux-generating step for other cells of the body such as cells of the immune system (Newsholme 1990; Newsholme & Leach 1983), under normal physiological conditions the dynamics of this system may be critical to the supply of glutamine to these tissues. Because of this, Newsholme states that the muscular system is part of the immune system and failure of the muscle to release sufficient glutamine may result in an impairment of immune function (Newsholme 1990). The author proposes that psychological, environmental and/or exercise stress may reduce the glutamine outflux from the muscle. Decreased motor unit recruitment and increased levels of fatigued muscle fibres at rest in the overtrained state may therefore result in depressed immune function due to a decreased availability of glutamine for glutaminolysis within otherwise immunocompetent cells (Newsholme 1990). Recently Parry-Bilings et al. (1990) have extended these concepts to involve central fatigue and the overtraining syndrome.

9.2 Cathecholamines and Immune Regulation in Overtraining

Cathecholamines stimulate adenyl cyclase which, in turn, modifies endogenous levels of cyclic adenosine monophosphate (cAMP). It is known that the intracellular levels of cAMP in regulating immune function (man 1979). Both enhance immunological function 1 cathecholamines in vitro s et al. 1988). Cathecholamines act on lymphocytes by its activity, leading to an increase in the production of interferon-γ, which can inhibit the replication of certain viruses.
Detailed discussions of immunoregulation can be found in the literature (Khansari et al. 1990; Ket et al. 1989) [see fig. 3]. There is a large number of possible mechanisms overtraining may affect the immune system, as suggested by the presence of catecholamines. The rate of glutamine utilisation by lymphocytes has been shown to increase in the absence of catecholamines (Griffiths & Newsholme 1989). Because glutamine is a key amino acid for cell replication and division, and dividing lymphocytes have an increased need for this glutamine, the authors propose that psychological and/or exercise stress might elevate the peripheral metabolism of glutamine. This might result in a decreased availability of glutamine to tissues such as the skeletal muscle. Newsholme states that the muscle-to-blood and muscle-to-cells fluxes of glutamine might be affected by exercise stress. Recently, some studies have suggested that intracellular levels of cAMP are important signals in regulating immune function (Burchiel et al. 1989). The diagram indicates some of the potential connections between the endocrine, nervous and immune systems. Open lines indicate neural connections, black lines indicate hormonal interactions, and broken lines indicate postulated connections for which the effector molecules have not been established.

**Fig. 3. Neuroendocrine interactions with the immune system (Burchiel & Melman 1979).**

The diagram indicates some of the known interactions between the endocrine system and the immune system. Open lines indicate neural connections, black lines indicate hormonal interactions, and broken lines indicate postulated connections for which the effector molecules have not been established.

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Intracellular levels of cAMP are important signals in regulating immune function (Burchiel & Melman 1979). The diagram indicates some of the potential connections between the endocrine, nervous and immune systems. Open lines indicate neural connections, black lines indicate hormonal interactions, and broken lines indicate postulated connections for which the effector molecules have not been established.

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Smith et al. 1977; Webel & Ritts 1977). The in vitro mixed lymphocyte reaction is also reduced by cortisone (Ilfeld et al. 1977; Katz & Fauci 1979). Cupps and Fauci (1982) state that corticosteroids have a generally immunosuppressive effect. Cooper et al. (1987) have demonstrated, on the other hand, enhanced Ig synthesis when low levels of cortisone are added to pokeweed mitogen-stimulated lymphocyte cultures.

In vivo administration of low doses of corticosteroids has induced some suppression of immune functions while greater than physiological concentrations have produced lethal modification to the immune system (Keast 1968, 1969). Administering hydrocortisone, dexamethasone and prednisolone in vivo has been shown to significantly modify the ability of lymphocytes to respond subsequently to both mitogens and by immunoglobulin production in vitro (Fauci 1976; Gillis et al. 1979; Saxon et al. 1978).

