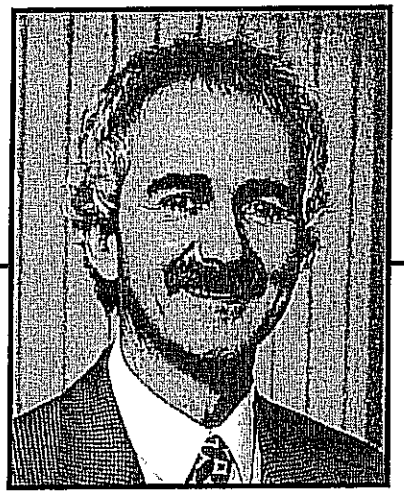

Phytotherapy Review & Commentary

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Treating Immune Suppression and Overtraining Syndrome in Endurance Athletes

by Andrew Grant, Guest Author

This article is written from my clinical experience and observation as a herbalist and as an athlete. The clinical experience has been gained first hand by being an active competitive cyclist and by treating myself after having badly overtrained six years ago. All of my clients are athletes from elite to competitor level of all ages.

The scientific literature on the subject of overtraining syndrome is very incomplete. A part of the reason for this is due to ethics. It is simply not ethical to overtrain an athlete to observe the progression of overtraining symptoms. Currently the medical literature does not distinguish between immune suppression and overtraining syndrome; it is all classified as overtraining syndrome. The current medical treatment for overtraining syndrome is rest. For the purposes of treatment and understanding what is happening with my clients at a physiological level, I separate immune suppression and overtraining syndrome as two distinct categories with the following definitions:

Immune suppression is defined as a reduced immune response to a passing pathogen or being unable to keep systemic viral infections dormant. These pathogens are usually viral and Epstein Barr virus or similar is very common. Immune suppression can be acute or chronic.

Overtraining syndrome is defined as a failure of the athlete's performance to progress, even though an adequate training load is being attempted.

Immune suppression and overtraining syndrome are two distinct syndromes or dysfunctions that can and often do occur together. My clinical observations show that the etiology of these two syndromes may be very similar, but the progression into immune suppression or overtraining syndrome may depend on the status of specific nutrients or constitutional weaknesses.

Common Etiology of Immune Suppression and Overtraining Syndrome

All athletes who present for treatment of immune suppression or overtraining syndrome have a history of training and racing without the use of a quality carbohydrate source and no structured use of carbohydrate-rich foods to promote recovery after training. Many athletes conscientiously calorie-restrict, by training without food or by using carbohydrate-based sports drinks. These athletes believe calorie restriction will reduce body fat. The start of the immune suppression and/or overtraining syndrome can always be traced back to a time of energy deficit, especially of carbohydrate (CHO).

The following abstracts summarize the current research into the immune response following exercise and the effectiveness of using carbohydrate before, during, and after training.

"Many components of the immune system exhibit adverse change after prolonged, intense exertion. During this "open window" of impaired immunity (which may last 3-72 h, depending on the immune measure), viruses and bacteria may gain a foothold, increasing the risk for subclinical and clinical infection. The influence of nutritional supplements, primarily zinc, vitamin C, glutamine, and carbohydrate, on the acute immune response to prolonged exercise has been measured in endurance athletes. Vitamin C and glutamine have received much attention, but the data thus far are inconclusive. The most impressive results have been reported with carbohydrate supplementation. Carbohydrate beverage ingestion has been associated with increased plasma glucose levels, an attenuated cortisol and growth hormone response, fewer perturbations in blood immune cell counts, decreased granulocyte and monocyte phagocytosis and oxidative burst activity, and a diminished pro-inflammatory and anti-inflammatory cytokine response. Overall these data indicate that the physiological stress to the immune system is reduced when endurance athletes use carbohydrate beverages before, during, and after prolonged and intense exertion. The clinical significance of these carbohydrate-induced effects on the endocrine and immune systems awaits further research."[1]

Carbohydrate usage during and after training also has an effect on maintaining plasma glutamine levels, as seen in the following extract.

"The low-CHO diet was associated with a larger rise in plasma cortisol during exercise, a greater fall in the plasma glutamine concentration during recovery, and a larger neutrophilia during the post-exercise period. Exercise on the high-CHO diet did not affect levels of plasma glutamine and circulating leukocytes. We conclude that CHO availability can influence the plasma glutamine and circulating leukocyte responses during recovery from intense prolonged exercise."[2]

Vitamin C status is also affected by intense exercise, possibly under the influence of cortisol. Plasma vitamin C levels were monitored before and after a 21 kilometre running race:

Immune Suppression & Overtraining Syndrome

"The concentration of ascorbic acid (AA) in plasma increased from 52.7 +/- 4.1 mumol/L before the race to 67.0 +/ - 5.3 mumol/L within 5 min after the race ($p<0.001$). This increase in plasma ascorbic acid (AA) concentration was positively correlated with the rise in plasma cortisol concentration during the race ($r = 0.89$; $p<0.01$). However, within 24 h after the race the plasma concentration of ascorbic acid (AA) fell 20 +/- 4% below pre-exercise values ($p<0.01$) and remained low for at least the next 2 days ($p<0.05$)."[3]

The use of carbohydrate during training and racing reduces the rise in plasma cortisol due to training stress and maintains plasma glutamine. Cortisol in turn influences the rise in plasma ascorbic acid during exercise, which is followed by 2 days post-race or training where ascorbic acid levels are lower than normal pre-event levels.

