1.2 Overtraining

1.2.1 Overtraining

Overtraining is a popular topic and it is a frequent subject of conversation amongst coaches, trainers, players and scientists. It is also a topic about which people hold strong opinions. When we objectively study the evidence, however, we have to conclude that this is scarce and very few studies have been conducted on the topic (Halson & Jeukendrup, 2004). Although everyone probably has some idea about what *overtraining* means, it is also pretty obvious that people have different ideas about overtraining. People use different definitions, have different opinions about what causes it, how it should be monitored and prevented, and even whether it is a good thing or not. We can find a fairly large number of publications on *overtraining* and even books dedicated to it, but these are often reviews of related articles. Surprisingly, there is little controlled research on this topic.

The term overtraining is used in a variety of ways (Jeukendrup & Gleeson, 2018).

Overtraining is often used as a verb indicating "performing too much training". When athletes say "I am overtrained," and it is used in this context, it simply means, "I have trained too much.".

However, this is not the most common definition used by scientists. Overtraining here is often used to indicate a situation where the athletes suffers from underperformance and there is no obvious reason to explain this. Usually the athlete has tried to recover more with no success and/or to train harder with no success. The underperformance is accompanied by a host of symptoms. These symptoms are diverse and highly individual, meaning that different athletes may show completely different symptoms. Because of the complex nature of the clinical symptoms the term overtraining syndrome is often used. In a review article with the title "Does overtraining exist?" that Dr Shona Halson and I wrote we discussed the defined overtraining or the overtraining syndrome not on the basis of the performance decrement or the symptoms but the duration of the recovery (Jeukendrup, 2015a, https://bit.ly/2S3Ckuf).

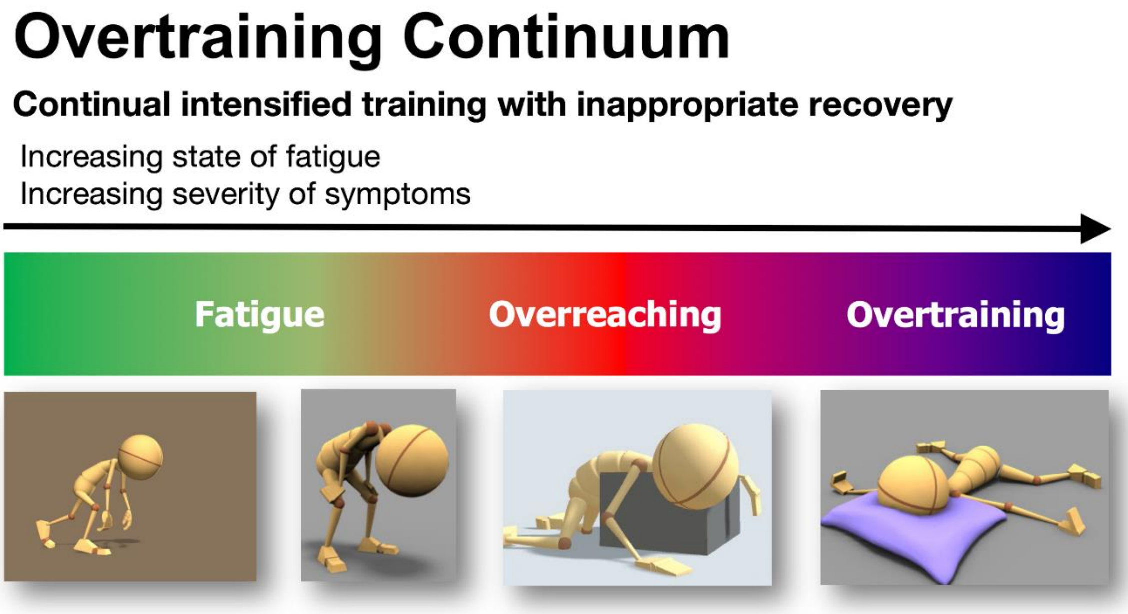
Performance decrements are common, but this can simply be due to fatigue. Or it can be because of extreme fatigue often referred to as overreaching which is observed usually after a block of hard training or a training camp, or in football in periods with more than one match per week. Athletes who come back from a training camp usually perform worse at first, but after sufficient recovery typically see a major jump in their performance as the reward for the hard work during the camp. Overreaching had therefore been described as functional overreaching. (The term functional overreaching was described in a consensus paper (Meeusen, , 2013). Athletes go through a phase like this because it is necessary to improve. (Jeukendrup, 2015a, https://bit.ly/2S3Ckuf).

Below are the definitions of overreaching and overtraining that will be used here:

Overtraining
An accumulation of training-and/or non-training related stress resulting in a long-term decrement in performance capacity, in which recovery of performance capacity may take several weeks or months (Jeukendrup, 2015a).

Thus overtraining seems to be at the far end of a spectrum of different forms of "fatigue" (Figure 3). On one side of the spectrum we find fatigue: a performance decrement which can be reversed with a few hours of rest. Severe fatigue may take 24 or 48 hours to recover from. When this fatigue gets really severe, usually after several days or weeks of training and it may take weeks to recover, we refer to this as overreaching. Typically this is a functional form of overreaching because athletes embark on such a program to cause extreme fatigue with the end goal to improve their performance. On the other end of the spectrum we have the overtraining syndrome that is very difficult to recover from, has a large range of symptoms and is not functional but rather pathological. The overtraining syndrome could mean the end of a season or even the career of an athlete. (Jeukendrup, 2015a, https://bit.ly/2S3Ckuf).

Figure 3: fatigue and overtraining are on different ends of a spectrum of fatigue.



