# Athletic Performance and Vitamin D

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## ABSTRACT

CANNELL, J. J., B. W. HOLLIS, M. B. SORENSON, T. N. TAFT, and J. J. ANDERSON. Athletic Performance and Vitamin D. *Med. Sci. Sports Exerc.*, Vol. 41, No. 5, pp. 1102–1110, 2009. **Purpose**: Activated vitamin D (calcitriol) is a pluripotent pleiotropic secosteroid hormone. As a steroid hormone, which regulates more than 1000 vitamin D–responsive human genes, calcitriol may influence athletic performance. Recent research indicates that intracellular calcitriol levels in numerous human tissues, including nerve and muscle tissue, are increased when inputs of its substrate, the prehormone vitamin D, are increased. **Methods**: We reviewed the world's literature for evidence that vitamin D affects physical and athletic performance. **Results**: Numerous studies, particularly in the German literature in the 1950s, show vitamin D–producing ultraviolet light improves athletic performance. Furthermore, a consistent literature indicates physical and athletic performance is seasonal; it peaks when 25-hydroxy-vitamin D [25(OH)D] levels peak, declines as they decline, and reaches its nadir when 25(OH)D levels are at their lowest. Vitamin D also increases the size and number of Type II (fast twitch) muscle fibers. Most cross-sectional studies show that 25(OH)D levels are directly associated with musculoskeletal performance in older individuals. Most randomized controlled trials, again mostly in older individuals, show that vitamin D improves physical performance. **Conclusions**: Vitamin D may improve athletic performance in vitamin D–deficient athletes. Peak athletic performance may occur when 25(OH)D levels approach those obtained by natural, full-body, summer sun exposure, which is at least 50 ng·mL$^{-1}$. Such 25(OH)D levels may also protect the athlete from several acute and chronic medical conditions. **Key Words**: PHYSICAL PERFORMANCE, PEAK ATHLETIC PERFORMANCE, ACTIVATED VITAMIN D, CALCITRIOL, 1, 25-DIHYDROXY-VITAMIN D, 25(OH)D

APPLIED SCIENCES

Vitamin D is not a vitamin in the usual sense of the word (12,32). That is, most natural human diets contain little vitamin D—unless those diets include much wild-caught fatty fish. Furthermore, activated vitamin D is a secosteroid hormone, not a cofactor in an enzymatic reaction or an antioxidant like most other vitamins. Moreover, the vitamin D steroid hormone system traditionally began in the skin, not in the mouth. Cutaneous production from incidental ultraviolet B (UVB) radiation in sunlight—not dietary intake—is the principal source of circulating human vitamin D stores (12,32).

Factors that affect cutaneous vitamin D production include latitude, season of the year, time of day, melanin content of the skin, use of sunblock, age, and the extent of clothing covering the body (12,32). When the sun is low on the horizon, atmospheric ozone, water vapor, and particulate air pollution all retard UVB penetration to the earth's surface. Thus, vitamin D–producing UVB radiation is effectively absent early and late in the day and for entire months during the winter at latitudes above 35°, causing the distinct seasonality of 25-hydroxy-vitamin D [25(OH)D] levels (Fig. 1).

Because cutaneous vitamin D production is absent or is drastically reduced during winter months, athletes who do not supplement or expose themselves to artificial UVB radiation have to rely on diet and summer vitamin D stores. The lack of significant amounts of vitamin D in most adult diets (even in diets containing fortified milk), ongoing catabolism of body stores, and declining UVB radiation as the autumn progresses cause serum 25(OH)D to precipitously decline in the fall and reach its nadir in the winter. Therefore, athletes competing in the northern half of the United States—and all of Canada and Europe—are at increased risk for vitamin D deficiency, especially during the late autumn and winter.