If the athlete has trained or raced hard without a carbohydrate source, the athlete will be depleted in carbohydrate stores (glycogen), have low blood glucose, be depleted in glutamine and ascorbic acid, and have high circulating levels of cortisol. In addition to this, the white cell components of the immune system will be less responsive to viral and bacterial pathogens.

The results from the above abstracts are controlled studies, where recovery has obviously been allowed to happen. Most athletes will train 6 out of 7 days, therefore they are stressing their body again before nutrient levels are back to normal. This is the point where the current research becomes invalid, as it does not accurately reproduce what athletes are doing in training.

Immune Suppression

Acute immune suppression is characterized by frequent and repeated viral infections, usually in the upper respiratory system. This is where the current research and data collection about immune suppression is being focused.

Chronic immune suppression is characterized by *specific but not obvious symptoms*. Chronic immune suppression usually presents as a systemic infection, and not localized to the upper respiratory tract as with an acute infection. These infections are usually viral. Epstein Barr virus (EBV) or similar is very common. The symptoms and clinical presentation of chronic immune suppression in athletes is erratic and inconsistent racing and training form. The inconsistent racing and training form is unrelated to rest or recovery and is coupled with the sensation of being hot without a measurable rise in body temperature. My current speculation, since these symptoms occur together, is that chronic viral infections uncouple oxidative phosphorylation from the citric acid (Krebs cycle). This means that minimal adenosine triphosphate is being produced for muscle contractions and the heat is created by the energy in the citric acid cycle being converted to heat. This is the same mechanism that bears use to hibernate. This symptom picture is very common in athletes, especially endurance athletes. The majority of these athletes have a history of EBV infection and have antibodies to the virus.

The following abstract points to the connection between viral infections and reduced ATP synthesis. "Dysfunctioning of human mitochondria is found in a rapidly increasing number of patients. The mitochondrial system for energy transduction is very vulnerable to damage by genetic and environmental factors. A primary mitochondrial disease is caused by a genetic defect in a mitochondrial enzyme or translocator. More than 60 mitochondrial enzyme deficiencies have been reported. Secondary mitochondrial defects are caused by lack of compounds to enable a proper mitochondrial function or by inhibition of that function. This may result from malnutrition, circulatory or hormonal disturbances, viral infection, poisoning, or an extramitochondrial error of metabolism. Once mitochondrial ATP synthesis decreases, secondary mitochondrial lesions may be generated further, due to changes in synthesis and degradation of mitochondrial phospholipids and proteins, to mitochondrial antibody formation following massive degradation, to accumulation of toxic products as excess acyl-CoA, to the depletion of Krebs cycle intermediates, and to the increase of free radical formation and lipid peroxidation."[4]

A summary of the current research into immune function and exercise shows that regular moderate aerobic exercise stimulates all aspects of immune function. These levels of exercise are less than what is required to be active in competition. Current research into training loads required to prepare for athletic competition suggests that such loads cause transient immune suppression. The duration of the transient immune suppression increases with the volume and intensity of the bout of exercise.

The following is the summary of the effect of vigorous, acute, intense exercise or exercise of long duration on the different components of the immune system. The research demonstrates that the majority of the components of the immune system are suppressed by intense exercise.

Natural Killer (NK) Cells

Immediately following vigorous, but not moderate, exercise, NK cell counts and cytolytic activity usually drop substantially below normal values, but resting function is often restored within a few hours, leaving only a very brief window of opportunity for viruses and neoplastic cells. It is difficult to reconcile a 2- to 3-hour reduction of NK cell activity with the reported 2- to 6-fold increase in the incidence of upper respiratory tract infections (URTIs) in the weeks following participation in a marathon or ultramarathon run.[5] Possible explanations for the suppression of NK cell activity following vigorous exercise have included a lack of IL-2 and an accumulation of prostaglandins (PG).[6,7] The first hypothesis can probably be discounted, since the addition of optimal quantities of IL-2 to isolated NK cells does not restore normal cytolytic activity. Various prostaglandins are released by tissue microtrauma and these substances could inhibit NK cell cytotoxic activity.[8,9]

Macrophage Activity

The activated macrophage is important to early immune defence as an initial phagocytic agent, an antigen-presenting cell, and an initial source of lymphocyte-stimulating cytokines. Cell counts are increased by exercise, but normal values are restored within several hours of ceasing physical activity. Moderate exercise increases the cytostatic activity of macrophages, apparently because their production of tumor necrosis factor (TNF) is increased, but very heavy exercise