Source: Jeukendrup, 2015a, https://bit.ly/2S3Ckuf

The process by which intensified training and/or limited recovery lead to overreaching or overtraining, is often viewed as a continuum (Fry, Morton, & Keast, 1991; Halson & Jeukendrup, 2004). On the left-hand (or beginning) of the continuum is the acute fatigue that occurs as a result of a single bout of exercise training. When single training sessions are applied repeatedly with appropriate recovery, a positive adaptation and improvement in performance generally occurs. However, if the balance between training and recovery is inappropriate, a state of overreaching may occur (Halson & Jeukendrup, 2004). If intensified training and limited recovery continues for longer periods of time, the most serious state of overtraining may ensue.

As we move from left to right on this spectrum (Figure 3) there is less and less data in the scientific literature to support any theory. This is partly because it is unethical to perform studies that result in long term effects including health effects. As we have discussed in the previous sections, there is a large amount of information about the causes of fatigue, at the muscle level as well as the level of the brain. It was discussed that fatigue is a multifaceted phenomenon and although there still is a lot of debate about the different factors and which factors play the most important role, we have a lot of data on the causes of fatigue. This is not the case with overreaching. There are only a few controlled studies. Many of the studies that have investigated periods of hard training did not measure performance and can therefore not draw any conclusions about overreaching where decreased performance is the key symptom. The well controlled studies can probably be counted on two hands. When we get to overtraining, there really isn't any solid data. There are some case reports and a lot of anecdotal information. Above all, there are a lot of theories. But the nature of the syndrome makes it impossible to study it systematically.

When athletes are asked if they have been overtrained during their career, most athletes will probably say that they have been overtrained at some point in their careers, but was this really overtraining as we defined it here? This is why the title of the paper we wrote was "Does overtraining exist?" It may exist but there is little data to go by. (Halson *et al.*, 2002; Halson & Jeukendrup, 2004; Halson, Lancaster, Achten, Gleeson, & Jeukendrup, 2004; Halson, Lancaster, Jeukendrup, & Gleeson, 2003).

For many years, the prevailing theory was that there were two different types of overtraining: a parasympathetic and a sympathetic form. A instance of hyperactivity was followed by a period of insensitivity. Hypothalamic dysfunction (a disturbance in the normal functioning of the hypothalamus) was believed at the origin of the switch from one form to the other (Barron, Noakes, Levy, Smith, & Millar, 1985).

A brief summary of the way it is often explained is that various stresses (including too much training) bring the body in a state of alertness: stress hormones are high all the time and this can interfere with a number of processes in the body. For example, increased levels of stress hormones (especially cortisol) have been shown to impair immune function and increases in adrenaline (epinephrine), may interfere with normal sleep. This causes the symptoms of the "sympathetic form of overtraining". The body is a constant state of "alertness". When this situation persists for a long period of time the body may respond by becoming less sensitive to these stress signals and this is where full scale overtraining might develop. Symptoms can be very similar between these two forms of overreaching/overtraining, but once the body becomes desensitized to stress signals it will take much longer to recover. This was referred to as the parasympathetic form of overtraining. It has also been suggested that once your body becomes desensitized, the symptoms become less severe, even though this a much more serious and unhealthy situation and thus it becomes much harder to diagnose the underperformance (Jeukendrup & Gleeson, 2018).

1.2.2 Symptoms of Overtraining

It is important to note that the most important symptom of overtraining (or overreaching) is underperformance (Halson & Jeukendrup, 2004).

Without a clear and objective measure of underperformance, overtraining cannot be diagnosed. In football this is sometimes difficult but this stresses the importance to include a battery of tests in the normal training in a standardised way. Some information might be obtained from match analyses but there are of course many variables that influence running and other stats from video analyses and these measurements are never accurate enough to be useful. (Jeukendrup, 2015bhttps://bit.ly/2aiLYGv).

Many published studies that claim to have investigated overtraining did not actually measure performance and therefore had no way of knowing if the athletes in their study were actually overreached or overtrained. In many practical situations this happens as well. Athletes are diagnosed as overtrained but performance could actually be normal. Therefore, this diagnosis is by definition incorrect.

So, the first symptom or cardinal symptom is underperformance (Jeukendrup, 2015a, 2015b).

The second series of symptoms are fatigue related (this is related to performance but more subjective). A third category of important symptoms are disturbances in behaviour and mood. This category is important because these are easy symptoms to pick up, especially for people that surround the athletes. It is common to see athletes become irritable and show signs of depression when training load is dramatically increased. Performance, fatigue and mood state are usually inexpensive to measure and monitor but provide very good indicators of overreaching or overtraining. (Jeukendrup, 2015b https://bit.ly/2aiLYGv).

In addition, physiological measurements like heart rate (especially during sleep) can sometimes be helpful. Blood measurements are often recommended as preventative measures but studies have not shown one clear marker that can predict overreaching or overtraining. The most common markers are testosterone and cortisol and the cortisol/testosterone ratio. CK is often measured as well as

Interleukin 6 or glutamine. However, none of these markers have been shown to be particularly meaningful. (Jeukendrup & Gleeson, 2018).

In the 1990s it was more common to measure various hormones. This could provide evidence for the existence of the sympathetic or parasympathetic form of overtraining. Drug challenge tests were developed where a drug was administered to trigger a hormone response. The idea was that of the hormone response was abnormal this could be an indication of overtraining. These tests were not only impractical (and perhaps unethical), they also did not prove to be very useful. (Jeukendrup, 2015b, https://bit.ly/2aiLYGv).

Monitoring fatigue and symptoms of overreaching may be key to preventing overtraining. A key measurement is performance, which needs to be monitored closely. In addition, it is recommended to measure mood state using simple questionnaires such as the Daily Analysis of Life Demands or a shorter version of the Profile of Mood States. Shorter derivatives of these questionnaires are also likely to be useful but haven't been validated. Sleeping heart rate (or resting heart rate) is another relatively easy and useful measurement. Many blood measurements appear to be a waste of resources.