Like all humans, athletes at any latitude who practice and compete indoors while avoiding sun exposure are at risk any time of the year (12,32). Even those residing at sunny lower latitudes are at risk for deficiency if they consciously avoid the sun or properly use sunblock. For example, a surprisingly high incidence of vitamin D deficiency exists in Miami, despite its sunny weather and subtropical latitude (45).
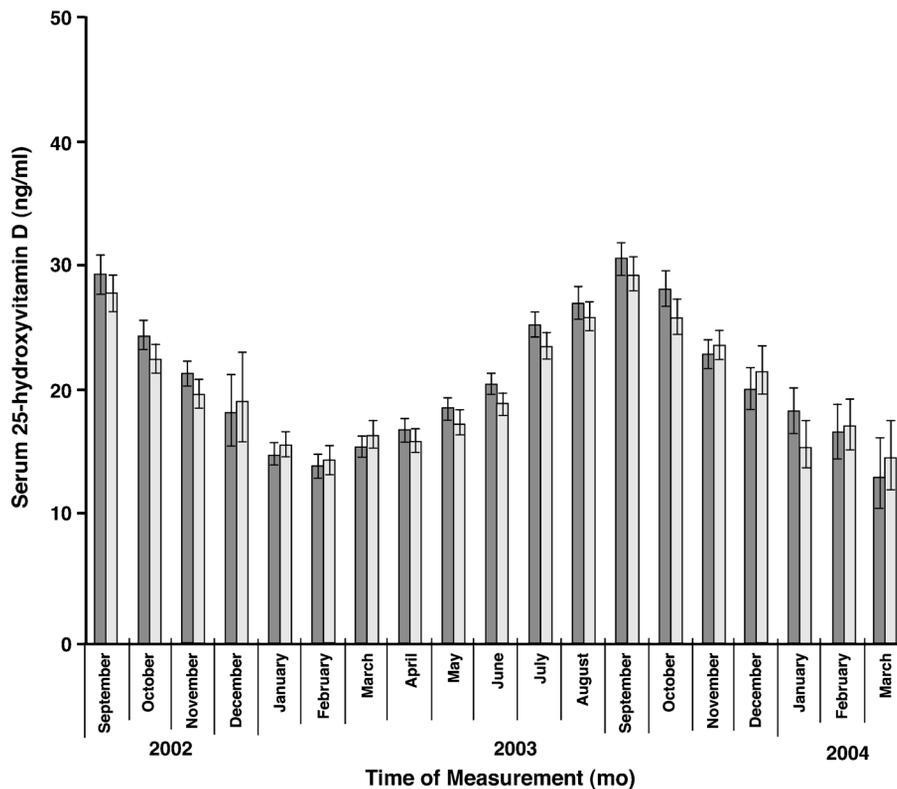
FIGURE 1—Geometric average monthly variations in serum 25-hydroxyvitamin D [25(OH)D] concentrations in men (*dark shade*, *n* = 3723) and women (*light shade*, *n* = 3712) in a 1958 British (England, Scotland, and Wales) cohort at age 45 yr. (Redrawn from figure in: Hyppönen E, Power C. Hypovitaminosis D in British adults at age 45 y: nationwide cohort study of dietary and lifestyle predictors. *Am J Clin Nutr*. 2007;85(3):860–868. Reproduced with kind permission of the *American Journal of Clinical Nutrition*, American Society for Nutrition.)

Dark-skinned athletes face additional problems. Because cutaneous melanin acts as an effective and ever-present sunscreen, athletes with high concentrations of melanin in their skin need up to 10 times longer UVB exposure times to generate the same 25(OH)D stores than do fair-skinned ones (12,32). Therefore, indoor and dark-skinned athletes, athletes who live at more poleward latitudes, wear extensive clothing, regularly use sunblock, or consciously avoid the sun, are all at risk for vitamin D deficiency.

In the last 10 yr, it has become clear that the secosteroid hormone, 1,25-dihydroxy-vitamin D (calcitriol), is not exclusively produced by the kidneys, although the kidney fulfills the endocrine function of vitamin D by secreting calcitriol into serum to maintain the calcium economy. Locally produced and intracellularly regulated calcitriol—independent of serum calcitriol—directly affects numerous cells and tissues, fulfilling vitamin D's multiple autocrine functions (46). Like all steroid hormones, calcitriol acts as a molecular switch to signal genetic transcription. More than 1000 human genes are direct targets of calcitriol (65). Organs involved in athletic endeavors, with evidence of intracellular autocrine manufacture and regulation of calcitriol, include the heart, lungs, adrenal medulla, neurons, muscle, pituitary, bone, and brain (17). A wide variety of brain and nerve cells produce and regulate intracellular calcitriol in an independent autocrine manner, including the balance centers of the cerebellum (19). For a review of vitamin D's genomic and more rapid nongenomic effects on muscle, see Pfeifer et al. (54).