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reduces macrophage function. Moderate training has little effect on macrophage function, but heavy training reduces macrophage response to inflammation.[10] Macrophage activity is down-regulated by PGE2, whether the prostaglandin is generated by muscle microtrauma or a tumor.[8]

T cell Counts

Any decrease in CD4+ (T-helper) cell count limits the output of cytokines that activate NK and T cells and stimulate the proliferation and maturation of B cells. An appropriate CD4+/CD8+ (helper/T-suppressor) cell ratio of 1.5 or greater is thus important to immune defences. Both heavy exercise and excessive training can cause this ratio to decrease.[6] Further information is needed regarding the importance of maintaining the absolute CD4+ count relative to that of maintaining a CD4+/CD8+ ratio of 1.5 or more. It also remains unclear to what extent an increase in CD4+ count or CD4+/CD8+ ratio, and thus a greater activation of NK cells, can compensate for a decrease in absolute NK cell numbers (and vice versa).[8]

Proliferative Response

Lymphocyte proliferation, stimulated by CD4+ cell-released IL-2, offers the main long-term defences against both viral infections and neoplastic cells. Heavy physical activity or rigorous training reduces this proliferative response.[6] This reduction sometimes persists for several hours, contributing to the window of opportunity for viruses and neoplastic cells. On the other hand, moderate training reduces the depression of proliferation induced by any single bout of heavy exercise.[8,11]

Impaired Neutrophil Function

Secondary bacterial infections can complicate and prolong URTIs. Circulating neutrophil counts often increase dramatically during and for some hours following exercise, but this does not necessarily increase resistance to secondary infection, since phagocytic activity may simultaneously decrease.[6,12] Intensive training may decrease the oxidative burst associated with bacterial killing in isolated neutrophils, but again, the athlete's susceptibility to respiratory infections is not necessarily affected.[8,13]

Cytokines

Exercise increases production of the cytokine IL-1, and resting levels of this substance may also be augmented by training.[6] IL-1 has a direct cytotoxic effect. It also stimulates the T cells to produce increased amounts of IL-1 and IL-2, augmenting the cytotoxicity of NK and lymphokine-activated killer (LAK) cells. IL-2 has an indirect effect on immune defenses, stimulating the function of NK, LAK, and T cells. *In vitro* studies suggest that exercise decreases free levels of IL-2, possibly by increasing the proportion of lymphocytes that express IL-2 receptors.[6,8]

Interferons slow viral replication. They also alter the surface properties of NK cells and macrophages, with resultant increases in lytic activity.[14] Moderate training may increase interferon (IFN) production, but the output of IFN-± is unchanged by several weeks of exhaustive training.[6] TNF-α is produced by monocytes. It is cytotoxic, stimulating the activity of macrophages and T and B cells. It also contributes significantly to muscle-wasting in cancer. TNF-ß is produced by active T cells. It is both cytostatic and cytotoxic against tumor cells. Acute exercise increases TNF output, but the effect of training is as yet unknown.[8]

Immunoglobulins

Moderate exercise does not change the concentration of salivary lgA or serum IgG. In contrast, very vigorous exercise decreases lgA concentrations in both saliva and nasal washings. One report found low concentrations for 18 hours following a 31-km race.[6] Moderate training increases salivary lgA, but concentrations fall progressively with rigorous training. Partial recovery is seen during pre-competitive tapering. Top competitors also show minor decreases in serum IgG concentrations during peak training.[8]

Decreases in mucosal lgA concentrations could have an important influence on immune defences, since secretory lgA inhibits attachment of the virus to the respiratory epithelium, penetration of epithelial cells and subsequent intracellular replication. Several studies have commented on the coincidence of decreases in salivary IgA and an increased prevalence of URTIs.[8,6]

Studies on Rates of Infection among Athletes

Linde studied URTI over a 1-year period among 44 elite orienteers. A nonathletic group matched for sex, age and occupational distribution acted as control group with regard to disease frequency. The orienteers had 2.5 URTI per year on average against 1.7 in the control group ($p<0.05$). The length of disease was 7.9 days and 6.4 days respectively (which was not significant).[16]

Peters and Bateman carried out a prospective study of the incidence of symptoms of URTI in 150 randomly selected runners who took part in the 1982 Two Oceans Marathon in Cape Town, and compared this with the incidence in individually matched controls who did not run. Runners were questioned on the day before and two weeks after the race. Symptoms of URTI occurred in 33.3% of runners compared with 15.3% of controls and were most common in those who achieved the faster race times.[17]

The largest epidemiological study on exercise and URTI was performed by Nieman and coworkers who researched the incidence of URTI in a group of 2311 marathon runners who took part in the 1987 Los Angeles Marathon race. It was found that 12.9% of Los Angeles Marathon participants reported an infectious episode during the week following the race in comparison to only 2.2% of similarly experienced runners who had applied but did not participate (for reasons other than sickness). Controlling for important demographic and training data by using logistic regression, it was determined that the odds were 6 to 1 in favor of sickness for the marathon race participants versus the nonparticipating runners.[18]

The above studies demonstrate that athletes are very susceptible to infection after strenuous competition or periods of heavy training.