Finally, in the figure you can see that there are a number of symptoms that are often seen with different levels of severity: sleep disturbances and insomnia, frequents colds that seem to linger and loss of appetite and weight loss. It is obvious that overtraining is a complex phenomenon that affects many systems in the body and therefore results in a very wide range of symptoms. (Jeukendrup, 2015b, https://bit.ly/2aiLYGv).

Some take home messages about overtraining:

- Overtraining (and overreaching) is characterized by a wide range of symptoms.
- The cardinal symptom is reduced performance. Without reduced performance there is no overtraining (or overreaching).
- Overtraining is at the far end of the fatigue continuum.
- Overtraining/overreaching symptoms are HIGHLY individual. Different athletes will show different symptoms with different levels of severity.
- No single symptom is unique to “being overtrained.”
- There may be different forms of overtraining but supporting evidence is scarce at best.
- Some of the symptoms in milder forms may be early indicators of overtraining.
- There is not one indicator that can be measured that will detect overtraining at an early stage. Rather, it is recommended to measure a number of markers, one of which needs to be performance and others could include measures of mood state, and sleeping heart rate. (Jeukendrup, 2015b)

Figure 4: Symptoms of Overtraining

Underperformance is the key symptom. Without underperformance there is no overtraining. The other symptoms can vary considerable between individuals. Source: Jeukendrup, 2015b, https://bit.ly/2aiLYGv

1.2.3 Nutrition to Prevent Overtraining: Carbohydrates

The development of overtraining or overreaching may be affected by nutrition. Inappropriate nutrition may accelerate development, and optimised recovery may prevent, or at least delay overreaching. Inadequate nutrition includes low energy intake, low carbohydrate intake, and generally a low quality diet. These have been associated with an increase in the risk of overtraining. It is therefore also likely that optimising nutrition can at least partly prevent overtraining. On the next section, the role of nutrition in preventing overreaching (and overtraining) will be discussed.

Overreaching and the Role of Muscle Glycogen

As overreaching is thought to be brought about by high intensity and/or high volume of training with limited recovery, it is perceivable that the fatigue and underperformance associated with overtraining is at least partly attributable to a decrease in muscle glycogen levels. No overreaching studies are available in football players and therefore we have to extrapolate from studies in swimmers, cyclists and runners.

One early study (Costill., 1988) investigated this possibility by examining the accumulative effects of 10 days of increased training volume on performance and muscle glycogen levels in swimmers. Of the 12 swimmers who volunteered to take part in this study, 4 were unable to tolerate the increase in training volume (from 4000 meters/day to 9000 meters/day)

The group that was unable to complete the intensified training program, consumed approximately 1000 kcal per day less than their estimated energy requirement and also consumed less carbohydrate than the swimmers that completed the program (5.3 g/kg bw/day versus 8.2 g/kg bw/day). Muscular power, sprint performance and endurance capacity were not affected in the completers and non-completers.

Costill et al. (Costill et al., 1988) concluded that the glycogen levels of the non-completers were sufficient to maintain performance. However, the glycogen levels were inadequate for the energy required during training and thus fatigue resulted. As overreaching and overtraining are primarily defined by a reduction in performance (which was not observed), it is difficult to conclude that the non-completers in this study were overreached (or overtrained).

Snyder et al. (Snyder, Kuipers, Cheng, Servais, & Fransen, 1995) examined the effects of intensified training on performance with increased carbohydrate intake, in an attempt to determine whether overreaching could still occur in the presence of normal muscle glycogen levels. To make sure that the cyclists in this study ingested sufficient carbohydrate, subjects consumed drinks containing 160 g of carbohydrate in the two hours following exercise. Subjects completed 28 days of training: 7 days of normal training, 15 days of intensified training and 6 days of recovery. Resting muscle glycogen was the same during normal training (531 mmol/kg dm) and intensified training (571 mmol/kg dm). Subjects were reported to be overreached and displayed a number of subjective symptoms. It must be noted however that despite all these signs of fatigue, maximal power output during an incremental cycle test was not statistically different after intensified training. Only four of the eight subjects demonstrated a decline in maximal power output as well as an increase in responses to questionnaires about mood disturbance. Therefore, it appears that in this study only half of the subjects could be classified as overreached.

The resynthesis of muscle glycogen after training or competition is undoubtedly one of the most important factors for subsequent running performance. In a study by Costill and colleagues at Ball State University well-trained runners ran 16 km on three consecutive days (Costill, Bowers, Branam, & Sparks, 1971). Muscle glycogen levels decreased from 141 mmol/kg ww after the first run to 73 mmol/kg ww after the third run when a 40-50% carbohydrate diet was consumed. The decrease in muscle glycogen was much smaller when the runners received a high-carbohydrate diet (and in fact, muscle glycogen levels were rather well maintained). (Jeukendrup & Gleeson, 2018).

