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.

J L DURSTINE
R W THOMPSON
K L DROWATZKY
W P BARTOLI

Department of Exercise Science
University of South Carolina
Columbia, SC 29208, USA
ldurstine@sph.sc.edu

1 Sinha MK, Ohannesian JP, Heiman ML, *et al*. Nocturnal rise of leptin in lean, obese, and non-insulin-dependent diabetes mellitus subjects. *J Clin Invest* 1996;**97**:1344–7.
2 Bramlett SB, Zhou J, Harris RBS, *et al*. Does beta-3-adrenoceptor blockade attenuate acute exercise-induced reductions in leptin mRNA? *J Appl Physiol* 1999;**87**:1678–83.
3 Nakata M, Yada T, Soejima N, I Maruyama. Leptin promotes aggregation of human platelets via the long form of its receptor. *Diabetes* 1999;**48**:426–9.
4 Dirlewanger M, Di Vetta V, Giusti P, *et al*. Effect of moderate physical activity on plasma leptin concentration in humans. *Eur J Appl Physiol* 1999;**79**:331–5.
5 Perusse L, Collier G, Gagnon J, *et al*. Acute and chronic effects of exercise on leptin levels in humans. *J Appl Physiol* 1997;**83**:5–10.
6 Essig DA, Alderson NL, Ferguson MA, *et al*. Delayed effects of exercise on the plasma leptin concentration. *Metabolism* 2000;**49**:395–9.
7 Tuominen JA, Ebeling P, Laquier FW, *et al*. Serum leptin concentration and fuel homeostasis in healthy man. *Eur J Clin Invest* 1997;**27**:206–11.
8 Hilton LK, Loucks AB. Low energy availability, not exercise stress, supresses the diunal rhythm of leptin in healthy young women. *Am J Physiol Endocrinol Metab* 2000;**278**:E43–9.
9 Thong FSL, Hudson R, Ross R, *et al*. Plasma leptin in moderately obese men: independent effects of weight loss and aerobic exercise. *Am J Physiol Endocrinol Metab* 2000;**279**:E307–13.
10 Kraemer, RR, Kraemer GR, Acevedo EO, *et al*. Effects of aerobic exercise on serum leptin levels in obese women. *Eur J Appl Physiol* 1999;**80**:154–8.
11 Gutin, B, Ramsey L, Barbeau P, *et al*. Plasma leptin concentrations in obese children: changes during 4-mo periods with and without physical training. *Am J Clin Nutr* 1999;**69**:388–94.

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 al*[1] 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 eversion 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 Lafortune[4] 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 al*[5] 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 pins[6] 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 would force them into a less efficient locomotory pattern. Nawoczenski *et al*[7] observed that different foot structures showed different amounts of frontal and transverse plane motion. Both of these factors could contribute to a range of responses in individual subjects, evident by the observation of internal tibial rotation changing from −80% to +60% with the use of an orthosis.[7]

Further, the foot motion observed may not be dictated primarily by foot structure. Movement patterns of the foot may be driven by (and the effects of orthosis found in) the proximal joints. Bellchamber and van den Bogert[8] calculated a proximal to distal energy flow between the tibia and foot among all subjects during walking and some subjects during running. From the observed direction of energy flow, the authors suggested that the use of foot orthoses may be ineffective in controlling tibial rotation. This conclusion, however, is countered by studies cited above.[7]

Finally, the variation in patients' response to foot orthoses may be largely influenced by the methods used in fitting. Foot orthoses are typically fitted on the basis of a static clinical examination of various measurements of lower extremity alignment. The assumption is that the position of the foot and ankle in the static position reflects the motion of the foot and ankle during ambulation—that is, an increased static pronation angle will produce an increased maximum pronation angle during ambulation. However, much evidence has shown a rather poor relation between static measures and dynamic lower extremity motion.[9] Hamill *et al*[9] showed that various static clinical measures of lower extremity alignment are limited in predicting dynamic lower extremity function. On the basis of similar results, Hunt *et al*[10] questioned the appropriateness of using such measures in prescribing and fitting foot orthoses. If the static measures do not accurately reflect the dynamic motions of the foot and ankle, then designing an orthosis on the basis of these static measures may not provide adequate correction for the dysfunction. Mueller[11] suggested that orthoses should not be prescribed on the basis of specific foot alignment measures, but rather on the patient's symptoms.

Summary

This article is not intended as a comprehensive review of the literature. Rather, its purpose is to bring to the reader's attention several of the key issues involved with foot orthoses and their prescription. In spite of the rather equivocal findings from the numerous investigations involving the efficacy of foot orthoses, their success in reducing pain and symptoms cannot be denied. However, the mechanism by which this is accomplished is certainly open to question. The determination of the mechanism may involve the procedures used to evaluate orthoses biomechanically.

B HEIDERSCHEIT

Osteopathic Medical Center
Des Moines University
Des Moines, Iowa, USA

J HAMILL

University of Massachusetts
jhamill@excsci.umass.edu

D TIBERIO

University of Connecticut

1 Johanson MA, Donatelli R, Wooden MJ, *et al*. Effects of three different posting methods on controlling abnormal subtalar pronation. *Phys Ther* 1994;**74**:149–61.
2 McPoil TG, Cornwall MW. The effect of foot orthoses on transverse tibial rotation during walking. *J Am Podiatr Med Assoc* 2000;**90**:2–11.
3 Rodgers MM, Leveau BF. Effectiveness of foot orthotic devices used to modify pronation in runners. *J Orthop Sports Phys Ther* 1982;**4**:86–90.
4 Perry SD, Lafortune MA. Influences of inversion/eversion of the foot upon impact loading during locomotion. *Clinical Biomechanics* 1995;**10**:253–7.
5 Reinschmidt C, van den Bogert AJ, Nigg BM, *et al*. Effect of skin movement on the analysis of skeletal knee joint motion during running. *J Biomech* 1997;**30**:729–32.
6 Stacoff A, Reinschmidt C, Nigg BM, *et al*. Effects of foot orthoses on skeletal motion during running. *Clinical Biomechanics* 2000;**15**:54–64.
7 Nawoczenski DA, Cook TM, Saltzman CL. The effect of foot orthotics on three-dimensional kinematics of the leg and rearfoot during running. *J Orthop Sports Phys Ther* 1995;**21**:317–27.
8 Bellchamber TL, van den Bogert AJ. Contributions of proximal and distal moments to axial rotation during walking and running. *J Biomech* 2000;**33**:1397–403.
9 Hamill J, Bates BT, Knutzen KM, *et al*. Relationship between selected static and dynamic lower extremity measures. *Clinical Biomechanics* 1989;**4**:217–25.
10 Hunt AE, Fahey AJ, Smith RM. Static measures of calcaneal deviation and arch angle as predictors of rearfoot motion during walking. *Australian Journal of Physiotherapy* 2000;**46**:9–16.
11 Mueller MJ. Invited commentary: effects of three different posting methods on controlling abnormal subtalar pronation. *Phys Ther* 1994;**74**:158–9.

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. Preseasonal screening was established, and after the second world war more than 300 000 preventive preseasonal 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 committees which controlled training content and procedural aspects. Initially, about two doctors started training every year. The specialists in sports medicine set up a separate section 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