The Clinical Challenges of Treating Athletes

The current research has many design faults and does not replicate with any accuracy what I find in a clinical situation.

The majority of the research is being conducted on one-off post-competition infections. These are acute episodes and usually URTIs. This type of infection will pass within a week and will equate to one week's loss of training. There is usually no lasting performance detriment caused by these infections. All of the studies cited have been conducted by the athlete filling out a questionnaire. There has been no diagnosis or pathological testing by a qualified person to see what pathogen is causing the infection. Athletes who present for treatment due to immune suppression have a latent viral syndrome that is detrimental to their sporting performance. The type of viral infections that these athletes have interferes with the production of ATP. To use cycling jargon, "they go like a train one day and cannot turn the pedals the next." The pattern seems to be that the immune system is suppressed by an intense racing or training effort. This allows the viral activity to increase, which in turn damages or reduces the mitochondrial production of energy. After a few days of low intensity training, immune function returns and suppresses the viral activity. Mitochrondial function is improved, the production of ATP increases and the athlete resumes hard training to repeat the pattern.

The challenge in treating athletes is to keep the athlete's immune function at a level where it suppresses latent systemic viral infections and prevents the local URTIs from establishing. Some new research was published showing that the pressed juice of *Echinacea purpurea* reduced the incidence of URT infection rates among a group of triathletes. The research demonstrated that the Echinacea preparation also induced favorable changes in the athletes' immune systems. This research is valuable in improving our understanding of mechanisms of action of Echinacea.

Details of the study are as follows: The effects of daily oral pretreatment for 28 days with the pressed juice of *Echinacea purpurea* or magnesium supplements on the changes in immunological variables in response to a triathlon sprint were investigated in a randomized, placebo-controlled, parallel group study. Forty-two male triathletes (mean age 27.5 years), undergoing regular training, all with a V02 max > 52 mL/kg/min, were randomized to one of three treatments. Fluorescence activated flow cytometry analysis of blood cell populations, serum and urine levels of interleukin-6 (IL-6) and soluble interleukin-2 receptor (sIL-2R) together with routine sports laboratory, clinical chemical and hematological variables were determined at baseline (day 0), after treatment (day 28) and 1 h and 20 h after the competition (days 29 and 30). Pretreatment with the Echinacea preparation produced changes in total T lymphocytes, NK cells and CD8+ lymphocyte (T-suppressor/cytotoxic cells) counts which remained within the range of baseline variation. In comparison to the placebo group, Echinacea markedly decreased sIL-2R in urine before the competition and enhanced the exercise-induced decrease in serum sIL-2R. It further enhanced the exercise-induced increases in urine IL-6 and serum cortisol. None of the Echinacea-treated athletes developed upper respiratory infections, which were reported by 3 of 13 and 4 of 13 subjects treated with magnesium and placebo respectively (significance not measured). Echinacea appears to reduce sIL-2R release, facilitate IL-6 release in response to exercise and in the present study reduced the documented incidence of respiratory infections, possibly as a result of monocyte/macrophage stimulation.[19]

While this research is a step in the right direction, it was only preventing upper respiratory tract infections. As I stated earlier, the treatment challenge is to maintain an athlete's immunity for 52 weeks of the year, not just for a 28-day period. Hard-learned clinical experience has taught me that fresh plant extracts do not provide the consistent clinical results needed to maintain an athlete's immune status throughout the training and racing season. To achieve consistent results an Echinacea root blend needs to be used. I dispensed 16.485 litres of Echinacea root blend (60% E. *purpurea* root 1:2 and 40% *E. angustifolia* root 1:2) last year with consistent treatment results.

Treating Immune Suppression in Endurance Athletes

The following factors should be considered in the treatment strategy:

1. The majority of immune suppression involves viral infections.
2. They are constitutionally hot to very hot due to chronic viral infections and/or vitamin B5 (pantothenic acid) deficiency. This can also be due to adrenal insufficiency.
3. They go from a chronic state to an acute state extremely rapidly.
4. They can be extreme in their demands on themselves.
5. They do not like to rest. Losing a training session is a crime punishable by repeated repetitions up a hill.
6. They are focused on outcomes (e.g. winning) and relate well to goals and objectives.

When I treat athletes I treat on a body systems basis. In every formula I include herbs to cover the following body systems: immune system, GIT, liver, lower respiratory tract, upper respiratory tract, nervous system. The herbs chosen either have an overlapping immune effect or other protective effect in the body. I use many herbs in a formula at, or near minimum dose. Exercise increases the circulation rate, which increases the distribution of the herbs to the target tissues. Large doses are not needed when treating athletes.

All athletes need some immune stimulation to provide adequate levels of protection against viruses. In every formula I dispense for an athlete, I use a blend of *Echinacea purpurea* and *E. angustifolia* roots. This combination of Echinacea seems to give best results for treating immune suppression.