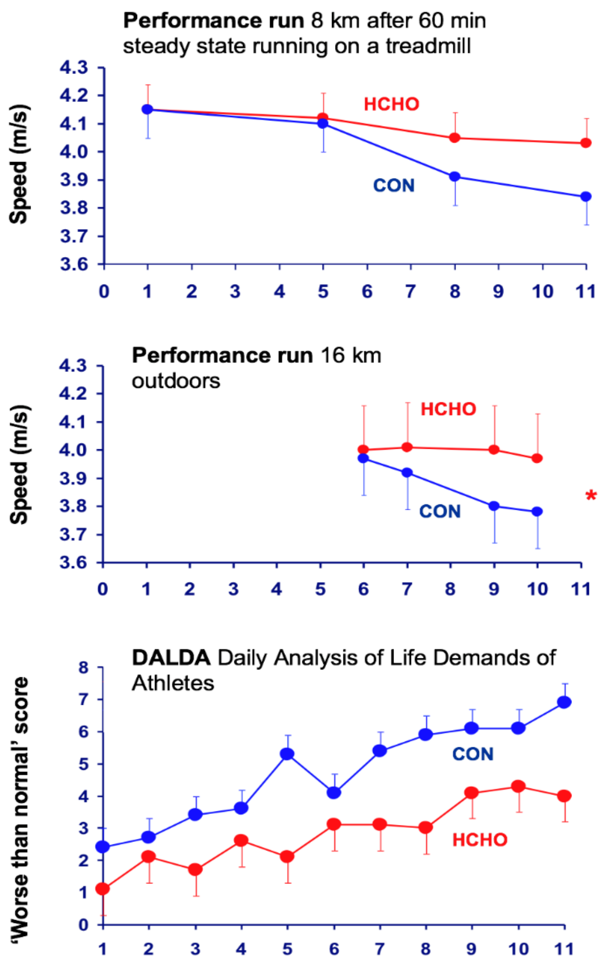
Muscle glycogen

In this section and throughout this course we will express muscle glycogen as it is used in the corresponding papers. The way muscle glycogen is expressed varies which makes it a bit difficult to compare the results of different studies. The units used most frequently are mmol glycosyl (glucose) units per kilogram dry mass or per kg wet mass. The difference is that in order to express the results per kg dry mass the muscle biopsy sample needs to be freeze dried. All the water is removed from the muscle sample by placing the frozen biopsy sample in a freeze dryer. Muscle contains approximately 75-80% water in order to convert values from wet mass to dry mass the concentration needs to be multiplied by 4.5. (Jeukendrup & Gleeson, 2018)

Although insufficient carbohydrate intake (with or without inadequate energy intake) can contribute to the development of an overtraining syndrome, overtraining may also develop when carbohydrate intake is adequate. In a study at the University of Maastricht in the Netherlands, the training intensity and volume of well-trained cyclists was increased for two weeks. All cyclists showed signs of overtraining and were classified as "overreached". The decrease in performance was accompanied by lower heart rates during exercise (time trial) and lower submaximal and maximal plasma lactate levels (Jeukendrup, Hesselink, Snyder, Kuipers, & Keizer, 1992). Lower lactate levels can theoretically be explained by three factors: First, lactate clearance may have been increased. This is unlikely because normal training does not induce such an effect in the same time frame. A second explanation could be a decreased glycogen concentration. When glycogen levels are low, rates of glycolysis are decreased and hence lactate formation is reduced. However, when the study was repeated by the same research group and carbohydrate supplements were provided to avoid a decrease in muscle glycogen breakdown, the cyclists still showed signs of overreaching (Snyder *et al.*, 1995). Submaximal and maximal lactate levels were again lowered, while muscle glycogen levels remained constant. A third explanation of the lower lactates therefore could be a decreased sympathetic drive or a reduced sensitivity of adrenoceptors. This view was put forward by Barron and colleagues (Barron et al., 1985) and can be the result of an increased stress level and increased levels of circulating catecholamines. After a while a downregulation of receptors occurs which results in a decreased sensitivity of the target tissues (e.g. liver, muscles, heart) to catecholamines and a decreased rate of glycolysis, hence, lower plasma lactate concentrations. This is one more factor to take into account when interpreting lactate concentrations measured during training.

Since repeated days of hard training and carbohydrate depletion seem to be linked to the development of overreaching it is tempting to think that carbohydrate supplementation can reverse the symptoms. In a group of runners who ran 16-21 km on a daily basis for 7 days and all these runs were treated as races, performance dropped significantly when a moderate carbohydrate intake of 5.5 g/kg bw/day was maintained (Achten, 2003). The runners also displayed a range of symptoms indicating they were overreached. However, when the daily carbohydrate intake was increased to 8.5 g/kg bw/day the drops in performance were much smaller and symptoms were reduced. In addition, recovery from this week of very hard training was more complete with the high carbohydrate treatment (Figure 5). (Jeukendrup & Gleeson, 2018).

Figure 5: The effects of 10 days of intense training on running performance and mood state (DALDA) with a normal diet (5.5 g/kg carbohydrate/day) or a diet with additional carbohydrates (8.5 g/kg carbohydrate/day).



Source: Achten et al., 2003, https://bit.ly/2DFjVzY

In this study the dietary intake was strictly controlled and the subjects were fed to maintain energy balance. In a follow up study subjects received a carbohydrate supplement but their dietary intake the rest of the day was recorded but not controlled (Halson et al., 2004). A group of well-trained cyclists were required to perform 8 days of intensive endurance training (normal training volume was doubled). This was performed on two occasions separated by a washout/recovery period of at least 2 weeks. On one occasion subjects consumed a 2% carbohydrate solution before, during and after training (moderate carbohydrate) and on the other occasion subjects consumed a 6.4% carbohydrate solution before training and during training and a 20% carbohydrate solution after training (high carbohydrate). The second conditions is more in line with current sports nutrition guidelines. Total carbohydrate intake was 6.4 g/kg bw/day with moderate carbohydrate and 9.4 g/kg bw/day with high carbohydrate. The intensified training protocol induced overreaching as indicated by a decrease in performance (time to fatigue at ~74% VO_2max), although the decrease in performance was significantly less with high-CHO intake, suggesting that high-CHO diets can reduce the severity of overreaching. By forcing the subjects to consume supplements that contained a larger amount of carbohydrate the total energy intake increased as well (4,500 kcal versus 3,300 kcal, respectively). Athletes in hard training seem to reduce their spontaneous food intake and unless supplemented with carbohydrate they may be in negative energy balance during periods of intensified training. Interestingly, it also appeared that the amount of carbohydrate ingested during training influenced the length of time needed for recovery. After two weeks of recovery (reduced volume and intensity) from intensified training with moderate carbohydrate intake performance remained below that of baseline, whereas performance was improved compared with baseline after two weeks of recovery from intensified training with the high carbohydrate intake. Besides carbohydrate depletion, dehydration and a negative energy balance can increase the stress response (increased catecholamines, cortisol and glucagon, while insulin levels are reduced) which increases the risk of developing overtraining. (Jeukendrup & Gleeson, 2018).

