Table 1 Years of training for a sports medicine physician including a breakdown of the years in each area of training

Specialty choice	*Years*
Primary care	
Basic education	13
Undergraduate degree	4
Medical school	4
Residency training	3
Fellowship training	1–2
Total	25–26
Orthopaedic surgery	
Basic education	13
Undergraduate degree	4
Medical school	4
Residency training	5
Fellowship training	1–2
Total	27–28

It is hoped that the preceding information will be instructional to those not familiar with our policies and practices in the United States and may help some students interested in sports medicine make educated and directed decisions. The road to becoming a doctor of sports medicine can be a long one (table 1), but for those interested in this field it can be a very fulfilling and lifelong career.

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1 Kaufman A, Mennin S, Waterman R, *et al*. The New Mexico experiment: educational innovation and institutional change. *Acad Med* 1989;**64**:285–94.
2 Neufeld VR, Woodward CA, MacLeod SM. The McMaster M.D. program: a case study of renewal in medical education. *Acad Med* 1989;**64**:423–32.
3 Lombardo JA, Wilkerson LA. Clinical sports medicine training and accreditation: the United States experience [editorial]. *Clin J Sport Med* 1996;**6**:76–7.
4 Henehan M, Jones R. Primary care sports medicine in the managed care environment: coping in today's culture. *Physician and Sports Medicine* 1997; **25**:96–106.

Insulin-like growth factor in muscle growth and its potential abuse by athletes

Skeletal muscle is an inherently plastic tissue. There is evidence to suggest that muscles are constantly adapting both in quantity and quality to the changing functional demands imposed by the types and amounts of physical activity routinely performed. To date, the evidence suggests that, in adults, activity induced adaptations of skeletal muscle are orchestrated by local—that is, tissue level as opposed to systemic—mechanosensitive mechanisms, which appear to include a number of growth factors and hormones. Of particular recent interest is the growth hormone (GH)/insulin-like growth factor-I (IGF-I) system. In the context of skeletal muscle homoeostasis, IGF-I is thought to mediate the majority of the growth promoting effects of circulating GH. In addition, it appears to function in a GH independent autocrine/paracrine mode in this tissue.[1]

As information on the mechanisms that modulate muscle adaptation has been elucidated in the scientific literature, it is tempting for athletes to apply this knowledge to enhance muscle mass and hence function by artificially manipulating these systems. In some cases, this has led to simplistic notions that exogenous anabolic agents can be used to safely and effectively stimulate or augment muscle. Unfortunately, many of these attempts have been unsuccessful, and, in truth, they ignore our understanding of the integrated nature of physiological systems.

The circumstances that militate against this approach are severalfold. The first and most obvious problem with anabolic substances is that they are invariably non-specific. Agents that can stimulate muscle cells to hypertrophy will undoubtedly have effects on other cells and tissues as well—for example, the impact of growth hormone on prostatic hypertrophy. Secondly, just as the body is made up of tissues and organs that function as an integrated whole, so muscle is comprised of a number of different cell types which must also function in unison. For example, a treatment that stimulates muscle cells to hypertrophy must also recruit fibroblasts to strengthen the connective tissues that will transmit the force generated by the muscle cells, and must also act to enhance angiogenesis and mitochondrial function. In the absence of this coordination, one may develop larger (therefore "stronger") muscle cells, but the application of this enhanced contractile function would serve only to damage the structure of the muscle when the unenhanced connective tissue fails.

With regard to manipulating IGF-I either directly or through GH, a number of results from animal studies are instructive. Researchers have long sought ways to mitigate the atrophy inducing effects of unloading on skeletal muscle. An animal model used to study this effect involves "tail suspension" whereby rats are placed in cages with only their front feet touching any surface. This results in muscle atrophy which mimics that seen in humans following space flight. When GH or IGF-I has been supplied exogenously during tail suspension, the results have clearly indicated that the mass of the normally weight bearing muscles was in fact conserved. However, owing to the effects of these treatments on other tissues, the overall body weight of the rats had increased. It was as if the growth and development programme from an earlier developmental stage had been re-activated. However, there was one difference. When compared with their body weight changes, the muscles had actually "grown" less—that is, the normalised muscle mass was less in treated than untreated animals—the end result of course being that the growth factor treated rats would actually be less well adapted to normal ambulatory activity than the rats that received no treatment at all.

