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Overtraining syndrome in horses

CM McGowan[1,*] and DJ Whitworth[2]

[1]The University of Helsinki, PO Box 57, Helsinki 00014, Finland
[2]The University of Queensland, St Lucia, Queensland 4072, Australia
* Corresponding author: catherine.mcgowan@helsinki.fi

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Abstract

Overtraining is a significant cause of poor athletic performance in both human and equine athletes. In humans, overtraining syndrome has been defined as an imbalance between training and recovery manifesting as a syndrome of chronic fatigue and poor performance that may be accompanied by physiological and psychological changes. A similar syndrome has been described in horses using both cross-sectional observations and longitudinal studies with progressively increasing training loads until signs of overtraining were observed. The original cross-sectional studies of overtraining in horses linked the syndrome to increased red cell volume. However, more recent longitudinal studies revealed that overtraining is not always associated with increased red cell volume. Once other causes of poor performance have been ruled out, overtraining syndrome should be suspected in horses with evidence of sustained decreased performance in association with one or more physiological or psychological (behavioural) signs. While no single physiological marker is able to identify the syndrome, accompanying physiological signs in horses may include a decrease in body weight, elevated heart rates during exercise, lower plasma cortisol response to exercise, or elevated muscle enzymes or gamma glutamyl transferase concentrations. Behavioural signs were consistent and an early marker of overtraining syndrome in longitudinal studies in horses, and more research on developing behavioural scores to assist in early detection of overtraining syndrome in horses -as has been achieved for humans- is warranted. Two successful models of overtraining syndrome in horses have been developed, both of which appear to meet the criteria of overtraining syndrome rather than overreaching. Overtraining syndrome in horses is real, reproducible and future research should ensure the criteria for diagnosis of overtraining syndrome are met.

Keywords: overtraining; overreaching; racehorse; exercise; training

Introduction

Overtraining syndrome is a significant cause of poor athletic performance in both human and equine athletes. In humans, overtraining syndrome is defined as an imbalance between training and recovery manifesting as a syndrome of chronic fatigue and poor performance, usually accompanied by physiological and psychological changes[1]. Early studies in horses, in particular Swedish Standardbred trotters[2,3], identified overtraining as a syndrome of poor performance and physiological signs, including diarrhoea. Overtraining was linked to increased red cell volume and altered adrenal cortical function[2,3]. While these studies concentrated on the physiological evidence of overtraining syndrome, psychological signs, such as nervousness, were also observed[4,5]. More recent studies have looked at the endocrinological[6,7], physiological[7,8], heart rate[7] and red cell volume effects[7,9] of overtraining syndrome in horses. These studies revealed that overtraining syndrome was not always associated with increased red cell volume, but occurred in horses with evidence of decreased performance in association with one or more physiological signs, including a decrease in body weight[10] and elevated heart rates during exercise[7]. Also associated with decreased performance was a lower plasma cortisol response to standardized maximal or near-maximal velocity exercise[6,7]. Other signs included increased muscle enzymes or elevated gamma glutamyl transferase (GGT) concentrations[8] and an increase in blood lactate concentration[7]. As in humans, psychological (behavioural), rather than physiological, signs were also a feature in overtrained horses[8]. The aim of this review was to compare and contrast overtraining syndrome in athletic humans and horses, with an emphasis on identification of suitable models of overtraining syndrome and diagnostic markers of overtraining syndrome in the equine athlete.

Overtraining syndrome in humans

Overtraining is a syndrome that has been extensively studied in human athletes[1,11–24] and it occurs when there is an imbalance between training stress and recovery. Overtraining syndrome, must be distinguished from overreaching, which may have some of the signs of overtraining syndrome, including a reduction in performance. While overreaching is an acute maladaptation to an increased training load, taking only a short time for recovery (an average of 2 weeks), overtraining syndrome is a chronic condition that may require many weeks or months for a full recovery[1,21].

