Initial information on the newly discovered hormone leptin suggests a primary role in energy balance and body weight maintenance. Recent published information suggests that leptin has an impact on several physiological systems, including neuroendocrine and immune function, as well as being involved in growth and development. Although the role of leptin in these areas is only partially understood at best, even less is known about the effect of exercise on plasma leptin concentrations. Further, if exercise has an impact on leptin concentration, how then does exercise affect overall leptin function? This article considers leptin function and the impact that exercise has on blood leptin concentrations, and suggests future directions for research on exercise and leptin.

Leptin, a hormone synthesized primarily by adipose tissue and secreted into the circulatory system, is a purported satiety factor with receptors in the hypothalamus. Human leptin is a relatively small protein (16 kDa) which shares a high degree of homology with other species such as mice (84%) and rats (83%). Although rare in humans, leptin mutations in mice result in leptin deficiency and lead to early onset obesity, hyperphagia, and hypothalamic hypogonadism. Unlike leptin deficient mice, humans deficient in leptin do not suffer from hyperinsulinaemia, hyperglycaemia, hypercorticism, or hypothermia. Although regulation of leptin synthesis and release is poorly understood, a distinct circadian pattern for plasma leptin concentrations has been observed and is similar to that of prolactin and thyroid stimulating hormone. Conflictive evidence implicates insulin as a stimulator for leptin release and/or synthesis; however, plasma leptin concentrations may change independently of plasma insulin change. To date, two interventions consistently lower plasma leptin concentrations: dietary energy restriction and a single exercise session. In contrast, tumour necrosis factor α, which is present during immune response to foreign bodies, stimulates leptin release into the circulation.

Although many different factors are involved in leptin regulation, recent evidence suggests that it is stored in vesicles within adipose tissue, and this may complicate any association between plasma leptin concentrations and leptin synthesis regulation. For example, β-3 receptor blockade in adipose tissue decreases leptin mRNA concentrations in adipose tissue when compared with non-blocked control groups, despite unaltered plasma leptin concentrations. Interestingly, plasma leptin concentrations are consistently associated with adiposity in a negative feed back loop (possibly to regulate food intake).

Leptin acts through a membrane bound receptor (Ob-R) identified in hypothalamic and haemopoietic cells. A homodimer, the Ob-R receptor is a member of the class I cytokine receptor family which includes interleukin-6. In the hypothalamus, leptin activated Ob-R receptors are thought to regulate eating behaviour, cortisol release, immune function, and other growth factors—for example, elevated leptin concentration signals adequate nutrition and permits growth and sexual development while maintaining eating behaviour. In haemopoietic cells, however, activated leptin receptors stimulate platelet aggregation possibly leading to accelerated vascular disease. Ob-R mutations are rare in humans, but lead to the same disorders as found in leptin deficient patients.

Studies examining the effects of a single exercise session and plasma leptin have found conflicting results. Dirlewangser et al found no changes in plasma leptin in response to moderate exercise performed during a three day period. Energy intake was kept isocaloric at either 1.3 times basal metabolic rate or increased to meet the energy expenditure induced by the exercise. Perusse et al measured plasma leptin before, after 10–12 minutes of 50 W cycle ergometry, and immediately after reaching maximal exertion. No differences in plasma leptin were found when compared with the baseline value.

In contrast, Essig et al found a 30% reduction in leptin 48 hours after an exercise that required about 6270 kJ (1500 kcal) of energy expenditure. Using the same subjects and experimental design, no differences were found at any time points after an exercise session requiring 3344 kJ (800 kcal) of energy expenditure. Tuominen et al also found a 34% decrease in serum leptin 44 hours after a two hour exercise period performed at 75% V0MAX. Hilton and Loucks examined calorie restriction, exercise, and exercise with calorie restriction. Exercise with calorie restriction was the only treatment that produced a decrease in plasma leptin concentrations 24 hours after exercise. Thong et al found somewhat similar results by inducing weight loss through diet restriction only, exercise only, or exercise without weight loss. The diet only and exercise only protocols both resulted in significant reductions in body weight (7.5 kg) and in plasma leptin (about 5 ng/ml), yet the exercise without weight loss intervention produced no change in plasma leptin concentration.

Longitudinal exercise training studies have also reported conflicting results. Kraemer et al did not find any changes in leptin in obese women after a nine week aerobicics class (estimated energy expended was 1256 kJ per session). Subjects maintained their normal diets throughout the study. Even though physical fitness improved, subject energy expenditure in conjunction with no reduction in energy intake may not have been enough of a stimulus to reduce plasma leptin. Conversely, Gutin et al found decreased plasma leptin in overweight children participating in a four month structured exercise and play programme.