Although these studies were not performed in team sports, it is likely that the results can be extrapolated to football. Glycogen concentrations are lowered after intense training days or after matches, and it takes days for glycogen concentrations to recover. It has also been shown that football players tend to eat much less carbohydrate and do not even meet the minimum recommendations of 5 g/kg bw/day. Another factor to consider is that in football the eccentric component of exercise is greater, resulting in a greater extent of muscle damage and inflammation. This appears to negatively affect glycogen restoration after exercise.

Muscle Damage and Glycogen Resynthesis

Repeated days of intensified training (especially running) can lead to muscle damage which can result in a leakage of the glucose transporter GLUT4 out of the muscle cell (Asp, Daugaard, & Richter, 1995). GLUT4 is involved in the transport of glucose across the cell membrane. Exercise and insulin normally result in a translocation of GLUT4 from its intracellular stores to the cell surface, where it will facilitate glucose transport. The number of glucose transporters (GLUT4) present at the sarcolemma is believed to be the rate limiting step for glucose transport across the cell membrane. Glucose transport, in turn, has been suggested to be an important step for glycogen resynthesis (Burke, Van Loon, & Hawley, 2017; Murray & Rosenbloom, 2018). It is therefore feasible that exercise induced muscle damage as seen with overtraining may result in an impaired ability to restore muscle glycogen. Although this is an attractive theory, the same authors did not confirm this in a study with marathon runners (Asp, Rohde, & Richter, 1997). If indeed it is the muscle damage itself that causes the reduction in glycogen resynthesis after eccentric exercise, there may not be much we can do. However, if it is related to inflammation and the secondary processes of muscle damage then it is likely that nutritional interventions can play a role in speeding up the process.

Here we have established that during periods of intensified training it is extra important to pay attention to carbohydrate intake. Insufficient intake may result in fatigue, overreaching, and even overtraining. What is adequate will depend on the intensity and the volume of training, and this will be discussed in more detail in a section on the practicalities of restoring muscle glycogen as part of a recovery protocol. In that section, the best strategies to synthesise glycogen will also be discussed.

1.2.4 Nutrition and Overtraining: Other Nutrients

Glutamine, Overtraining and the Immune System

Newsholme and colleagues suggested that hard training and overtraining result in a decreased glutamine concentration in the blood (Newsholme, Acworth, & Blomstrand, 1987; Newsholme, Parry-Billings, McAndrew, & Budget, 1991). When the glutamine concentration decreases below a critical level, this could result in immunodepression (glutamine is an important fuel for the immune system). On the basis of these thoughts it is often claimed that glutamine supplements would help reduce immunodepression after strenuous exercise or periods of hard training. However, there is little evidence to support this notion. Several studies could not find decreased glutamine levels after strenuous training and supplementation studies did not improve markers of immune function. In addition, it is unclear whether plasma glutamine levels provide reliable information about body glutamine stores, since 90% of all glutamine is present in the muscle and very small amounts in plasma. Therefore, at present, there is little reason to advise athletes to take glutamine supplements to improve immune function or prevent overtraining.

Branched Chain Amino Acids

In 1987, another hypothesis was launched by Professor Eric Newsholme in which the amino acid tryptophan was associated with central fatigue (Newsholme, 1991, p.212). Tryptophan is the precursor of 5-hydroxytryptamine (5-HT or serotonin) in the brain. Only about 10% of the plasma tryptophan is in the free form and there is evidence to suggest that only this fraction is available for uptake by the brain. The remainder of the tryptophan is bound to plasma albumin. The binding sites on albumin, however, are shared with the fatty acids. During exercise, fatty acids are mobilised from adipose tissue and via the plasma transported to the muscle to serve as fuel. The plasma fatty acid concentration will gradually increase and compete for the same binding sites on albumin as tryptophan. The higher the fatty acid concentrations, the more tryptophan will be prevented from binding to albumin or will be removed from binding sites by fatty acids. Therefore, the free tryptophan concentration in the blood will rise. Simultaneously, the oxidation of the branched chain amino acids (BCAA) leucine, isoleucine and valine in muscle will increase during prolonged exercise (Wagenmakers, Brookes, Coakley, Reilly, & Edwards, 1989) resulting in a decrease of the concentrations of the BCAA in the blood. The free tryptophan/BCAA ratio will increase substantially. Since BCAA and tryptophan compete for carrier-mediated entry into the central nervous system by the large neutral amino acid (Wagenmakers, A. J. M., 1999, https://bit.ly/2Fun7AL) (LNAA) transporter, the increase in this ratio would lead to increased tryptophan transport across the blood- brain barrier (Chaouloff, Kennett, Serrurrier, Merino, & Curzon, 1986).