In elite human athletes, overload training techniques are commonly employed to optimize performance at the time of competition. This involves progressively increasing training loads followed by appropriate periods of reduced training or tapering prior to competition[25–27]. While it was originally theorized that the principle of overload training was based on a positive period of physiological adaptation (or supercompensation) during the recovery phase, this has been shown to be less important than the reduction of accumulated fatigue associated with training[25]. The tapering period is more important to reduce the negative physical and psychological factors associated with training[25]. When overload training and tapering are poorly managed, overreaching manifest as a performance decrement can result[26,27]. If this is allowed to continue unchecked for a long period, overtraining syndrome can result.

Overtraining syndrome results from a combination of physical and psychological stresses, with emotional stress and anxiety often as important as physical stress[28]. Sources of physical stress are not only limited to training but also include environmental temperature, altitude and improper nutrition[28]. It has been reported that an increase in volume of training is a more potent stressor than that in the intensity of training[17], but increases in the training volume and/or intensity can contribute to overtraining syndrome[29]. Another well-recognized contributing factor is a monotonous routine[29]. The accumulation of these physical and psychological stresses without appropriate recovery periods can result in a state of prolonged fatigue characterized by decreased or stagnant performance despite continued training[16,29].

Overtraining syndrome has been associated with a reduction or stagnation of work capacity, which may be accompanied by a range of endocrinological, metabolic and psychological changes (Table 1). These have included loss of body weight with decreased appetite, muscle tenderness, susceptibility to respiratory and gastrointestinal infections and elevated resting heart rate[1,11–24]. Psychologically, overtrained athletes exhibit symptoms of depression, irritability and anxiety[28]. Markers reflecting the onset of overtraining syndrome

Table 1 Major signs and symptoms of overtraining in humans[1,11–24]

Physiological	Psychological
Decreased performance	Feelings of depression
Prolonged recovery	General apathy
Reduced tolerance of loading	Decreased self-esteem
Decreased muscular strength	Emotional instability
Decreased maximum work capacity	Difficulty in concentrating on work/training
Loss of coordination	Sensitivity to environmental or emotional stress
Decreased efficiency/amplitude of movement	Fear of competition
Reappearance of mistakes/technical faults	Changes in personality
Chronic fatigue	Reduced concentration/increased distractibility
Amenorrhoea	Inability to meet challenges
Headaches	Insomnia +/− night sweats
Nausea	Irritability
Increased aches and pains, muscle tenderness	Biochemical, metabolic and neuroendocrine
Inappetence, gastrointestinal disturbances	Shift of the lactate curve towards the *x*-axis
Cardiovascular and respiratory	Decreased evening post-workout weight/weight loss
Abnormal T-wave pattern on ECG	Elevated BMR
Changes in blood pressure	Decreased body fat
Increased frequency of respiration	Negative nitrogen balance
Changes in heart rate at rest, exercise and recovery	Decreased muscle glycogen concentration
Increased difference between lying and standing heart rate	Hypothalamic dysfunction
Increased O_2 consumption at submaximal workloads	Flat glucose tolerance curves
Haematological and immunological	Decreased body weight
Low plasma glutamine	Decreased bone mineral content and mineral depletion
Increased susceptibility to illnesses/allergy/colds	Increased urea concentrations and increased uric acid production
Decreased functional activity of neutrophils	Elevated cortisol concentrations
Decreased total lymphocyte counts	Elevated ketosteroids in urine
Increased blood eosinophil count	Low free testosterone
Decreased Hb, serum iron concentration and ferritin	Decreased ratio of free testosterone to cortisol of more than 30%
Increased serum hormone-binding globulin	Decreased urinary excretion of catecholamines

have been proposed: these include haematological and plasma biochemical indices, plasma enzyme indices, muscle glycogen concentration and endocrinological abnormalities[14,15,17,22]. Meeusen and colleagues[22] identified a dysfunctional hypothalamic–pituitary axis response to exercise in an overtrained athlete. Blood lactate concentration and heart rate responses during standardized exercise tests are also often considered abnormal in overtrained athletes[14,15]. During submaximal exercise, heart rates are usually elevated and blood lactate concentrations decrease during and after exercise in affected individuals[14,15]. Overtraining syndrome has also been shown to affect oxygen uptake in human athletes. This may be reflected as a higher oxygen uptake at submaximal workloads, due to a higher oxygen cost of exercise[1] and may be associated with unaltered[28] or decreased[11] maximal oxygen uptake.