Athletes usually have poor digestive function due to the blood spending a lot of time in the motor muscles, away from the digestive tract. This coupled with an excessively sweet diet that does not stimulate digestion, leads to a breakdown of the digestive processes. Usually I will use blessed thistle *(Cnicus benedictus)*, as it is a bitter and reputed to be antiviral.

Athletes often have poor liver function. When treating immune suppression I will use greater celandine *(Chelidonium majus)* or *Picrorrhiza kurroa* if the problem is extreme. Picrorrhiza is only used occasionally as the taste is not well tolerated. I think it is the herb of choice with immune suppression, but compliance is a problem.

The lungs of an athlete are sites of potential infection and irritation. Exercise-induced asthma is caused by the drying of the mucous membrane by the constant rush of air in and out ➤

of the lungs. Once the mucous membrane is thinned out, the cells below the membrane dehydrate and shrink. When the cells shrink, it exposes the tight junction between the cells to the air. This in turn triggers the dry cough reflex and bronchospasm. In cycling circles the dry hack is known as "pursuiter's cough." A pursuit is a type of bike race that is 4000 metres long and is very intense. This event places a lot of strain on the lung function.

For maintaining lung mucous membrane function, my favorite 2 herbs are thyme *(Thymus vulgaris)* and sundew *(Drosera longifolia)*. These herbs are protective against and will treat exercise-induced asthma of recent onset. Established asthma is a different story and requires different treatment.

If the athlete presents with regular sore throats, I will alternate between myrrh *(Commiphora molmol)* and marigold *(Calendula officinalis)* for the resin content in the herbs. In chronic EBV type viral syndrome boneset *(Eupatorium perfoliatum)* and poke root *(Phytolacca decandra)* are my herbs of choice.

In immune suppression St John's wort *(Hypericum perforatum)* with high hypericin content is used for its antiviral effect and nervine effect.

A herbal formula for an EBV-type viral syndrome:

Echinacea purpurea and *E. angustifolia* root blend	1:2	15 mL
Blessed thistle *(Cnicus benedictus)*	1:2	20 mL
Phyllanthus *(Phyllanthus amarus)*	1:2	15 mL
Thyme *(Thymus vulgaris)*	1:2	15 mL
Sundew *(Drosera longifolia)*	1:5	10 mL
Boneset *(Eupatorium perfoliatum)*	1:2	15 mL
Poke root *(Phytolacca decandra)*	1:2	2.5 mL
St John's wort *(Hypericum perforatum*)*	1:2	15 mL
		107.5 mL

* High hypericin grade / Dose: 5 mL t.d.s.

This formula can be used in both chronic and acute phases of infection. Many of my clients are on the other side of the world, so changing the formula for an acute stage is not logistically easy, due to the time and cost to ship herbal medicine around the world. Also the formula is predominantly cooling, due to the constitutionally hot nature of the athletes.

When providing a herbal treatment program, I dispense in six-week cycles with documented treatment aims and expected clinical outcomes. These are given to the client so they know what changes they are looking for. Dispensing in six-week cycles is done to avoid getting caught up in clinical "spot-fires" of acute flare-ups and losing sight of the long-term treatment aims.

Case Study: Immune Suppression

M. is a professional cyclist with a European-based professional cycling team. He is a likely contender for the Australian Olympic Road Cycling Team and will ride the Tour de France this year.

He emailed me from Italy in August, saying that he had returned a blood test with active EBV antibodies and that he was sick.

A treatment program was started by email while M. was still in Italy. This started with powdered vitamin C until bowel tolerance was reached. Powdered glutamine at 2 g three times daily was also used. On returning to Australia a full consultation was conducted.

Herbal Treatment

Initial Consultation: Infection started in August 1999. Infection symptoms first appeared after completing a seven-day stage race and a night out to celebrate the completion of the race! Blood test in August showed active EBV antibodies. Presenting symptoms included severe lethargy, being unable to train or race. Swollen lymph nodes. Being hot without a measurable rise in body temperature.

Echinacea purpurea and *E. angustifolia* root blend	1:2	30 mL
Blessed thistle *(Cnicus benedictus)*	1:2	40 mL
Boldo *(Peumus boldus)*	1:2	10 mL
Myrrh *(Commiphora molmol)*	1:5	20 mL
Boneset *(Eupatorium perfoliatum)*	1:2	30 mL
Thyme *(Thymus vulgaris)*	1:2	30 mL
Sundew *(Drosera longifolia)*	1:5	10 mL
St John's wort *(Hypericum perforatum*)*	1:2	30 mL
Poke root *(Phytolacca decandra)*	1:5	5 mL
		205 mL

* High hypericin grade / Dose: 5 mL t.d.s.

Second Consultation (6 weeks later): Response to treatment was slower than anticipated. Steady improvement occurred, and began very light training that does not deplete the body. Still having "bad" days with little energy and not being able to train, indicating that the viral infection was still causing problems with ATP production. EBV antibodies on blood test showed that the infection was not active which did not correlate with the presenting symptoms.

Further questioning revealed that the lethargy symptoms were re-established after attending a friend's wedding and having consumed four beers during the night.