## VITAMIN D DEFICIENCY

Holick (32), writing in the *New England Journal of Medicine*, recently warned that the number of diseases now associated with vitamin D deficiency—including many that afflict athletes—is growing. Very recently, Melamed et al. (50), using population data, found that total mortality was 26% higher in those with the lowest 25(OH)D levels compared with the highest. A meta-analysis of 18 randomized controlled trials found that supplemental vitamin D significantly reduced total mortality, that is, it prolongs life (2). The definition of vitamin D deficiency is changing almost yearly as research shows the low end of ideal 25(OH)D ranges is much higher than we thought only a few years ago (28,34). For example, very recent evidence indicates ideal levels may be above 50 ng·mL$^{-1}$ (27,33).

The parent compound (cholecalciferol) does not begin to be routinely stored in fat and muscle tissue for future use until 25(OH)D levels reach 40–50 ng·mL$^{-1}$ (27,33). At lower levels, the initial 25-hydroxylation in the liver usually follows first-order mass action kinetics, and the reaction is

not saturable. That is, at levels below 40–50 ng·mL$^{-1}$, the body diverts most or all of the ingested or sun-derived vitamin D to immediate metabolic needs, signifying chronic substrate starvation.

At modern man's unnatural and abnormally low 25(OH)D levels—because of avoidance of sunlight—adequate cellular levels of calcitriol directly depend on the availability of adequate serum substrate [25(OH)D], which in turn, depends on vitamin D input; both reactions follow first-order mass action kinetics (67). Only when 25(OH)D levels reach 50 ng·mL$^{-1}$—and few people have such levels—that the initial hydroxylation in the liver reliably switches from first-order to zero-order kinetics (27,33). Thus, at 25(OH)D levels lower than 50 ng·mL$^{-1}$, tissue levels of the secosteroid, calcitriol, directly depend on the amount of vitamin D made in the skin or put in the mouth.

Although it seems improbable that many athletes—especially young ones consuming a good diet—could be vitamin D–deficient, recent evidence suggests many are. Large cross-sectional studies found that vitamin D deficiency is common in otherwise apparently healthy adult populations, and one must assume some of these adults participate in athletics (13,32,40,47,73). A surprisingly high number of otherwise healthy adolescents are also vitamin D–deficient (24,47). Likewise, the mean vitamin D intake in adolescents and young adults in the United States (from milk, other fortified foods, fish, and supplements combined) is 200–300 IU·d$^{-1}$ (73), an intake too low to have significant effects on 25(OH)D levels (67,68). Very recently, Gordon and her colleagues at Boston Children's Hospital found that 40% of 365 healthy infants and toddlers had 25(OH)D levels less than 30 ng·mL$^{-1}$, and it appears from extrapolating their data that more than 85% had levels below 40 ng·mL$^{-1}$ (25). The above studies indicate that few modern humans living in temperate latitudes—of any age—now achieve levels of 50 ng·mL$^{-1}$.

## VITAMIN D DEFICIENCY AMONG ATHLETES

As Willis et al. (72) recently warned us, a surprisingly high percentage of athletes, especially indoor athletes, are probably vitamin D–deficient. For example, 77% of German gymnasts had 25(OH)D levels below 35 ng·mL$^{-1}$ and 37% had levels below 10 ng·mL$^{-1}$, which are in the osteomalacic range (data extrapolated from graph) (3). Forty-five percent of these gymnasts had hypocalcemia on at least one measurement, and several had undetectable 25(OH)D levels together with hypocalcaemia, which may lead to grand mal seizures. Quite surprisingly, the author did not suggest that these severely deficient gymnasts were unable to practice, although bony abnormalities were common. More recently, Lovell (48) found 15 of 18 elite gymnasts had levels below 30 ng·mL$^{-1}$ and 6 had levels below 20 ng·mL$^{-1}$.

A study of young Finnish female athletes (gymnasts and runners) found that athletes did not differ from nonathletes in either vitamin D intake or serum 25(OH)D levels, and both were more likely than not to be vitamin D–deficient (44). Sixty-seven percent of the young women had levels below 15 ng·mL$^{-1}$ during winter. Mean levels increased to 25 ng·mL$^{-1}$ at the end of a particularly sunny summer but fell to 16 ng·mL$^{-1}$ at the end of the yearlong study. A total of 400 IU of vitamin D daily (the equivalent of four glasses of American milk daily) for 3 months did not prevent these deficiencies. More recently, seven French cyclists training 16 h·wk$^{-1}$ had mean 25(OH)D levels of 32 ng·mL$^{-1}$, surprisingly low for a sport where sun exposure is common (49). No attempt was made to associate athletic performance with 25(OH)D levels in these four studies—or any study that we could locate.