Muscle damage and loss of strength are also important physiological changes reported in overtraining syndrome in man. Kibler and colleagues[30] defined overtraining syndrome as a series of physiological, biomechanical and anatomical stresses that eventually cause either a relative, or an absolute, force overload on the musculotendinous unit. These authors showed that overtraining syndrome in some individuals may present as lameness or injury causing decreased performance rather than the physiological and psychological aberrations commonly ascribed to the condition[30]. More recently, Fry and colleagues[24] have demonstrated that overtrained athletes suffer a decrease in muscle force and power, probably due to a downregulation of β-2 adrenergic receptors in skeletal muscle.

The variability and inconsistency of the manifestations of overtraining syndrome remain problematic for overtraining syndrome research. One difficulty in the characterization of overtraining syndrome is that much available information has come from cross-sectional studies. Measurements of abnormalities in chronically fatigued, overtrained athletes may be misleading, as the purported abnormality may be a normal or individual physiological response to intense training. In fact, a dependence on the results of cross-sectional studies has often led to erroneous conclusions concerning the pathophysiology of the training response[14].

While it is necessary to conduct longitudinal studies to investigate the normal physiological responses to intense training, there has been a lack of suitable scientific criteria for the diagnosis of overtraining syndrome and the differentiation between overtraining syndrome and overreaching. Two factors are critical to the diagnosis of overtraining syndrome: firstly, a decrease in the level of performance must be measured and, secondly, performance must not have returned to baseline levels after a regeneration period[31]. However, the length and the level of activity permitted during the regeneration period have not been scientifically established. It has also been suggested that an athlete may need to have well-established overtraining syndrome symptoms before a significant performance decrement is experienced[31]. Overtraining syndrome is, therefore, difficult to reproduce in any controlled situation and many longitudinal studies have induced overreaching rather than overtraining syndrome[17,26,27,32,33]. In fact, it has been considered unethical to increase training loads for more than 4 weeks[17,29]. Whether or not results and abnormalities detected from athletes who are experiencing overreaching can be of use in the characterization of overtraining syndrome remains to be elucidated.

Relatively few of the reported physiological variables occur consistently in overtraining syndrome. Consistently, reported changes include performance decrements and reduced ability to perform high-intensity exercise, persistent high fatigue ratings, decreased maximal heart rate, changes in blood lactate variables, such as the blood lactate threshold or blood lactate concentration at maximal exercise and neuroendocrine changes, such as reduced nocturnal excretion of norepinephrine[17,24,29,34]. Overtraining syndrome has several parallels with chronic fatigue syndrome and clinical depression, in particular persistent fatigue, mood state disturbances and muscle soreness[35]. Alterations in psychological variables or mood states, including changes in athletes' self-reported indicators of 'well-being' such as fatigue and quality of sleep, are now accepted as one of the more consistent measures of overtraining syndrome in athletes[12,18,26,27].

While the manifestations of overtraining syndrome are now better recognized, the pathophysiological mechanisms underlying the condition are poorly understood. Overtraining syndrome may be the result of a chronic, systemic inflammatory response to exercise, principally due to repeated trauma or microtrauma to muscles, joints and connective tissues[23,36]. The presence of repeated trauma or microtrauma is supported by Kibler and colleagues[30] and many studies reporting elevated muscle enzymes in athletes with overtraining syndrome[26,32]. The inflammatory cytokine response to such trauma may be responsible for central actions resulting in 'sickness'-type behaviour patterns encouraging rest and recovery[23]. Systemic cytokine stimulation may also be responsible for some of the changes in immune function shown in overtrained athletes[29,34]. Robson[20] proposes that IL-6 may be the specific cytokine responsible. Halson and colleagues[19] found no differences in the levels of IL-6, TNF-α or salivary IgA between control and overreached athletes. Yet in a cross-sectional study of fatigued athletes, decreased IFN-γ function was detected[37]. Further research is warranted to support or refute this potentially unifying hypothesis.