A two-week formula was given to see if Mr M. could tolerate *Picrorrhiza kurroa*.

Echinacea purpurea and *E. angustifolia* root blend	1:2	30 mL
Blessed thistle *(Cnicus benedictus)*	1:2	40 mL
Picrorrhiza *(Picrorrhiza kurroa)*	1:2	15 mL
Myrrh *(Commiphora molmol)*	1:5	20 mL
Boneset *(Eupatorium perfoliatum)*	1:2	25 mL
Thyme *(Thymus vulgaris)*	1:2	30 mL
Sundew *(Drosera longifolia)*	1:5	10 mL
St John's wort *(Hypericum perforatum*)*	1:2	30 mL
Poke root *(Phytolacca decandra)*	1:5	5 mL
		205 mL

* High hypericin grade / Dose: 5 mL t.d.s.
Boneset *(Eupatorium perfoliatum)* dried herb as infusion before bed every night for two weeks.

Third Consultation (2 weeks later): Good improvement, starting to increase training load. One bout of lethargy again, after drinking alcohol at a party. The amount consumed was 4 to 5 beers. Discussed the pattern of the lethargy following the

alcohol ingestion. M's response was that as this is an Olympic year and his first ride in the Tour de France he could live without the alcohol, as his career was more important. Dispensed six months of herbs similar to the mixes listed above, but increased the *Echinacea purpurea* and *E. angustifolia* root blend to 50 mL per bottle to improve the antiviral activity of the herb mixes. Dispensed boneset *(Eupatorium perfoliatum)* dried herb to be used as an infusion at the onset of any signs of lethargy. (The lethargy indicating that the viral infection was being re-established.)

The initial response to the treatment was slower than expected. I think that this was due to EBV infection still being in the acute active phase. This case also demonstrated that presenting symptoms of the patient do not necessarily correlate to the antibody blood test results and that a continuation of the viral symptoms is seen after the blood tests show that the active viral infection has ended.

Overtraining Syndrome

Performance improvement generated by training loads is currently referred to as "adaptation." Adaptation is an undefined term that refers to the successful completion of many different biochemical processes that are stimulated by the stress of training. These processes include improvement in aerobic capacity, muscular protein development, muscular recruitment patterns, and nervous and endocrine response to the physical and mental components of training and competition.

As all of the stimulators and processes of adaptation are not fully understood, it is difficult to understand and assess all of the reasons why athletes fail to adapt. Essentially overtraining syndrome is a failure to adapt to the stress generated by the training loads.

The current scientific literature divides overtraining syndrome into two types. These are based on the presentation of nervous system symptoms of overtraining. I believe that these are in fact stages of overtraining, not distinct syndromes.

1. Sympathetic nervous system-dominant overtraining. This stage of nervous system-mediated overtraining is characterized by high sympathetic nervous system activity. Symptoms include:

- High resting heart rate (all serious athletes use a heart rate monitor and can tell you their resting heart rate).
- Easily irritated or excited.
- Usually have the shakes.
- Very fast gastric transit time, down to eight hours. Stool mass is variable from watery diarrhea to a non-forming mass.
- Heart rate during training is higher for the same perceived effort.

2. Parasympathetic nervous system-dominant overtraining. This stage of nervous system-mediated overtraining is where the parasympathetic system becomes dominant. This is due to a lack of sympathetic nervous system response. Symptoms include:

- Resting heart rate appears normal, but the athlete cannot maintain heart rate at, or near anaerobic threshold. This equates to a loss of heart rate variability. An increase in heart rate is generated by the sympathetic nervous system.
- Unquenchable thirst.
- Easily fatigued during training.
- Loss of blood glucose regulation. Lack of sympathetic response to low blood glucose.
- Spaced out, poor concentration, getting the shakes, all due to low blood glucose.
- Incomplete evacuation of the stool. Usually presenting as passing a bowel motion on waking, then needing another fifteen minutes into training (for morning training).
- Craving caffeine products, which stimulate the sympathetic response.
- Lack of motivation (cannot train a tired body).
- Dizzy or light-headed every time the athlete stands up. This is due to the sympathetic nervous system not adjusting the blood pressure quickly enough.

Using the current research, and observation of athletes, I view nervous system-mediated overtraining as a loss of regulation of the nervous system, with a progression of neurotransmitter depletion. This is a cascading model where one nutrient deficiency, coupled with inadequate rest, leads to another deficiency.

This starts with glutamine deficiency. Glutamine is converted to gamma-amino butyric acid (GABA) in the central nervous system (CNS). GABA is secreted by nerve terminals in the spinal cord, the cerebellum, the basal ganglia, and many areas of the cortex. It is believed always to cause inhibition. GABA is important in sleep onset. It seems to have some regulatory role within the sympathetic nervous system. At the onset of the sympathetic dominant mediated overtraining stage, all athletes have reported sleep onset difficulties and other symptoms associated with glutamine deficiency.