In humans, attempts to augment muscle mass using IGF-I have had less dramatic impacts. In studies designed to overcome the loss of muscle in the elderly, the overall impact of experimentally increasing circulating IGF-I levels has been negligible.[9–11] For example, in one study the investigators managed to double the circulating IGF-I levels in elderly subjects but found no effect on the rate of protein synthesis in muscles; nor was there any augmentation of strength.[11] In addition to this disappointing result, the supplementation of IGF-I in otherwise healthy—that is, GH normal—people is associated with (1) moderate to severe hypoglycaemia (it is after all insulin-like),[6] (2) decreased growth hormone secretion,[4 8] (3) a shift from lipid to carbohydrate oxidation for energy,[8] and (4) a general disruption of the insulin/glucagon system.[8 6] The issue of augmenting IGF-I is rendered even more complex

because the biological activity of IGF-I in the body is now known to be substantially influenced by the family of IGF binding proteins.[3] For example, recent work on the effects of hypoxia on rat growth suggests that it is, in fact, the impact of IGF binding protein-3 that is more closely related to overall growth than is IGF-I itself.[7]

There is also a more troubling aspect of IGF-I that has only recently begun to emerge. In addition to a direct anabolic effect on skeletal muscle—for example, the production of more protein—it has become clear that IGF-I is also capable of stimulating the proliferation and differentiation of muscle stem cells (satellite cells). In animal studies, there is evidence to suggest that this process is obligatory for muscle hypertrophy to proceed. However, this evidence that IGF-I is mitogenic should serve as a cautionary note to those who would use this agent to promote an anabolic state. There is increasing evidence to suggest that IGF-I signalling may also participate in cellular transformation.[2] Specifically, elevated IGF-I levels have been linked to prostate, colorectal, and lung cancers.[5]

In the light of the large number of potentially negative impacts, ranging from disruption of the insulin system to cancer, it would seem that the exogenous augmentation of IGF-I does not represent a very attractive or effective method of increasing muscle mass or function. Clearly, the therapeutic use of these powerful growth factors awaits more focused research on the mechanisms through which these mediators actually influence growth in the context of the whole organism.

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1 Adams, G.R. The role of IGF-I in the regulation of skeletal muscle adaptation. *Exerc Sport Sci Rev* 1998;**26**:31–60.
2 Baserga, R. The IGF-I receptor in cancer research. *Exp Cell Res* 1999;**253**:1–6.
3 Baxter, RC. Insulin-like growth factor (IGF)-binding proteins: interactions with IGFs and intrinsic bioactivities. *Am J Physiol* 2000;**278**:E967–76.
4 Carroll PV, Umpleby M, Alexander EL, *et al.* Recombinant human insulin-like growth factor-I (rhIGF-I) therapy in adults with type 1 diabetes mellitus: effects on IGFs, IGF-binding proteins, glucose levels and insulin treatment. *Clin Endocrinol* 1998;**49**:739–46.
5 Grimberg A, Cohen P. Role of insulin-like growth factors and their binding proteins in growth control and carcinogenesis. *J Cell Physiol* 2000;**183**:1–9.
6 Jenkins PJ. Growth hormone and exercise. *Clin Endocrinol* 1999;**50**:683–9.
7 Moromisato DY, Moromisato MY, Brasel JA, *et al*. Effect of growth hormone therapy in mitigating hypoxia-induced and food restriction-induced growth retardation in the newborn rat. *Crit Care Med* 1999;**27**:2234–8.
8 Sherwin RS, Borg WP, Boulware SD. Metabolic effects of insulin like growth factor-1 in normal humans. *Horm Res* 1994;**41**(suppl 2):97–102.
9 Taaffe DR, Jin IH, Vu TH, *et al*. Lack of effect of recombinant human growth hormone (GH) on muscle morphology and GH-insulin-like growth factor expression. *J Clin Endocrinol Metab* 1996;**81**:421–5.
10 Taaffe DR, Pruitt L, Reim J, *et al*. Effect of recombinant human growth hormone on the muscle strength response to resistance exercise in elderly men. *J Clin Endocrinol Metab* 1994;**79**:1361–6.
11 Yarasheski KE, Zachwieja JJ, Campbell JA, *et al*. Effect of growth hormone and resistance exercise on muscle growth and strength in older men. *Am J Physiol* 1995;**268**:E268–76.