Overtraining syndrome in horses

Racehorse trainers have long used the terms 'overtraining', 'staleness' or 'sourness' to describe a syndrome of poor performance, failure to recover from exercise and prolonged fatigue that does not resolve for weeks or months[38]. Investigation in horses has shown that this syndrome was indeed real and that overtraining syndrome occurs in horses, as it does in humans[3–5,7,10,39]. Similar problems in horses, as have occurred in research in humans, in characterizing overtraining syndrome have been identified. These include difficulties in interpreting data obtained from cross-sectional studies and in being able to differentiate overtraining syndrome from the more acute condition of overreaching that, in contrast to overtraining syndrome, usually resolves within a few days.

Cross-sectional studies

Overtraining syndrome was first reported to occur in racehorses[2–4] based on observations of horses with signs of fatigue and poor performance combined with weight loss, inappetence and signs of psychic stress including tachycardia, nervousness, muscle tremor, sweating and diarrhoea[2,4]. Persson[2] was the first to describe this condition associated with poor performance and red cell hypervolaemia in Swedish Standardbred trotters. He concluded that overtraining syndrome is the most common cause of fading racing performance in Standardbreds in Sweden, and that the severity of signs of overtraining syndrome was correlated with the degree of red cell hypervolaemia[2,3,5]. Persson[2] further observed that overtrained polycythaemic horses had reduced speed at a heart rate of 150 beats per minute (V_{150}). In another study, Persson and colleagues[4] also reported lower plasma cortisol concentrations and a decreased response by the adrenal cortex to adrenocorticotrophic hormone (ACTH). Horses diagnosed with red blood cell hypervolaemia had lower earnings and increased race times in the year of diagnosis compared with their previous performance, although, interestingly, many had been superior athletes prior to diagnosis[40].

A more recent cross-sectional study involved a single evaluation of two groups of Italian Trotters that had differing racing and training frequencies[41]. Horses with more frequent racing showed some mild haematological and biochemical changes, including elevated serum muscle enzymes, elevated red cell number without elevated haematocrit, and elevated total leucocytes. However, this study lacked any objective measures of performance and so overtraining syndrome, or overreaching, cannot be confirmed and provides limited information on the syndrome.

Longitudinal studies

Bruin and colleagues[39] attempted to induce the syndrome of overtraining syndrome in a group of seven normal Standardbred racehorses; however, despite the fact that the definitions of overtraining syndrome and overreaching were stated in the introduction of the paper, a reduction in performance was only evidenced as a failure to complete training sessions, and not recorded during a standardized exercise test. The horses had been training for 187 days at a slowly increasing training load, followed by an increase in intensity until day 261, by which time no abnormal signs were observed in the horses. At day 261, the intensity of the light work days was increased and by day 270 all the horses were unable to complete their training sessions and signs of presumed overtraining syndrome had developed. Three days later they were re-tested for signs of overtraining syndrome and the study concluded. In order for overtraining syndrome to be diagnosed, decreased performance must persist beyond a short period of reduced workload[1]. However, the horses in this study had normal run-times 3 days after the cessation of training at 270 days, thus it appears that the study only induced overreaching in the horses, rather than overtraining syndrome. The study concludes that overtraining is unlikely to occur in the horse as long as 'exhaustive' training is alternated with periods of light exercise, since signs of overtraining were not noted until the horses' 'light' work days had been increased in intensity. They also found that no single variable could be used to detect the early signs of overtraining. However, conclusions made from data generated when there was no reduction in run-time are unlikely to represent overtraining syndrome or, for that matter, overreaching.