Once the CNS has become deficient in GABA due to low glutamine levels, the sympathetic nervous system-dominant overtraining symptoms become obvious. This is due to a loss of inhibition and regulation of the sympathetic nervous system by GABAminergic neurons. The result of this is seen as high circulating levels of epinephrine and norepinephrine, with the sympathetic nervous system symptoms stated above. The lack of regulation and exaggerated sympathetic response leads to a depletion of phenylalanine and tyrosine, the nutrient precursors for epinephrine and norepinephrine. This phase is accelerated by insufficient protein in the diet.

The depletion of the sympathetic nervous system neurotransmitters gives rise to the symptoms of parasympathetic nervous system-dominant overtraining syndrome, due to the loss of sympathetic nervous system response.

For the basis of treatment, I divide overtraining syndrome into two basic types:

1. Overtraining syndrome created by nutrient deficiencies. These are usually created by training demands, and insufficient dietary intake of a specific nutrient. Examples of nutrient deficiencies that prevent an athlete's performance from progressing include protein, iron, glutamine, carbohydrate and tyrosine.

2. Overtraining syndrome created by body system or tissue dysfunction. The most common noticeable body systems to malfunction are the central nervous system and the adrenal system. ➤

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Treating Deficiency Types of Overtraining Syndrome

Many athletes go "carbo crazy" i.e. eating excessive amounts of carbohydrate and not eating enough protein or fat. Endurance athletes have the highest protein usage of all sports people. This is 1.6 to 2.0 g/kg of body weight. Getting enough quality protein is the athlete's greatest dietary challenge. When the athlete is protein-deficient the blood test will usually show all normal results, but the athlete is struggling for training and racing form. A challenge test for protein deficiency is to use glutamine at one gram three times daily. If the athlete responds it indicates a specific glutamine deficiency as well as a general protein deficiency. The athlete needs to increase the dietary intake of protein.

Iron deficiency in sport can be caused by a number of factors. The most common factors are:

1. Reduced dietary iron intake. This usually occurs on a diet where red meat is excluded.
2. Low hydrochloric acid (HCl) secretion in the stomach. Hydrochloric acid keeps the dietary iron in the absorbable ferrous form. When hydrochloric acid secretion in the stomach is low, the iron converts to the non-absorbable ferric form. This prevents the uptake of dietary iron.
3. Poor bile secretion from the liver. Iron is absorbed in the small intestine after it binds with apotransferrin. The liver secretes apotransferrin into the bile. The apotransferrin binds with free iron, hemoglobin and myoglobin from meat sources to form transferrin. The transferrin is absorbed from the gastrointestinal tract and released into the circulating blood as plasma transferrin.
4. Chronic infection (viral or bacterial) usually leads to the body withdrawing the serum iron in an attempt to starve out the infection.

To improve athletes' iron status, their digestion requires treatment. The stomach HCl secretion needs to be improved with bitters and the bile flow from the liver needs to be improved with liver herbs. It is also important to give a good quality iron supplement with powdered vitamin C in juice at the evening meal.

Treating Nervous System-Mediated Overtraining Syndrome

The following factors should be considered in the treatment strategy:

1. They have *failed to adapt to stimulation.*
2. They are constitutionally hot to very hot, with a temperament to suit.
3. They go from chronic to acute extremely rapidly.
4. They can be extreme in their demands on themselves, and push their bodies beyond the point it can adapt to.
5. Do not like to rest (losing a training session is a punishable crime).
6. Are focused on outcomes (like winning); relate well to goals and objectives.
7. Are *not* robust and tough as perceived. Athletes are not homeostatic, they tend to be steady state. The term homeostasis is defined as the maintenance of a constant or unchanging internal environment. A similar term, steady state, is often used by exercise physiologists to denote a steady physiological environment. Although the terms steady state and homeostasis are often used interchangeably, homeostasis generally refers to a relatively constant internal environment during unstressed conditions resulting from many compensating regulatory responses. In contrast, a steady state does not necessarily mean that the internal environment is completely normal, but simply that it is unchanging. In other words, a balance has been achieved between the demands placed on the body and the body's response to those demands.[21]

When treating athletes, consideration needs to be given to the fact that they are steady state. If using herbs with an action on the nervous system, the action needs to be normalizing or trophorestorative. Using herbs that have a sedative or stimulating action will unbalance the athlete's steady state. Consideration also needs to be given to the fact that they have *failed to adapt to stimulation* and are constitutionally hot. It is important NOT to use tonic, stimulating or heat-producing herbs as these will aggravate the existing condition. These herbs include *Panax ginseng*, *Withania somnifera*, *Eleutherococcus senticosus* and *Glycyrrhiza glabra*. The nervous system has been stressed beyond its ability to cope and is in a dysfunctional state. It should not be further stimulated by giving stimulating tonic herbs. Treatment must start by normalizing the athlete's nervous system.

Nervous system function can be gauged by observing the athlete's heart rate variability. If the athlete's resting heart rate is normal and the athlete is able to sustain the heart rate at anaerobic threshold for extended periods, the nervous system is functioning as it should. Anaerobic threshold is the highest level of intensity, where the body's demand for oxygen is met by the body's delivery of oxygen. This means the body is not accumulating excessive amounts of lactic acid.