## ULTRAVIOLET IRRADIATION AND ATHLETIC PERFORMANCE

In 1938, Russian authors (26) reported that a course of ultraviolet irradiation improved speed in the 100-m dash in four students compared with matched controls, both groups undergoing daily physical training. Mean times improved 1.7% in the nonirradiated controls undergoing training but 7.4% in irradiated students undergoing identical training.

Indeed, in reading the early German literature, it seems the athletic benefits of UV radiation were widely known by the 1930s, at least in Germany (51):

"It is a well-known fact that physical performance can be increased through ultra-violet irradiation. In 1927, a heated argument arose after the decision by the German Swimmers' Association to use the sunlamp, as an artificial aid, as it may constitute an athletic unfairness, doping, so to speak." (p. 17).

In 1944, German investigators irradiated 32 medical students, twice a week for 6 wk, finding irradiated students showed a 13% improvement in performance on a bike ergometer, whereas the performance of the control students was unchanged (42). In 1945, Allen and Cureton (1) measured cardiovascular fitness and muscular endurance for 10 wk in 11 irradiated male Illinois college students, comparing them with 10 matched unirradiated controls, both groups undergoing similar physical training. The treatment group achieved a 19.2% standard score gain in cardiovascular fitness compared with a 1.5% improvement in the control students.

Several years later, Spellerberg (61) reported on the effects of an extensive program of irradiation of athletes training at the Sports College of Cologne—including many elite athletes—with a "central sun lamp." They reported a "convincing effect" on athletic performance and a significant reduction in chronic pain due to sports injuries. Improved athletic performance with irradiation was so convincing that Spellerberg notified the "National German and International Olympic Committee" (p. 570).
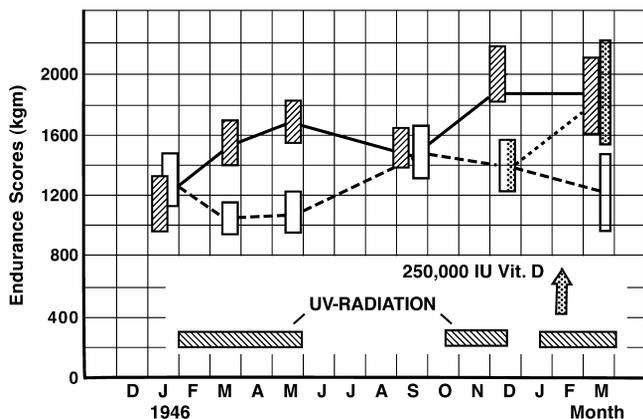
FIGURE 2—Seasonal cardiovascular endurance scores on a bike ergometer of 120 intermittently irradiated German schoolchildren versus unirradiated controls with one group of 30 children given 250,000 IU of vitamin D in February. Note: Treatment group was not irradiated during summer or Christmas vacation (*cross-hatched*, irradiated treatment group; *clear*, unirradiated control group; *stippled*, given 250,000 IU vitamin D as a single dose in February) (Redrawn from figure in: Ronge HE. Increase of physical effectiveness by systematic ultraviolet irradiation. *Strahlentherapie*. 1952;88:563–566. Reproduced with kind permission of Urban and Vogel.)

In 1952, Ronge (55) exposed 120 German schoolchildren to overhead UV lights for 9 months of the year. He installed the lights in classrooms and compared the treated children with 120 nonirradiated control children on a series of cardiovascular fitness tests on a bike ergometer (Fig. 2). He found that nonirradiated children showed a distinct seasonality in fitness and a 56% greater fitness in the irradiated compared with the nonirradiated control children in the early spring. He gave 30 children in the control classrooms 250,000 IU of vitamin D as a single dose in February and found that their cardiovascular performance improved dramatically, approaching the irradiated group 1 month later. Ronge seems to be the first to conclude "…the production of vitamin D (or of a related steroid) explains the success of UV-radiation with regards to physical performance…" (p. 565).

In 1954, researchers at the Max-Planck-Institute for Physiology (43) found that ultraviolet light in the vitamin D–producing UVB range was the most effective wavelength in consistently reducing resting pulse, lowering the basal metabolic rate, and increasing work performance on a bike ergometer. Two years later, Hettinger and Seidl (30) reported that UV radiation induced improvements in forearm muscle strength or performance on a bike ergometer in six of seven subjects.