Tyler and colleagues[10] were able to successfully induce overtraining syndrome in Australia, again in Standardbred horses, using a controlled longitudinal study design. This was the first study to successfully develop a longitudinal model of overtraining syndrome in horses, and remains as the only controlled study. Training was on an inclined treadmill and included an endurance phase of 7 weeks, training at intensity $\sim$60% VO_{2max}, 5 days per week; a high-intensity phase of 9 weeks of training at $\sim$80% VO_{2max} 3 days per week and 100% VO_{2max} 2 days per week; and an overload training phase. In the overload training phase, control horses continued high-intensity training at a slowly increasing training load, while overload training horses exercised at higher intensities (110% VO_{2max}), more frequently (6 days per week) and for longer durations than control horses, until signs of overtraining syndrome were observed. This occurred in week 31, or after 15 weeks of this phase of training. Overtraining syndrome was defined as a significant decrease ($P < 0.05$) in treadmill run-time during a

standardized exercise test. Horses in the study had both a reduction in run-time to fatigue during a standardized exercise test and a decrease in body weight. Furthermore, the signs persisted after 2 weeks of a reduced training load, confirming that overtraining syndrome, rather than overreaching, had occurred. Subsequently, performance was monitored during a 3-month detraining period and the significant reduction in performance persisted in the overtrained compared with the control group for the duration (Fig. 1)[10]. Horses demonstrated a dramatic reduction in performance, and several physiological, endocrine and behavioural signs accompanying the diagnosis[6,8,9,42].

Hamlin and colleagues[7] in New Zealand were also able to successfully induce overtraining syndrome in Standardbreds in a field situation using a similar increasing training protocol over 34 weeks, although a control group was not used. The final 8 weeks of training were intensified to induce overtraining syndrome. Overtraining syndrome was evidenced as reduced time trial performance, confirmed by continued reduced performance after 2-week recovery. Hamlin and colleagues[7] found a similar set of physiological and endocrine signs in overtrained horses, but Tyler and colleagues[6,8–10] reported no behavioural changes in the overtrained horses, and it is unclear how involved the investigators were in husbandry of their horses.

Physiological signs of overtraining syndrome in horses

Body weight and feed intake

A decrease in body weight was found in both models of overtraining syndrome in horses, by Tyler and colleagues[10] in Australia and by Hamlin and colleagues[7] in New Zealand. The decrease was small and only represented 10–15 kg body weight or around 3% of body weight on average, and therefore required accurate electronic horse scales for diagnosis. The decrease in body weight in the Australian study was accompanied by a decrease in resting glycogen measured prior to training[42]. This decrease was likely to be related to the increased training frequency of 6 days per week in the overtrained horses (compared with 5 days per week in the controls), providing insufficient time for complete glycogen resynthesis[43] since the glycogen utilization rate was non-significantly different between the overtrained and control horses. However, the reduction in resting muscle glycogen may have been at least partly responsible for the reduction in body weight in the overtrained horses, as muscle glycogen increases muscle weight by increasing muscle water content[44].

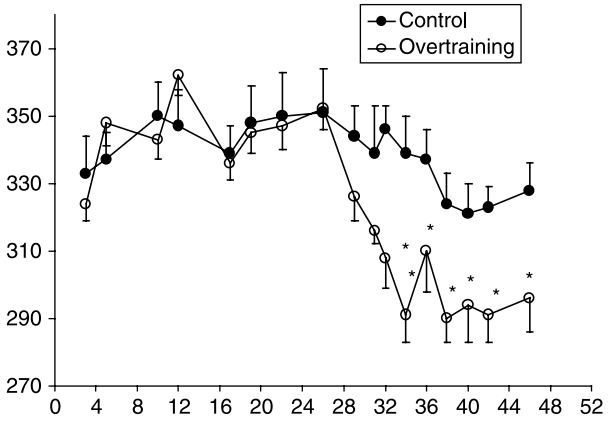

FIG. 1 Run-times (mean ± SEM) for an incremental test to fatigue (VO_{2max} test) during training, overtraining and detraining for six control and seven overtrained Standardbred horses. Overtraining was diagnosed using a significant reduction in run-time in week 31 and was confirmed in week 34. *Significant difference between groups. Note the persistently lowered run-time during the 14 weeks of detraining. Adapted from data in table form presented in Tyler *et al.*[10]

Despite the reduction in body weight, there was no alteration in feed intake in overtrained horses in the Australian study[8], with no alteration in the daily food intake or concentrate:roughage ratio. Feed intake is unlikely to be an accurate marker of overtraining syndrome, since overtraining syndrome is induced by undertaking a greater volume of exercise and hence greater energy requirements are generated, which are met by increasing feed intake. Furthermore, a reduction in feed intake or selective feed intake (e.g. altering the concentrate:roughage ratio) may also be associated with illness, especially gastric ulceration, which is a common phenomenon in racehorses[45].