If the athlete's resting heart rate is high then the athlete is overtrained or currently has an infection (usually viral). High resting heart rate indicates high sympathetic tone and sympathetic nervous system-dominant overtraining. If the athlete's resting heart rate appears normal, but is unable to sustain the heart rate at anaerobic threshold when training, the athlete is in the parasympathetic dominant stage of overtraining. The resting heart rate appearing normal is usually after a period of high resting heart rate. This indicates the change from sympathetic to parasympathetic dominant overtraining syndrome.

Regardless of what stage of nervous system-mediated overtraining the athlete is in, the trophorestorative nervine herbs work extremely well. Treatment is based on the body systems mentioned above. The herbs chosen either have an overlapping nervine effect or other protective effect in the body.

Herbs used for treatment of nervous system-mediated overtraining

Immune System

A blend of *Echinacea purpurea* and *E. angustifolia* roots – every athlete needs it.

For treating the GIT in this condition, *Matricaria recutita* has good gastric stimulating effect with consistent nervine activity. If the athlete has a fast gastric transit rate, *Viburnum*

opulus is indicated, with psyllium husks/seeds/powder added to the breakfast cereal.

For the liver, *Chelidonium majus* has a GABAminergic action. It also improves bile secretion and leaves a lot of room in the bottle for other herbs.

Thymus vulgaris or *Drosera longifolia* to protect the lung mucous membranes.

To treat nervous system-mediated overtraining the following herbs are used in combination: *Achillea millefolium, Crataegus oxyacantha* (folia), *Hypericum perforatum* (high hypericin content) and *Matricaria recutita* as outlined above.

Herbal formula for nervous system overtraining

Echinacea purpurea and *E. angustifolia* root blend	1:2	15 mL
Chamomile *(Matricaria recutita)*	1:2	20 mL
Boldo *(Peumus boldus)*	1:2	7.5 mL
Thyme *(Thymus vulgaris)*	1:2	15 mL
St John's wort *(Hypericum perforatum*)*	1:2	15 mL
Yarrow *(Achillea millefolium)*	1:2	20 mL
Hawthorn leaves *(Crataegus spp.)*	1:2	15 mL
		107.5 mL

* High hypericin grade / Dose: 5 mL t.d.s.

Herbal formulae that incorporate these nervine herbs have returned nervous system-mediated overtrained athletes back to full training in as little as three weeks. This is after the athlete has had a raised resting heart rate for up to three months. My initial concern was that the herbs were suppressing symptoms. On normalizing the resting heart, full heart rate variability was returned. This indicated a normalizing of nervous system tone and function.

To get consistent results with athletes, their diet and supplementation also need to be addressed. The aim of the diet and supplementation is provide stable blood glucose levels through a program of nutrients, complex carbohydrates and sufficient protein.

Case Study: Nervous system-mediated overtraining

A. presented with inconsistent and erratic form. The treatment regime included increased primary protein intake and re-structured use of carbohydrates.

Initial Consultation: Blood test showed EBV antibodies and iron deficient anemia. Resting waking heart rate 52 - 57 BPM. Diet deficient in protein.

Echinacea purpurea and *E. angustifolia* root blend	1:2	30 mL
Chamomile *(Matricaria recutita*)*	1:2	40 mL
Greater celandine *(Chelidonium majus)*	1:2	15 mL
Yarrow *(Achillea millefolium)*	1:2	40 mL
Marigold *(Calendula officinalis)*	1:2	20 mL
Hawthorn *(Crataegus oxyacantha)*	1:2	40 mL
Nettle *(Urtica diocia)*	1:2	30 mL
		215 mL

* High ±-bisabolol grade / Dose: 5 mL t.d.s.

Second Consultation (6 weeks later): Waking resting heart rate returned to 42 - 47 BPM within the first two weeks. Full heart rate variability was returned, indicating a resolution of the overtraining symptoms. Developed a lung infection.

Echinacea purpurea and *E. angustifolia* root blend	1:2	30 mL
Chamomile *(Matricaria recutita*)*	1:2	40 mL
Fumitory *(Fumaria officinalis)*	1:2	20 mL
Yarrow *(Achillea millefolium)*	1:2	30 mL
Hawthorn *(Crataegus oxyacantha)*	1:2	20 mL
Grindelia *(Grindelia camporum)*	1:2	20 mL
Thyme *(Thymus vulgaris)*	1:2	30 mL
Burdock *(Arctium lappa)*	1:2	20 mL
Poke root *(Phytolacca decandra)*	1:5	1 mL
		211 mL

* High a-bisabolol grade / Dose: 5 mL t.d.s.

A's lung infection cleared and her nervous system continued to adapt to her training loads without any further symptoms of nervous system overtraining.

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Andrew Grant specializes in treating immune suppression and overtraining syndrome in athletes. He has completely automated his herbal practice and is able to do consultations from remote locations. His consultation procedures are specific for athletes.

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