Although existing published information is inconclusive, the results from studies evaluating a single exercise session indicate that leptin concentrations can be reduced in the days after exercise if the exercise session meets an energy expenditure threshold. Results from longitudinal exercise training studies are less clear. If plasma leptin concentrations are to be altered, an undefined threshold for total energy deficit as a result of either exercise training or reduced calorie intake probably exists.

Considering that obesity is a primary health concern and that many people who lose weight regain part if not all of that weight and that leptin in animal models is involved in regulating eating behaviour, understanding the impact of various lifestyle interventions such as exercise on plasma leptin concentrations and the regulation of leptin release and/or synthesis is an important public health concern.
Future exercise research studies should focus on the following areas.

1. Are adverse health consequences associated with elevated plasma leptin concentrations? If so, are the adverse consequences a direct result of leptin concentrations or the result of adiposity or lifestyle behaviour?

2. What are the benefits of lowering plasma leptin concentrations through exercise and/or diet if they are involved in the negative feedback loop regulating eating behaviour?

3. Does a single exercise session alter plasma leptin concentrations directly or are altered plasma leptin concentrations a result of a change in the balance of energy intake and expenditure? Currently, the evidence suggests that the energy balance is more important. However, positive energy balance states have not been tested with or without exercise.

4. What is the mechanism(s) for exercise altered regulation of leptin synthesis and release?

5. What impact does both a single exercise session and habitual exercise participation have on leptin synthesis and/or release, and how does an altered plasma leptin concentration impact on leptin receptor density (Ob-R receptors in the hypothalamus)?

In conclusion, leptin is known to be involved in physical and sexual maturity; however, we do not know whether elevated leptin concentration is a symptom or underlying factor in obesity, nor do we understand how exercise regulates plasma leptin concentrations.

A biomechanical perspective: do foot orthoses work?

Foot orthoses have become an integral part of the treatment of injuries of the foot, ankle, and lower extremity. From a biomechanical perspective, they offer a means of resolving symptoms by placing the foot and the lower extremity in a more advantageous position thus altering applied tissue stresses. Ample evidence exists, based on subjective pain relief and symptom resolution, to support the continued use of these devices. However, scientific evidence to confirm these observations is equivocal.

Research findings

If there is a biomechanical basis for patient improvement, one of many possible kinematic or kinetic parameters should be altered by foot orthoses. Increased magnitude of the pronation angle and increased pronation velocity have been postulated as risk factors for lower extremity injury. A number of investigations have shown the potential of an orthosis with an external medial post to decrease the magnitude of pronation.¹ Not unexpectedly, a decrease in tibial internal rotation has also been shown with medially posted orthoses.² However, Johanson et al³ observed that a non-posted orthotic shell reduced the maximum pronation angle as much as either a forefoot or a rearfoot post, as well as a combination of a forefoot and rearfoot post.

However, attempts to reduce the velocity of pronation through foot orthoses have proved less successful.³ Pronation velocity may be influenced more than the magnitude of the motion by the evasion moment that results from the point of application of the ground reaction force. Investigations showing reduced motion often find no change in pronation velocity. With a restriction to normal pronation, Perry and Lafortune⁴ found no change in impact loading during walking. However, during running, the same pronation restriction produced an increase in impact loading. This suggests that the influence of an orthosis differs between walking and running and should be considered at the time of prescription.

Research contradictions

It appears that for every study showing a positive change in a biomechanical parameter produced by foot orthoses, another study can be cited showing no change. Some of these discrepancies could be due to methodological differences. These include the measurement system, marker placement, skin movement artefact, variable subject/patient groups, lack of statistical power, individual subject response, and the type of orthotic intervention. Reinschmidt et al⁵ showed substantial errors between skin markers and intracortical pins in the frontal and transverse planes (63% and 70% respectively). Advances in measurement technique should resolve some of the contradictions; however, a recent study using intracortical pins⁶ showed that orthotic effects were subject specific and non-systematic across conditions.

Orthotic behaviour is generally assessed using some measure of rearfoot motion to describe the subtalar joint action. Unfortunately there is no direct method to do this. Subtalar and talocrural joint motion can only be inferred from the measures that biomechanists often use. Part of the problem may be that the wrong parameters have been measured or that the changes made by the orthoses are too subtle for the measurement system to detect.

In many studies, the subjects are not patients and therefore may not respond to the orthotic intervention as a patient may. The unimpaired subjects may attempt to override any of the “unnecessary” effect of the orthosis that
Sports medicine in the Netherlands

Sports medicine can be defined in different ways. In the Netherlands the definition of sports medicine, the field of work in sports medicine, and training in sports medicine have changed several times since specific sports medical activities began in the 1920s. The Olympic games in Amsterdam (1928) saw the beginning of specific preventive activities in sports medicine. Presessional screening was established, and after the second world war more than 300,000 preventive presessional screenings were performed a year. Another 200,000 children were screened annually by school doctors.