Haematological findings

Despite the association between red cell hypervolaemia and overtraining syndrome in cross-sectional studies on Swedish Standardbred trotters, this association has not been repeated in longitudinal studies of overtraining syndrome in the same breed of horse[7,9,39]. In Standardbred horses overtrained in the study in Australia, there was actually a significant reduction in measured red blood cell volume and maximal packed cell volume (PCV) in the overtrained horses compared with the controls[9]. In these overtrained horses, the reduction in red cell volume was only weakly reflected as small but non-significant decreases in PCV, red cell number and haemoglobin (Hb) compared with the controls[8]. In the study of overtraining syndrome in Standardbred horses in New Zealand, there was a decrease in maximal PCV in overtrained horses, but no change in red cell volume[7]. However, the authors suggest that technical difficulties in measuring blood volume may account for the incongruity of a decreased PCV and unchanged red cell volume[7]. Decreases in PCV have been reported as consistent in horses with viral respiratory tract disease, the actual decrease in red cell indices merely

indicating the presence of disease and not a reduction in oxygen-carrying capacity[46]. This has been postulated to be due to reduced resting sympathetic activity and relatively more splenic sequestration of erythrocytes[47]. Studies in humans, horses and dogs have demonstrated an increase in plasma volume early in training followed by a gradual increase in red cell volume, which plateaus or decreases over time, such that red cell hypervolaemia is avoided[38]. It is possible that the syndrome of red cell hypervolaemia is unique to Swedish Standardbred trotters and thus it is not seen in other populations of Standardbreds, or that cross-sectional studies have been misleading in equine as they have in human research on overtraining syndrome.

Leukograms have long been used as an indicator of stress in horses. Typically, a 'stress leukogram' consists of an elevated total leukocyte count, with a neutrophilia, lymphocytopaenia and eosinopaenia. In the study by Tyler-McGowan and colleagues[8], there was no evidence of typical stress leukograms, even during the overtraining period. The authors noted an increase in monocytes, a mild increase in neutrophils and a slight decrease in both eosinophils and lymphocytes during training[8]. However, this was possibly associated with prolonged activity of the cortisol response to exercise, as shown by Rose[48] as horses in this study were trained intensively 6 days a week. Eosinophils were the only leukocyte that fell out of normal range (below $0.1 \times 10^9 l^{-1}$), and individual horses that had clinical signs of illness showed an absolute eosinopaenia[8]. Thus, eosinophils may be a more sensitive indicator of training stress than other leukocytes.

In human athletes, intensive training is associated with immune dysfunction and consequent recurrent infections[13,29]. Raidal and colleagues[49] found a significant decrease in the ability of peripheral blood neutrophils to internalize bacteria in horses in the Australian overtraining syndrome study. A similar decrease in the oxidative burst activity of peripheral blood neutrophils, lymphocytes and pulmonary alveolar macrophages was observed[49]. However, there was no significant difference between those horses considered to be showing signs of overtraining syndrome at the end of the 32-week period and those in the control group[49]. Thus, the observed effects on neutrophils, lymphocytes and pulmonary alveolar macrophages appear to be due to prolonged high-intensity training rather than overtraining syndrome. Observed increases in infectious diseases during the overload training period for overtrained horses appear to be more difficult to characterize than have been observed in human athletes[29]. In the overtrained Standardbreds in Australia, there were four cases of various illness (submandibular abscess, nasal discharge, mild diarrhoea and pastern dermatitis) during the final overload phase of training in overtrained horses compared with none in controls, yet no cases in the final 3 weeks of overload training when poor performance was detected[8].