Once taken up, conversion of tryptophan to 5-HT would occur and lead to a local increase of this neurotransmitter. This increase indeed has been found in certain brain areas in the rat, but it has not been established whether it also occurs in humans. (Wagenmakers , 1999, https://bit.ly/2Fun7AL)

According to the 'central fatigue hypothesis' the increase in serotoninergic activity would subsequently lead to central fatigue, forcing athletes to stop exercise or reduce running or cycling speed. Several studies have shown that serotonin plays a role in the onset of sleep and that it is a determinant of mood and aggression. It is uncertain, though, that it also could play a role in the perception of fatigue during prolonged exercise. Prof. Newsholme and colleagues also suggested that overtraining can lead to chronically elevated fatty acids levels and a chronically elevated free tryptophan/BCAA ratio. According to the hypothesis this would lead to increased 5-HT concentrations in the brain and it has been used to explain some of the (central) fatigue symptoms of overtraining. (Committee on military nutrition research, 1999).

One of the implications of the “central fatigue hypothesis” is that ingestion of BCAA, which compete with tryptophan for transport into the brain could reduce the exercise-induced increase of tryptophan uptake by the brain and thus delay fatigue. Another implication is that ingestion of tryptophan prior to exercise would reduce time to exhaustion. (Wagenmakers, 1999, https://bit.ly/2Fun7AL).

The effect of BCAA ingestion on physical performance was investigated for the first time in a field test by Blomstrand et al (Blomstrand, Hassmen, Ekblom, & Newsholme, 1991). Almost 200 male subjects were studied during a marathon in Stockholm. The runners were divided into an experimental group receiving 16 g of BCAA in plain water during the race and the placebo group receiving flavoured water. (Wagenmakers, 1999, https://bit.ly/2Fun7AL)

The subjects additionally had ad libitum access to carbohydrate (CHO)-containing drinks. No difference was observed in the marathon time of the two groups. However, when the original subject group was divided into groups of fast and slower runners, then a small significant reduction in running time during the second half of the marathon was observed in the slower runners only. (Committee on military nutrition research, 1999,)

This study had several experimental flaws and retrospectively this first study has been the one and only study claiming a positive effect of BCAA ingestion during exercise. Varnier et al. (Varnier *et al.*, 1994) investigated 6 moderately trained subjects after glycogen depleting exercise followed by overnight fasting. Subject were investigated the following morning during graded incremental exercise to exhaustion and received an intravenous infusion of BCAA (260 mg/kg/h for 70 min) or saline only. No significant differences were observed between the tests in total work performed. Blomstrand et al. (Blomstrand, Andersson, Hassmen, Ekblom, & Newsholme, 1995) also investigated performance in the laboratory in five male endurance-trained subjects during exhaustive exercise on a cycle ergometer at a work- rate corresponding to 75%VO_2max after reduction of their muscle glycogen stores. During exercise the subjects were given in random order a 6% CHO solution containing 7 g/L of BCAA, a 6% CHO solution and flavoured water. The positive effect of the field test was not confirmed in this controlled laboratory study as no difference in performance was seen when the subjects were given carbohydrate + BCAA or only carbohydrate. (Wagenmakers , 1999, https://bit.ly/2Fun7AL)

Blomstrand et al. (1997) compared flavoured water with a solution of BCAA in seven trained endurance cyclists and did not find an effect on total work performed during a 20 min cycling time trial following I h of exercise at 70%VO_2max.

Madsen and colleagues (1996) investigated performance in 9 trained cyclists in a 100 km time trial in the laboratory. Subjects used their own bike at a freely chosen power output, simulating field conditions, and were studied while ingesting flavoured water only (placebo), a 5% carbohydrate solution (66 gram per h) and carbohydrates (66 gram per h) plus BCAA (6.8 gram per h). There was no difference between treatments in the time needed to finish the 100 km. (Committee on military nutrition research, 1999).

In a well-controlled study, Dr. Van Hall and colleagues (10) studied the effect of BCAA supplementation on endurance performance at 70-75%VO_2max. No differences were found in endurance capacity (time to exhaustion) when a

6% sucrose solution was ingested compared to a 6% sucrose solution in combination with 6g/L BCAA and 18 g/L BCAA. Also, tryptophan supplementation (6% sucrose met 3 g/l tryptophan) had no effect on performance. Other studies came to the same conclusion. The general conclusion therefore is that BCAA supplementation has no effect on performance and although the effect in overtraining has not been directly studied, the efficacy of BCAA feedings should be questioned. A recent short but critical review also questioned the use of the very popular BCAA supplement (Tipton, 2018).

Protein and Overtraining

Few studies investigated the potential effects of protein on symptoms of overreaching. Generally, protein intake is associated with recovery and recognised by athletes as an important factor. However, the effects of protein ingestion are long term effects and therefore more difficult to quantify. Witard, Jackman, Kies, Jeukendrup, and Tipton (2011) investigated the tolerance to intensified training with normal and high protein intake. Trained cyclists completed a week of intensified training preceded by a week of normal training and followed by a week recovery. They were fed a diet containing a normal amount of protein 1.5 g/kg/day or high protein 3 g/kg/day. Dietary carbohydrate content was 6 g/kg/day during both treatments and energy balance was maintained during each training week. Endurance performance was assessed with a VO_2max test and a time trial. Time trial performance was decreased with both diets but the decrease was attenuated with the high protein diet. During the recovery phase performance returned faster with the high protein intake. The reported symptoms of overreaching also appeared to be less. "Therefore, the authors concluded that additional protein intake can reduced symptoms of psychological stress and may result in a worthwhile amelioration of the performance decline experienced during a block of high-intensity training." (Witard et al., 2011)

The same authors also reported that high dietary protein during high-intensity exercise training restores leukocyte trafficking, and aspects of immune surveillance, to levels observed during normal-intensity exercise training. This restoration of immune surveillance with a high protein diet occurred in parallel with a reduced incidence of upper respiratory tract infections in athletes.