Transmission of cutaneous infections in athletes

Myriad dermatoses can affect athletes. One of the most common cutaneous manifestations of athletic activity are skin infections. Bacteria,[1–4] viruses,[1 2 4–8] and fungi[1 2 4 9–11] cause these infections. Many are contagious and may have serious ramifications for team practices and competitions. Knowledge of these infections facilitates implementation of rapid treatment and preventive measures to ensure the least disruption in daily team activities.

Several specific sports related dermatological conditions are caused by bacterial infection. Staphylococcal infection is the most common but streptococcal infection also commonly occurs.[1–4] Both organisms may present as varying clinical entities including impetigo, erysipelas, folliculitis,[1 2 4] and furunculosis.[3] In general, they are probably contagious to some degree. Impetigo, characterised by well defined, erythematous, yellow crusted, scaling plaques, and erysipelas, characterised by well defined, advancing, erythematous plaques, can be treated with topical warm soaks and oral antibiotics.[1 4] Folliculitis presents as small follicular pustules that can be treated with topical or oral antibiotics.[1] These bacterial infections occur in sports in which close personal contact occurs, including rugby, judo, and wrestling.[2 4] Furunculosis outbreaks, however, have been noted also in football and basketball athletes. One study showed that 25% of high school athletes in these sports developed furunculosis.[3] Direct contact with furuncles was significantly associated with transmission, while exposure to equipment seemed to be less important in its transmission. Some authors, however, have suggested that athletic bags and wrestling mats may also facilitate transmission of the organisms.[4] Rapid treatment and isolation of the affected athlete from other competitors is of major importance in decreasing the rapid spread of the bacteria.[1 2 4] Other authors have suggested that if the incidence of infection is low, then bandaging techniques may be a reasonable means to prevent transmission.[3] If outbreaks continue within a team, the bacterial carrier status of the members can be evaluated by culturing crural and nasal passages,[4] and appropriate treatment can be instituted.

The three main viral infections that affect athletes are verruca (warts), molluscum contagiosum, and herpes simplex. Verruca can occur on any skin surface and can be transmitted by direct contact, but shared showers and locker room floors may also act as reservoirs.[4] Swimmers may be particularly susceptible to plantar verruca.[4] Destroying verruca will help to prevent it from growing and possibly causing pain. Furthermore the destruction may decrease transmission of the virus to other members of the team. Athletes with plantar verruca should wear sandals while showering in shared facilities.[4] Molloscum contagiosum presents similar problems for athletes. This disease is characterised by discrete, white to skin coloured papules, and can be found particularly in wrestlers. Molloscum contagiosum can be, through direct contact, quite contagious and should be promptly treated with destructive methods to decrease the transmission to other athletes.[1 4] Herpes simplex infection, known as herpes gladiatorum when identified in wrestlers, has been extensively reviewed.[1 2 4 6–8] Herpes simplex may also be endemic in rugby players.[5] Clinically the infection can appear as an erythematous plaque upon which are vesicles. These vesicles may rupture resulting in erosions. In a study of one wrestling camp, 34% of participants were infected with herpes simplex.[7] It is primarily transmitted through skin to skin contact, and transmission through fomites is felt to be less important.[7 8] Identifying infected athletes promptly and excluding them from direct contact with other wrestlers will help to halt epidemic occurrences.[7 8] Rapid administration of antiviral treatment may accelerate an athlete's return to wrestling.