Clinicopathological findings

Serum activity of the muscle enzymes was significantly elevated in overtrained horses in the Australian study[8] and also at the beginning of the intensified training period in the New Zealand study[7]. The increase in serum aspartate transaminase (AST) concentration was interpreted as being due to subclinical muscle damage, and may be representative of delayed onset muscle soreness (DOMS) or low-grade muscle tear. Equine rhabdomyolysis syndrome (ERS), or 'tying-up', can also result in increased serum AST; however, no other clinical signs of rhabdomyolysis were detected in over 31 weeks of training, and increases in muscle enzymes were most prevalent in the final weeks of the study. There were seven cases of muscle enzyme elevations in six overtrained horses during the final 2 weeks of training compared with just one in the previous month and two in the month before that. All cases occurred during the overload training phase in overloaded (eventually overtrained) horses. Further, the increases in creatine kinase (CK) and AST were only moderate (CK 523–1119 $U l^{-1}$, AST 647–4612 $U l^{-1}$), while much higher increases might have been expected had ERS been the cause[8]. Muscle damage can occur in horses that have not overtrained, and over 6% of racing Thoroughbreds have recurrent episodes of rhabdomyolysis[50,51], and so while elevated AST may support a diagnosis of overtraining syndrome, it is not specific for the syndrome. Coutts and colleagues[26] also found increased CK in overreached rugby players and muscle damage associated with training has been studied in man[24,30]. Muscle damage or decreases in muscle strength are of particular interest in the horse, as this may predispose to incoordination and injury, which is one of the most common reasons for lost days to training or racing, or for premature retirement of an animal from competition[52].

GGT is a liver enzyme that appears to be induced with training, although the mechanism for induction is unclear[8]. In Standardbred horses undergoing prolonged training and overtraining, prolonged training resulted in a linear increase in GGT, with this increase being greater in overtrained horses[8]. GGT increased from a mean value of 14 $U l^{-1}$ prior to training (after 4 months of detraining) to a mean of 51 $U l^{-1}$ in the control and 70 $U l^{-1}$ in the overtrained horses after 32 weeks of training. This increase in GGT in the overtrained horses has also been described in some horses with poor performance where very high values of 100 $U l^{-1}$ or more were detected[53]. Whether these high values are associated with overtraining syndrome

or other disease is not known, but it is of interest that those horses with high GGT values showed a markedly reduced incidence of increased GGT following reduction in training intensity in subsequent years[54].

Metabolic responses to exercise

Hamlin and colleagues[7] found a significant decrease in the velocity at a heart rate of 200 bpm (V_{200}) in overtrained, as compared with control, horses, supporting earlier cross-sectional studies[4]. This was also found in the Australian study[55], but had a considerable behavioural component where horses in both the overtrained and control groups would become very excited on the treadmill, waiting for the treadmill to increase in speed and push against the front restraining strap. Due to the labile nature of heart rate, some horses in the warm-up at $4\,m\,s^{-1}$ had a heart rate greater than 200 bpm, and whether this was a behavioural or physiological effect could not be interpreted.

Hamlin and colleagues[7] also reported increased submaximal and maximal lactate concentrations in the overtrained Standardbreds in New Zealand, which were in contrast to reports in humans[17] where decreased lactate concentrations in response to exercise occurred in overreached athletes. Increased blood lactate in response to submaximal exercise was also found in the Australian study[55], but was attributed to the behaviour of the horses, specifically the tendency to push against the front strap during treadmill exercise, rather than physiological effects of overtraining syndrome. Further, in a standardized exercise test to fatigue, post-exercise plasma lactate concentration was decreased in the same horses[42] similar to the finding in human athletes and was most likely as a result of the decreased runtime in the overtrained horses[42].