In general, good nutrition practices can help to reduce fatigue and overreaching. Therefore, the nutrition guidelines discussed in this course will help towards the prevention of overreaching. It is important to note that overtraining happens when the sum of all stresses exceeds the capacity to recover. This means that efforts should be made to reduce ALL stresses. Players may need support to deal with stress at home or to manage the pressure from fans and the media. Maybe training needs to be adapted. It is important to understand that training is not the only stress for players. On the other hand, when there is little recovery time, recovery strategies have to be used with greater consistency. A much more aggressive approach to recovery is needed when more than one game per week is played. Nutrition plays an extremely important role during these periods.

References

Achten, J., Halson, S. L., Moseley, L., Rayson, M. P., Casey, A., & Jeukendrup, A. E. (2003). Higher dietary carbohydrate content during intensified running training results in better maintenance of performance and mood state. *Journal of Applied Physiology*, *Apr;96(4):1331-40. Epub 2003 Dec 5.*

Asp, S., Daugaard, J. R., & Richter, E. A. (1995). Eccentric exercise decreases glucose transporter GLUT4 protein in human skeletal muscle. *J Physiol., 482*(3), 705-712.

Asp, S., Rohde, T., & Richter, E. A. (1997). Impaired muscle glycogen resynthesis after a marathon is not caused by decreased GLUT-4 content. *J Appl Physiol., 83*(5), 1482-1485.

Bangsbo, J. (1994). Energy demands in competitive soccer. *J Sports Sci., 12 Spec No*, 5-12.

Bangsbo, J., Norregaard, L., & Thorso, F. (1991). Activity profile of competition soccer. *Can J Sport Sci, 16*(2), 110-116.

Bangsbo, J. (2014). Physiological demands of football. Retrieved from https://www.gssiweb.org/sports-science-exchange/article/sse-125-physiological-demands-of-football

Barron, J. L., Noakes, T. D., Levy, W., Smith, C., & Millar, R. P. (1985). Hypothalamic dysfunction in overtrained athletes. *J Clin Endocrinol Metab., 60*(4), 803-806.

Blomstrand, E., Andersson, S., Hassmen, P., Ekblom, B., & Newsholme, E. A. (1995). Effect of branched-chain amino acid and carbohydrate supplementation on the exercise-induced change in plasma and muscle concentration of amino acids in human subjects. *Acta Physiol Scand., 153*(2), 87-96.

Blomstrand, E., Hassmen, P., Ek, S., Ekblom, B., & Newsholme, E. A. (1997). Influence of ingesting a solution of branched-chain amino acids on perceived exertion during exercise. *Acta Physiol Scand., 159*(1), 41-49.

Blomstrand, E., Hassmen, P., Ekblom, B., & Newsholme, E. A. (1991). Administration of branched-chain amino acids during sustained exercise--effects on performance and on plasma concentration of some amino acids. *Eur J Appl Physiol., 63*(2), 83-88.

Bradley, P. S., Di Mascio, M., Peart, D., Olsen, P., & Sheldon, B. (2010). High-intensity activity profiles of elite soccer players at different performance levels. *J Strength Cond Res, 24*(9), 2343-2351. doi:10.1519/JSC.0b013e3181aeb1b3

Burke, L. M., van Loon, L. J. C., & Hawley, J. A. (2017). Postexercise muscle glycogen resynthesis in humans. *J Appl Physiol., (1985), 122*(5), 1055-1067. doi:10.1152/japplphysiol.00860.2016

Carling, C., & Dupont, G. (2011). Are declines in physical performance associated with a reduction in skill-related performance during professional soccer match-play? *J Sports Sci., 29*(1), 63-71. doi:10.1080/02640414.2010.521945

Chaouloff, F., Kennett, G. A., Serrurrier, B., Merino, D., & Curzon, G. (1986). Amino acid analysis demonstrates that increased plasma free tryptophan causes the increase of brain tryptophan during exercise in the rat. *J Neurochem, 46*(5), 1647-1650.

Coggan, A. R., & Coyle, E. F. (1987). Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. *J Appl Physiol., 63*(6), 2388-2395.

Costill, D. L., Bowers, R., Branam, G., & Sparks, K. (1971). Muscle glycogen utilization during prolonged exercise on successive days. *J Appl Physiol, 31*, 834-838.

Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R., & Park, S. H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Med Sci Sports Exerc., 20*(3), 249-254.

Di Mascio, M. & Bradley, P. S. (2013). Evaluation of the most intense high-intensity running period in English FA premier league soccer matches. *J Strength Cond Res., 27*(4), 909-915. doi:10.1519/JSC.0b013e31825ff099

Fry, R. W., Morton, A. R., & Keast, D. (1991). Overtraining in athletes: an update. *Sports Med., 12*(1), 32-65.

Halson, S. L., Bridge, M. W., Meeusen, R., Busschaert, B., Gleeson, M., Jones, D. A., & Jeukendrup, A. E. (2002). Time course of performance changes and fatigue markers during intensified training in trained cyclists. *J Appl Physiol., 93*(3), 947-956.

Halson, S. L. & Jeukendrup, A. E. (2004). Does overtraining exist? An analysis of overreaching and overtraining research. *Sports Med., 34*(14), 967-981.

Halson, S. L., Lancaster, G. I., Achten, J., Gleeson, M., & Jeukendrup, A. E. (2004). Effects of carbohydrate supplementation on performance and carbohydrate oxidation after intensified cycling training. *J Appl Physiol., 97*(4), 1245-1253.

Halson, S. L., Lancaster, G. I., Jeukendrup, A. E., & Gleeson, M. (2003). Immunological responses to overreaching in cyclists. *Med Sci Sports Exerc., 35*(5), 854-861.