In 1956, Sigmund (59) studied reaction times during the autumn in a series of controlled experiments on 16 children and an unspecified number of adults (Fig. 3). UV radiation improved choice reaction time by 17% in treated subjects, although it worsened in controls as the autumn progressed. In the late 1960s, American researchers found that even a single dose of ultraviolet irradiation tended to improve the strength, speed, and endurance of college women (14,56,57).

## SEASONALITY OF PHYSICAL FITNESS

In active people, 25(OH)D levels are highly seasonal (Fig. 1). If vitamin D affected athletic performance, then measurements of physical performance should peak in the late summer, when 25(OH)D levels peak, start to decline in early autumn, as 25(OH)D levels fall, and reach their nadir in late winter, when 25(OH)D levels reach their nadir. Conversely, such seasonal changes in physical fitness could be due to seasonal changes in time spent exercising.

However, Hettinger and Muller (29) controlled for time spent exercising and found a distinct seasonal variation in the trainability of musculature (defined as percent strength increase divided by average strength increase, thus allowing small increases in trainability to be detected) by studying wrist flexor strength in subjects undergoing daily training. In seven subjects undergoing daily training for 7–10 consecutive months, they found highly significant seasonal differences in trainability with peak trainability during the late summer, a sharp autumn decline, and nadirs in the winter (Fig. 4).

A study of Polish pilots and crew found that physical fitness and tolerance to hypoxia were highest in the late summer with an unexplained sharp decline starting in September (39). Cumulative work ability among 1835 mainly sedentary Norwegian men during maximal bicycle exercise tests showed an August peak, wintertime nadir, and a sharp decline starting in the autumn (18).

Koch and Raschka (37) reviewed mostly German literature on the seasonality of physical performance, commenting on early studies indicating that strength and maximal oxygen uptake peak in the late summer. The authors then attempted to control for seasonal variations in
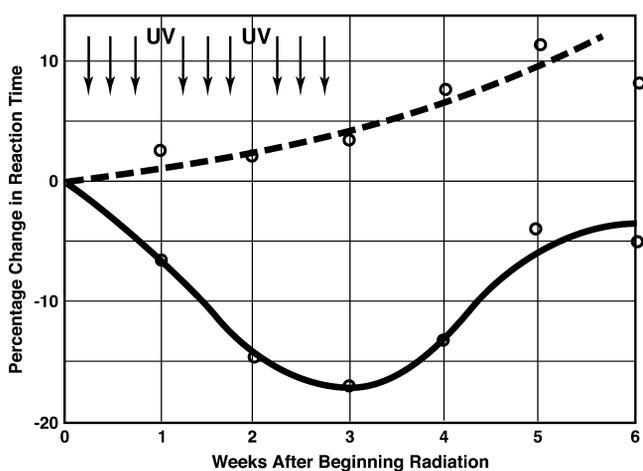


FIGURE 3—Effects of 3 wk of UV radiation on choice reaction times of 16 adults compared with 16 unirradiated controls during October and November. *Dashed line*, unirradiated group, *solid line*, irradiated controls. Note that lower percentages indicate improved reaction times and that control reaction times slowly worsened as autumn progressed, whereas the treatment group, after improving during irradiation, then also worsened. (Redrawn from figure in: Sigmund R. Effect of ultraviolet rays on reaction time in man. *Strahlentherapie*. 1956;101(4):623–629. Reproduced with kind permission of Urban and Vogel.)
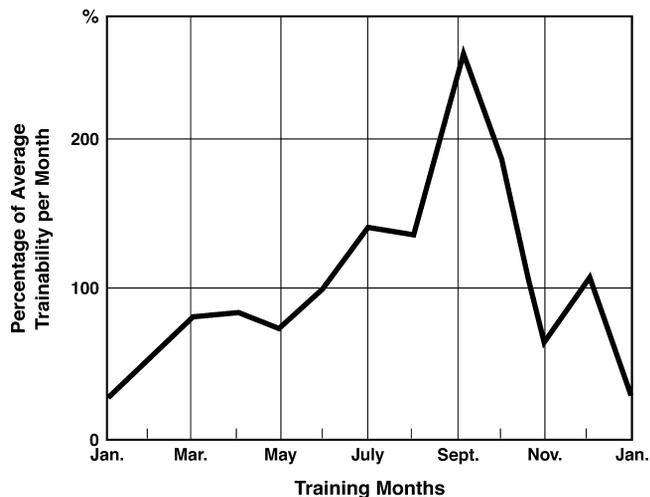
**FIGURE 4**—Average trainability of forearm wrist flexors during the course of the year in seven subjects who trained every day in a uniform manner. The *y*-axis is the percent strength increase of wrist flexors that month divided by average percent strength increase for the entire year. (Redrawn from figure in: Hettinger T, Muller EA. Seasonal course of trainability of musculature. *Int Z Angew Physiol*. 1956;16(2):90–94. Reproduced with kind permission of Springer.)