Endocrine changes

Measurements of the hormonal response to intense exercise may be useful for identifying the overtrained horse. Early cross-sectional studies reported reduced cortisol response to ACTH administration seen with overtraining syndrome and poor performance in racehorses, especially those horses with red cell hypervolaemia[4]. These results imply that racehorses may eventually develop adrenocortical exhaustion with prolonged training stress. However, the concept of adrenal exhaustion has not been supported in longitudinal studies where there was no reduction in cortisol response to administration of ACTH[6]. Overtraining syndrome was associated with a decrease in the cortisol concentration measured after a maximal field exercise test in Standardbred pacers[7]. A decreased cortisol response to maximal exercise in the overtrained state was also found in a treadmill study of Standardbred horses[6]. The mean peak cortisol concentrations after intense treadmill exercise were $320\,nmol\,l^{-1}$ before overtraining, and had decreased to $245\,nmol\,l^{-1}$ when horses were overtrained[6]. However, as performance was also reduced in each instance, the cortisol measured may simply have reflected the reduced overall work effort during the exercise period rather than a physiological variation associated with the syndrome. It is interesting that neuroendocrine changes are less consistent in overtrained horses than in humans, but part of the problem is that testosterone measurement in geldings has been difficult compared with that in male humans due to the lower magnitude of values in castrated horses[56]. More research on urinary catecholamines[17,18] and markers used in human research like growth hormone[22] may prove more rewarding.

Behavioural signs

In the Australian study of overtraining syndrome in horses[8], the only consistent change in overtrained horses, other than the body weight and run-time, was behavioural change. This change occurred from 2 weeks prior to the diagnosis of overtraining syndrome by reduced run-time and may have been contributory to poor performance during training sessions[8]. While the general theme was irritability and an unwillingness to complete training, it was manifest in a variety of ways, including not wanting to get on or off the treadmill, tossing the head while exercising, barging, pushing very hard against the front or back straps of the treadmill and sudden stopping when galloping on the treadmill without warning[8]. Some of the behavioural changes could be attributed to the interval training techniques. However, the behavioural changes occurred in all seven of the overtrained horses and none of the six control horses that were also trained in intervals over the same time period. The other studies of inducing overtraining syndrome[7] or overreaching[39] in horses also demonstrated that behavioural problems in their horses manifested principally as reduced ability to complete or perform during training sessions, while Bruin and colleagues[39] also noted irritability in their treadmill study. It is unclear how much involvement the authors of the New Zealand study[7] had in the day-to-day management of the horses on the track, and as such subtle changes in behaviour may have been missed.

It has been observed in humans that scores of 'well-being' based on subjective ratings of fatigue, stress, muscle soreness and sleep are able to more accurately predict and detect the onset of overtraining syndrome than any physiological markers[13,18,26–29]; further, that overtraining syndrome in humans is also often precipitated by stresses other than from the physiological stresses of training[28,29]. Such stresses can include a monotonous training routine, illness or social stresses and these may well play a part in overtraining

syndrome in horses. A horse in training is exposed to the stresses of intensive housing, absence of foraging and inability to exercise for most of the day and loss of its natural herd structure, all of which may contribute to external stresses[57]. In addition, transport stress, and subclinical or low-grade diseases, such as gastric ulceration, respiratory disease or lameness, might occur, adding additional stresses that could precipitate overtraining syndrome.

Conclusions

Overtraining syndrome is a significant problem in the equine athlete, with striking similarities to the syndrome in humans. Once other causes of poor performance have been ruled out, overtraining syndrome should be suspected in horses with evidence of sustained decreased performance in association with one or more physiological or psychological (behavioural) signs. While a number of physiological signs may occur with overtraining syndrome, including neuroendocrine alterations, and increases in muscle enzymes and GGT, loss of body weight and alterations in heart rate and lactate responses to exercise, these signs may or may not occur in association with performance decrements, and no single marker is consistent enough to accurately predict the onset of overtraining syndrome before performance is adversely affected. Further, it may be difficult to determine which changes are directly associated with the decreased run-time and performance effort and which are associated with true physiological alterations. Behavioural signs were both consistent and an early marker of overtraining syndrome in studies in horses, and more research on developing behavioural scores to assist in early detection of overtraining syndrome in horses as have been achieved for humans, is warranted. Two successful models of overtraining syndrome in horses have been developed[7,10], both of which appear to meet the criteria of overtraining syndrome rather than overreaching. Overtraining syndrome in horses is real, reproducible and future research should ensure the criteria for diagnosis of overtraining syndrome are met.

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