Jeukendrup, A. E. (2015a). Overtraining: is it real? Retrieved from http://www.mysportscience.com/single-post/2015/03/02/Overtraining-is-it-real

Jeukendrup, A. E. (2015b). Symptoms of overtraining. Retrieved from http://www.mysportscience.com/single-post/2015/03/03/Symptoms-of-overtraining

Jeukendrup, A. E. & Gleeson, M. (2018). *Sport Nutrition: an introduction to energy production and performance* (3rd ed.). Champaign, IL: Human Kinetics.

Jeukendrup, A., Hesselink, M. K. C., Snyder, A. C., Kuipers, H., & Keizer, H. A. (1992). Physiological changes in male competitive cyclists after two weeks of intensified training. *Int J Sports Med., 13*, 534-541.

Kirkendal, D. (1990). Mechanism of peripheral fatigue. *Med.Sci.Sports Exerc., 22*, 444-449.

Krustrup, P., Mohr, M., Steensberg, A., Bencke, J., Kjaer, M., & Bangsbo, J. (2006). Muscle and blood metabolites during a soccer game: implications for sprint performance. *Med Sci Sports Exerc., 38*(6), 1165-1174. doi:10.1249/01.mss.0000222845.89262.cd

Marqués-Jiménez, D., Calleja-González, J., Arratibel, I., Delextrat, A., & Terrados, N. (2017). Fatigue and Recovery in Soccer: Evidence and Challenges. *The Open Sports Sciences Journal, 10*(Suppl 1: M5), 52-70. doi:10.2174/1875399x01710010052

McKenna, M. J., & Hargreaves, M. (2008). Resolving fatigue mechanisms determining exercise performance: integrative physiology at its finest! *J Appl Physiol (1985), 104*(1), 286-287. doi:10.1152/japplphysiol.01139.2007

Meeusen, R., Duclos, M., Foster, C., Fry, A., Gleeson, M., Nieman, D. (2013). Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Med Sci Sports Exerc., 45*(1), 186-205. doi:10.1249/MSS.0b013e318279a10a

Meeusen, R. & Roelands, B. (2018). Fatigue: Is it all neurochemistry? *Eur J Sport Sci., 18*(1), 37-46. doi:10.1080/17461391.2017.1296890

Mohr, M., Krustrup, P., & Bangsbo, J. (2003). Match performance of high-standard soccer players with special reference to development of fatigue. *J Sports Sci., 21*(7), 519-528. doi:10.1080/0264041031000071182

Murray, B., & Rosenbloom, C. (2018). Fundamentals of glycogen metabolism for coaches and athletes. *Nutr Rev., 76*(4), 243-259. doi:10.1093/nutrit/nuy001

Newsholme, E. A., Acworth, I. N., & Blomstrand, E. (1987). Amino acids, brain neurotransmitters and a functional link between muscle and brain that is important in sustained exercise. In G. Benzi (Ed.), *Advances in myochemistry* (pp. 127-138). London, UK.

Newsholme, E. A., Parry-Billings, M., McAndrew, N., & Budget, R. (1991). A biochemical mechanism to explain some mechanisms of overtraining. In F. Brouns (Ed.), *Advances in nutrition and topsport* (Vol. 32, pp. 79-93). Basel, Switzerland: Karger.

Reilly, T., & Thomas, V. (1979). Estimated daily energy expenditures of professional association footballers. *Ergonomics, 22*(5), 541-548. doi:10.1080/00140137908924638

Smith, M. R., Coutts, A. J., Merlini, M., Deprez, D., Lenoir, M., & Marcora, S. M. (2016). Mental Fatigue Impairs Soccer-Specific Physical and Technical Performance. *Med Sci Sports Exerc., 48*(2), 267-276. doi:10.1249/MSS.0000000000000762

Smith, M. R., Zeuwts, L., Lenoir, M., Hens, N., De Jong, L. M., & Coutts, A. J. (2016). Mental fatigue impairs soccer-specific decision-making skill. *J Sports Sci., 34*(14), 1297-1304. doi:10.1080/02640414.2016.1156241

Snyder, A. C., Kuipers, H., Cheng, B., Servais, R., & Fransen, E. (1995). Overtraining following intensified training with normal muscle glycogen. *Med Sci Sports Exerc., 27*(7), 1063-1070.

Sparks, M., Coetzee, B., & Gabbett, J. T. (2017). Variations in high-intensity running and fatigue during semi-professional soccer matches. *International Journal of Performance Analysis in Sport, 16*(1), 122-132. doi:10.1080/24748668.2016.11868875

Tipton, K. (2018). The truth about BCAA. *Mysportscience*. Retrieved from http://www.mysportscience.com/single-post/2017/10/02/The-truth-about-BCAA

Varnier, M., Sarto, P., Martines, D., Lora, L., Carmignoto, F., Leese, G. P., & Naccarato, R. (1994). Effect of infusing branched-chain amino acid during incremental exercise with reduced muscle glycogen content. *Eur J Appl Physiol., 69*(1), 26-31.

Wagenmakers, A. J. M., Brookes, J. H., Coakley, J. H., Reilly, T., & Edwards, R. H. T. (1989). Exercise-induced activation of branched-chain 2-oxo acid dehydrogenase in human muscle. *Eur J Appl Physiol., 59*, 159-167.

Wagenmakers, A. J. M. (1999). Supplementation with Branched-Chain Amino Acids, Glutamine, and Protein Hydrolysates: Rationale for Effects on Metabolism and Performance. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK224625/

Witard, O. C., Jackman, S. R., Kies, A. K., Jeukendrup, A. E., & Tipton, K. D. (2011). Effect of increased dietary protein on tolerance to intensified training. *Med Sci Sports Exerc.*, *43*(4), 598-607. doi:10.1249/MSS.0b013e3181f684c9