the time spent exercising by instituting a controlled yearlong training regimen for a 36-yr-old male, beginning in December. The relative increase in the maximum number of press-ups peaked in late summer followed by a rapid decline in the fall, and a nadir in the winter, despite continued training. They speculated that seasonal variations in an unidentified hormone best explained their results.

Ten elite runners from the Swedish national track and field team, in year-round training, displayed maximal oxygen uptake during the summer months (63). Others found significant summer/winter differences in heart rate variability in 120 healthy male Israeli mechanical engineers (38). Lower heart rate variability, which is associated with cardiac pathology, occurred during the winter. Ten healthy male Japanese students showed a significant seasonal variation in $CO_2$ sensitivity with unexplained improvements in late summer, a rapid decline beginning in September, and a nadir in January (31).

## VITAMIN D'S EFFECT ON MUSCLE

Birge and Haddad (5) found that exogenous 25(OH)D affected *ne novo* protein synthesis in muscle, concluding it acts directly on muscle to increase protein synthesis. Administration of vitamin D to deficient rats leads to improved muscle protein anabolism and an increase in muscle mass, weight gain, and a decrease in the rate of myofibrillar protein degradation (70).

Three human muscle biopsy studies confirmed the findings in animals. Biopsies on 12 vitamin D–deficient

patients, before and after vitamin D treatment, found atrophy of Type II muscle fibers before treatment and significant improvement after treatment (74). Muscle biopsies on 11 older patients with osteoporosis, before and after administration of a vitamin D analog together with 1000 mg of calcium for 3–6 months, showed significant increases in both the percentage and area of Type II fibers, despite the lack of any physical training (60). Two years of treatment with even a low dose of vitamin D—1000 IU of ergocalciferol per day—significantly increased muscle strength, doubled the mean diameter, and tripled the percentage of Type II muscle fibers in the functional limbs of 48 severely vitamin D–deficient elderly hemiplegic women (58). The placebo control group suffered declines in muscle strength and in the size and percentage of Type II muscle fibers.
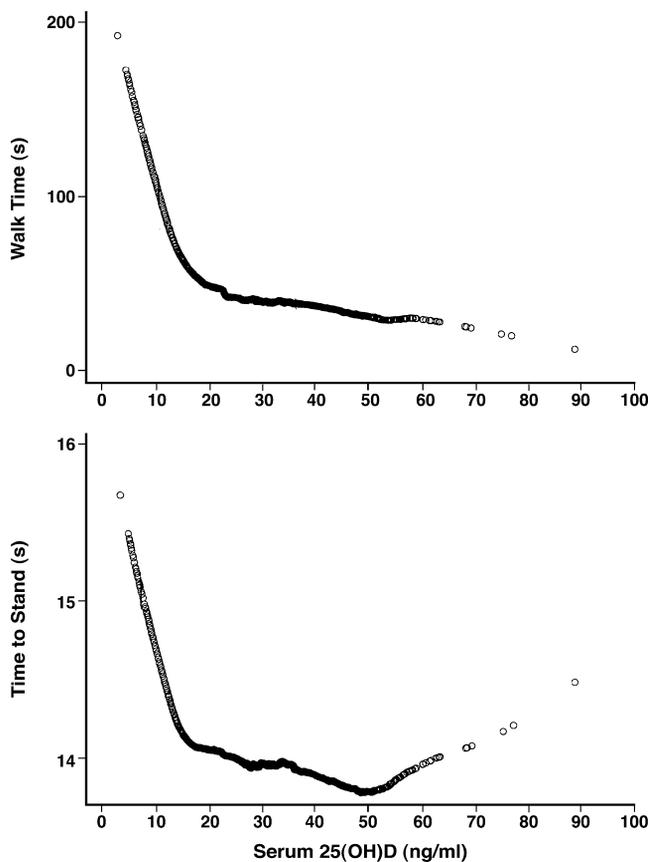