In 1965 the Netherlands Association for Sports Medicine was established. Doctors interested in sports medical problems could attend a specific course. The programme was broad and offered general topics ranging from cardiology to orthopaedic surgery and exercise physiology. The character of the course was a retraining course. Its duration was about 40 hours and it formed the basis for membership of the Netherlands Association for Sports Medicine.

Ten years later the first doctor was fully trained in sports medicine partly modelled on East European standards. This education took four years and consisted of one year clinical cardiology, one year clinical orthopaedic surgery, one year exercise physiology in a university exercise laboratory, and one year practical work in the field of sports medicine in places such as the national centre for soccer and the national centre for sport (Olympic centre). Beside these training activities, there was a (general) course in social medicine (12 weeks). For the organisation and quality control of this new discipline, a foundation for training of specialists in sports medicine (Stichting Opleiding Sportartsen; SOS) was established. The SOS had several separate sections of the Netherlands Association for Sports Medicine (1982) and wrote a profile “Fields of activity of specialists in sports medicine” (1983). The aims of training for specialisation in sports medicine were formulated on...
Toxocara: dogwalking and playing fields

In villages, towns, and cities throughout the country, land is set aside for human recreation. Public and private playing fields enable the population to indulge in sport and recreation, but these areas are often convenient for another form of human activity: dog walking. Sportsmen may be concerned that toxocaral infection may be acquired from canine defaecation on public playing fields, and the fastidious groundsmen may remove offending articles before matches are played. However, in doing so, they do little to reduce the risk of toxocaral infection.

Life cycle

Toxocara canis is the round worm (ascarid) parasite of canids: dogs and foxes. It has a complex and unusual life cycle. Eggs, when ingested, hatch in the small intestine and invade the intestinal wall. They are taken up in the portal system and distributed through the liver-lung migration cycle, going through larval moults, and being coughed up and swallowed when they develop into adult worms. This characteristic ascarid life cycle is confined mainly to dogs under the age of six months; above this age, larval development is arrested at the second larval stage (L2). This apparent dead end is important for the survival of the parasite as these L2 larvae are re-activated during pregnancy and migrate across the placenta to invade the puppies, and larvae are also excreted in the milk. Consequently, almost all puppies develop an active toxocaral infection. Eggs are also infective for a wide range of mammalian hosts, but larvae are unable to develop beyond the L2 stage and continue to migrate through the body. Toxocara larvae excrete a complex mixture of glycoproteins that are potent stimulators of the host immune system, and it is this that is responsible for the characteristic symptoms and signs of toxocaral infection.

Human disease

Toxocariasis takes three main forms: an occult form characterised by failure to thrive and abdominal pains in children and visceral larva migrans characterised by fever, wheeze, cough, and eosinophilia. In addition, there is an ocular form when trapped larvae stimulate a potent immune response leading to a spectrum of problems including endophthalmitis, uveitis, pars planitis, and subsequent granuloma formation. Ocular toxocariasis is relatively uncommon: some estimates suggest between 50 and 100 new cases of ocular disease a year in the British Isles. In contrast, asymptomatic toxocariasis is common, with 5–7% of an adult population in industrialised countries having evidence of previous infection.

So what are the risks to sportsmen on the playing fields? Firstly, fresh Toxocara infected faeces pose no threat to human health because eggs must embryonate, a process that takes up to one month, and only 6–15% of dogs excrete eggs. The consequences of this are detected in
surveys of parks within cities in the United Kingdom, which indicate that between 5 and 10% of samples will have infective *Toxocara* eggs. Visceral larva migrans peaks in children aged three years and is more common in boys. Ocular disease presents slightly later in life, between 7 and 10 years. Adults appear to be relatively resistant to symptomatic infection—for example, surveys of hydatid control in New Zealanders who have intense contact with dogs show that more than 25% have antibodies to *Toxocara* without any evidence of clinical disease. Thus one would expect the risk for adult sportsmen from contaminated fields to be relatively low, but the risk for children is higher but hard to quantify.

In summary, promiscuous canine defaecation poses a significant aesthetic hazard in playing fields. For children, there is a risk of serious toxocaral infection that may lead to visceral larva migrans or visual loss. For adults, the risk is much lower. Therefore everyone involved in the management of playing fields, whether public or private, should encourage dog owners to be responsible, clear up after their pets, and take steps to enforce this behaviour.

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