Circadian rhythm of the PHA-induced lymphocyte transformation has been shown to coincide directly with changes in plasma cortisol, suggesting a positive effect of cortisol on lymphocyte function at physiological levels (Tavadia et al. 1975). Increased cortisol levels may, at least partly, account for the immunomodulation associated with acute exercise. Changes in the hypothalamic-pituitary axis reported during overtraining may also be manifest in immunomodulation mediated by altered cortisol levels. Much work is required to determine the effects of exercise-induced hormonal modifications on the immune system.

Exercise-induced changes in the concentrations of immunological cytokines such as the interleukins, interferons, prostaglandins or other differentiation, growth or communicational factors may be responsible for exercise-induced immunomodulation. This represents a field of investigation yet to be exploited.

10. Psychology

It may also be difficult to distinguish the effect of exercise on immune function in athletes from that of the psychic stress of competition and train-
mpic and high level sporting or more emotional and psychic demands on them to perform, athletes (Asgeirsson & Bellanti 1984) and the comnsional work and psychic unrestive immunosuppressive effect mmott 1984; Schouten et al. ablished that various types of affect immune function (As-1987; Bartrop et al. 1977; Bor-1982; Fauman 1982; Gruc-1983; Jemmott & usari et al. 1990; Kiecolt-Glaser 1982; Locke et al. 1984; Rog-1983; Solomon et al. 946). Psychological stress can major factor contributing to the lining (Nideffer 1988). O'Brien that overtraining results from hlete either physically and/or at promotes ‘tiredness’ in the been shown to influence the strain and contribute to per-1990). Some emotional fademands of competition, de-failure; setting unrealisticxations of coach or fam-1957).

8) demonstrated that, follow-training volume, athletes expe-1984; Fauman et al. 1988). Based on their study of 100 chronic fatigue syndrome suf-fers, Lloyd et al. (1989) reported that patients with chronic fatigue syndrome appeared to have an impaired immune response. Immunological events identified include atypical lymphocytosis, or lymphopenia in peripheral blood, isolated selective IgG subclass deficiency (IgG1 and IgG3), impaired cell-mediated immunity based on decreased lymphocyte response to mitogen, impaired responses to delayed type hypersensitivity in skin tests, and decreased absolute numbers of T (CD2), T helper/inducer (CD4) and T cytotoxic/suppressor (CD8) lymphocytes. These observations were based on comparison with age- and sex-matched healthy controls (Gin et al. 1989).

11. Biochemistry/Physiology

Causes of decreased efficiency of muscular perform-1986) but could be attributed to incomplete restoration of cellular homeostasis resulting in early fatigue of the motor units normally involved in movements. Additional and therefore less efficient motor units must be recruited resulting in increased oxygen cost, heart rate and ventilation at any given submaximal workload due to less effective summation of forces (Costill 1986; Kuipers et al. 1985; Kuipers & Keizer 1988; Van Handel et al. 1988a). The lower the fractional utilisation of oxygen at any time point, the greater the reserve capacity and it is the fractional utilisation that has been shown to determine the perception of stress and the extent to which anaerobic metabolism has contributed to the total energy demand (Van Handel et al. 1988a). Metabolic efficiency has been shown to vary due to genotype of metabolic regulatory enzymes (Mitton & Grant 1984) and other factors which affect the function of these enzymes (Van Handel et al. 1988a). Several authors report higher heart rates at any given submaximal workload in overtrained subjects (Carlile 1964; Conconi et al. 1982; Costill 1986; Van Handel et al. 1988b) [see fig. 4].

Bouts of endurance (Costill 1986; Costill et al. 1988), sprint (Jacobs et al. 1982) and weight-training (Jacobs et al. 1982; Teich et al. 1986) have been shown to cause significant reductions in muscle glycogen stores which may reduce the capacity of the athlete to perform short term, high intensity work (Jacobs et al. 1982; Pascoe et al. 1990) and