**FIGURE 5**—Lowess regression plots of lower-extremity function on the 8-ft walk test and the sit-to-stand test by 25(OH)D concentrations. Plots are adjusted for sex, age, race or ethnicity, BMI, calcium intake, poverty–income ratio, number of medical comorbidities, self-reported arthritis, use of a walking device, month of assessment, activity level (inactive or active), and metabolic equivalents in active elderly. (Redrawn from figure in: Bischoff-Ferrari HA, Dietrich T, Orav EJ, Hu FB, Zhang Y, Karlson EW, Dawson-Hughes B. Higher 25-hydroxyvitamin D concentrations are associated with better lower-extremity function in both active and inactive persons aged > or =60 y. *Am J Clin Nutr*. 2004;80(3):752–758. Reproduced with kind permission of the *American Journal of Clinical Nutrition*, American Society for Nutrition.)

APPLIED SCIENCES

## SERUM 25(OH)D AND NEUROMUSCULAR FUNCTION

Several cross-sectional studies (6,7,22,35,36,53,64, 69,75) have assessed associations between 25(OH)D and various parameters of neuromuscular performance, finding direct associations between 25(OH)D levels and some measure of physical performance. Many of the studies corrected for factors known to be inversely associated with 25(OH)D levels, such as age, BMI, and serum PTH, so colinearity may be masking stronger associations. Correlations were more frequent and strongest for reaction time, balance, and timed tests of physical performance.

Three additional cross-sectional community studies (9,21,71) found direct associations between 25(OH)D and physical performance with the most dramatic differences noted between 10 and 30 ng·mL$^{-1}$. They also found evidence of a 25(OH)D threshold around 40–50 ng·mL$^{-1}$, above which further improvements in neuromuscular performance were not seen. The largest cross-sectional community study (9) found a linear correlation and suggestion of a U-shaped curve (Fig. 5) with performance on time to stand tests peaking at 50 ng·mL$^{-1}$. However, some longitudinal studies have shown associations (69,71), and others have not (4,20,66), raising the possibility of reverse causation.

## INTERVENTIONAL TRIALS OF VITAMIN D AND NEUROMUSCULAR PERFORMANCE

Several randomized controlled trials (6,16,23,52,58) in older adults found that vitamin D improves various parameters of neuromuscular functioning, including balance, muscle strength, and reaction time, whereas others found no effect (10,36,41). A case–control study of younger subjects (22) showed dramatic improvements in 55 severely vitamin D–deficient younger subjects. Comparisons between studies are difficult because the authors used a wide variety of vitamin D preparations and doses, and subjects began treatment with widely varying baseline 25(OH)D levels.

Another test of the theory are interventional studies in reducing falls, assuming falls are failures of athletic performance. Bischoff-Ferrari et al. (8) recently reviewed that literature and concluded that vitamin D, even in relatively low doses (800 IU·d$^{-1}$), reduces falls in the elderly.

## DISCUSSION

We reviewed five independent lines of evidence, all of which converge to support the hypothesis that vitamin D may improve athletic performance. Ultraviolet B radiation seems to improve various measurements of athletic performance, but data are largely descriptive and adequate randomized controlled trials are lacking. Several studies showed performance improves with vitamin D–producing UVB light but not UVA. Another unidentified component of UVB radiation, not connected to vitamin D, may explain these findings. However, other than the production of the precursor for the steroid hormone, activated vitamin D, we are not aware of any other biological effects of UVB radiation that could do so.

Associations between peak physical performance and summer season are quite significant, even when physical conditioning was constant. However, this may be secondary to reverse causation. That is, improved physical performance in the summer—and thus high 25(OH)D levels—might be secondary to additional outdoor physical activity in the warmer weather. However, if this is true and using the same logic, physical performance should not begin to decline until late autumn, because—at most temperate latitudes—early fall weather is ideal for outdoor athletics. Besides a consistent positive association of late summer season with peak athletic performance, the above studies found an abrupt, and unexplained, reduction in physical performance beginning when 25(OH)D levels decline (early autumn). Another explanation, that is, testosterone peaks in the summer, fails; the two largest studies of such seasonality show testosterone levels peak in December (15,62).

Studies of muscle biopsies of severely vitamin D–deficient patients, before and after treatment, indicate that vitamin D increases the number and diameter of fast, Type II muscle fibers. Several large community-based cross-sectional studies of neuromuscular functioning and serum 25(OH)D found positive associations, but prospective cohort studies are conflicting, raising the possibility of reverse causation. Most, but not all, placebo-controlled interventional studies in older adults found that vitamin D improves various parameters of neuromuscular functioning.

Few athletes live and train in a sun-rich environment, thus few have "natural" 25(OH)D levels, with the exception of equatorial athletes, such as the runners of Kenya. Another possible exception was the 1968 Summer Olympics in Mexico City, where athletes had to arrive early to acclimatize to the 7400-ft altitude. Because UVB penetration of the atmosphere is higher at high altitudes, because Mexico City is relatively close to the equator, and because of the summer season, ambient UVB irradiation from sunlight would have been intense during the 1968 summer games and should have rapidly increased 25(OH)D levels of any athlete acclimatizing outdoors.

Many new world records were set that summer, and the Americans, perhaps unexpectedly, won more gold and total medals than either the Russians or East Germans. Although most experts attribute the impressive number of world records to decreased ambient air pressure, vitamin D may also have contributed. For example, the Americans dominated in outdoor sports, winning 42 of their 45 gold medals in outdoor sports, whereas the Russians won most their gold medals (18 of 29) in indoor sports. Both the number of new world records, almost entirely in outdoor sports, and the percentage improvement in outdoor world records, for example, Bob Beamon added 21 inches to the long jump

FIGURE 6—On October 18, 1968, at the Summer Olympics in Mexico City, Bob Beamon set a world record for the long jump with a jump of 8.90 m (29 ft 2.5 inches). He bettered the existing record by 55 cm (21.75 inches) and became the first person to reach both 28 and 29 ft. (Reproduced with kind permission of Icon Sports Media.)

(Fig. 6), are consistent with the theory that vitamin D improves athletic performance.

## CONCLUSIONS

Extant evidence suggests that adequate treatment of vitamin D–deficient athletes may improve their athletic performance. If such a treatment effect exists, the largest improvements in performance will probably occur in those with the lowest levels; that is, a significant improvement in athletic performance may occur when levels increase from 15 to 30 ng·mL$^{-1}$, but less improvement will occur when levels increase from 30 to 50 ng·mL$^{-1}$. However, both the theory and any existence of any ideal 25(OH)D levels needed for peak athletic performance need confirmations by properly conducted interventional trials. If an effect exists, what is its magnitude? Which athletic performance variables (reaction time, muscle strength, balance, coordination, or endurance) improve the most? What is the optimal 25(OH)D level for peak athletic performance? Do higher levels impair it?

Only direct interventional studies in vitamin D–deficient athletes will answer the athletic performance questions. A double-blind, placebo-controlled, multiple-dose crossover study with long washout periods using variable but relatively high physiological doses, such as 2000, 4000, and 6000 IU of vitamin D·d$^{-1}$, combined with periodic 25(OH)D levels, might answer the question of whether peak performance levels exist for any particular serum 25(OH)D. Alternatively, vitamin D–deficient athletes could ingest doses calculated to increase baseline 25(OH)D levels to approximately 50 ng·mL$^{-1}$, the levels Bischoff-Ferrari et al. (9) found to be associated with peak neuromuscular performance. However, given the growing medical literature on the dangers of vitamin D deficiency (32), ethical concerns may arise in identifying, but not treating, a vitamin D–deficient control group.

Because activated vitamin D is a secosteroid hormone, questions may arise if use of its precursor, vitamin D, constitutes an unfair advantage, "doping, so to speak," as the Germans noted in 1940 (51). However, unlike testosterone or growth hormone, vitamin D deficiency is probably common among athletes. Furthermore, untreated vitamin D deficiency is associated with numerous serious illnesses (32) and is a risk factor for early death (50). Withholding vitamin D in vitamin D–deficient athletes seems to violate most rules of modern medical ethics and may expose the sports medicine physician to needless future liability (12).

Although science may or may not find performance-enhancing effects of vitamin D in the future, good medical practice in the present always supersedes performance-enhancing theories awaiting future research. Vitamin D deficiency may be quite common in athletes. Stress fractures, chronic musculoskeletal pain, viral respiratory tract infections, and several chronic diseases are associated with vitamin D deficiency (11,12,32,72). Those caring for athletes have a responsibility to promptly diagnose and adequately treat vitamin D deficiency. Adequate treatment requires thousands, not hundreds, of IU of vitamin D daily, doses that may make many sports physicians uncomfortable (12).

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