Low 25(OH) vitamin D concentrations in international UK track and field athletes

Noel Pollock, Paul Dijkstra, Rob Chakraverty, Bruce Hamilton

UK Athletics, Hospital of St John and St Elizabeth, London, UK
Noel Pollock, MB BCh, MSc, FFSEM (UK), CESR
Paul Dijkstra, MB ChB, MPhil, FFSEM (UK)

UK Athletics, Loughborough, UK
Rob Chakraverty, MB BCh, MSc, FFSEM (UK)

Qatar Orthopaedic and Sports Medicine Hospital, Aspetar, Qatar
Bruce Hamilton, MB ChB

Corresponding author: *N Pollock (npollock@uka.org.uk)*

Abstract

Objective. While it is recognised that vitamin D deficiency is common in the general population, there have been no studies in elite athletes in the UK. This observational study aimed to assess the 25 hydroxy-vitamin D (25(OH)D) status of elite athletes on the Great Britain track and field team.

Methods. A cross-sectional observational study was performed by analysing blood results from elite athletes on the British athletics team (*N*=63; mean ± standard deviation (SD) age 24.9±4.2 years). Athletes on the elite programme were offered blood tests through the winter and summer of 2009 and were eligible for inclusion in the study.

Results. Nineteen per cent (*n*=12) of athletes in the current study can be classified as 25(OH)D deficient (<20 mcg/l), while a further 29% (*n*=18) can be classified as having insufficient serum 25(OH)D levels (20 - 30 mcg/l). Female sex (insuffcient and deficient OH(D) prevalence 58%, *n*=18) and dark skin (prevalence 65%, *n*=20) were found to be independent predictors of serum 25(OH)D levels of <30 mcg/l.

Conclusion. This study reveals a notable prevalence of low serum 25(OH)D levels in elite athletes and subsequent management of deficient athletes is likely to be of importance for athlete health. The impact of these results on athletic performance remains to be determined, and clinical trials to assess performance, particularly muscular performance, following correction of 25(OH)D status in deficient athletes are required.

S Afr J SM 2012;24(2):55-59.

Introduction

The epidemiology, clinical relevance and management of vitamin D deficiency are medical issues of both academic and public interest. The role of vitamin D in calcium regulation and bone health has been well established,[1] but recent evidence has identified associations between vitamin D deficiency and cardiovascular disease, diabetes, autoimmune disease, cancer of the prostate, breast and colon, as well as all-cause mortality.[2-11] As vitamin D has over 1 000 human genes as direct targets, including skeletal muscle, heart, lungs and adrenal medulla,[12,13] there may be potentially significant consequences of vitamin D deficiency on athletic performance. However, there are very few publications about the prevalence of vitamin D deficiency in elite athletes or the effects of vitamin D deficiency on athletic performance. This is the first article regarding vitamin D status in elite track and field athletes.

Recent population studies have illustrated a significant prevalence of vitamin D deficiency in the general population,[14-17] and athletes also seem to be susceptible.[18-20] Serum 25(OH)D is widely accepted as a biomarker for vitamin D status.[21] While some debate persists as to optimal levels of 25(OH)D for health, it is generally accepted that levels of 20 - 30 mcg/l represent vitamin D insufficiency while levels below 20 mcg/l and 10 mcg/l are defined as deficient and severely deficient, respectively.[10]

Vitamin D is unique among nutrients in that almost all diets contain very little vitamin D and production primarily occurs in the skin, after exposure to ultraviolet B (UVB) sunlight.[10,22] Vitamin D cannot be effectively absorbed in the autumn and winter months in the UK because of the angle of the sun and atmospheric UVB absorption.[23,24] In the UK, reduced levels of 25(OH)D have been reported in a number of studies.[25-28] A large study of post-menopausal women reported 77% of women with 25(OH)D levels <28 mcg/l.[27] Deficiencies have also been noted in 78% of patients attending a UK rheumatology clinic[26] and over 90% of an Asian cohort during a UK winter.[25]

It has been widely recognised that mean 25(OH)D levels are lower in dark-skinned individuals at all ages, with greater risk of insufficiency and deficiency.[29-31] This racial difference is primarily due to increased melanin pigmentation which reduces UVB absorption and subsequent vitamin D production.[32] However, with recent public health campaigns emphasising the dangers of sunlight exposure and advocating intensive sun-block cream use, reports have also found high insufficiency rates, of around 60%, in the UK Caucasian population, and those with the fairest skin type to be, in fact, most deficient.[28]

There have been very few publications on vitamin D status in athletes. Recently, in a study of 93 Middle Eastern male athletes, 91% were found to have a 25(OH)D level <20 mcg/l.[18] A report of 18 elite gymnasts in Australia noted 9 to be vitamin D insufficient and a further 6 to be vitamin D deficient.[19] In a study of 9 - 15-year-old Finnish female athletes and non-athletes, 68% of all participants were found to have 25(OH)D levels <15 mcg/l.[20] However, a small study on seven competitive road cyclists in the south of France identified adequate mean 25(OH)D levels of 32.4 mcg/l.[33]

While there are limited data on the effects of vitamin D deficiency on performance in athletes, the potential relationship with fracture risk[34-36] and altered muscle function,[37,38] in addition to the additional pathological associations noted above, would suggest that the identification and subsequent treatment of vitamin D deficiency in athletes is prudent. In the UK, track and field athletes would appear to be at significant risk of deficiency given the UK latitude (51 - 54°N), the indoor training environment and the proportion of dark-skinned athletes in the elite UK Athletics Track and Field team. Therefore, the aim of this study was to assess the 25(OH)D status of funded elite track and field athletes in the Great Britain team.

Table 1. Socio-demographics and other characteristics of the subjects

Variable	Frequency	Percentage
Gender		
Females	31	49
Males	32	51
Endurance		
Endurance	19	30
Sprint/power	44	70
Skin		
Dark	31	49
Fair	32	51
Residence		
Japan	1	2
London	32	51
Midlands	17	27
North England	7	11
Southern Europe	2	3
Southern USA	4	6
Training environment		
Indoors	20	32
Outdoors	43	68
Month of test		
Dec	2	3
Jan	9	14
Feb	5	8
Mar	8	13
Apr	8	13
May	11	18
Jun	8	13
Jul	11	18
Aug	1	2
Season during test		
Summer	39	62
Winter	24	38

Methods

Participants

All elite athletes funded on the UK Athletics World Class Performance Plan and training at UK Athletics High Performance Athletics Centres (*N*=80) were offered 25(OH)D testing throughout the 2008 - 2009 season as part of a routine blood screening programme. Any athlete on this funded plan is considered to have the potential to win a medal at a World Championship or the Olympic Games. No athletes were taking high-dose vitamin D (>1 000 IU/day) supplementation in the 3 months prior to the study. Other vitamin or mineral supplementation, including calcium intake, was not recorded. The study group comprised all athletes who underwent the blood test between December 2008 and August 2009 (*N*=63) and completed informed written consent for the study.

Data collection

Age, sex and skin colour were recorded. Skin colour was defined as either fair-skinned (white Caucasian) or dark-skinned (Afro-Caribbean). The athlete's place of residence and training location (indoor or outdoor) for the previous 2 months were also recorded. Their competitive event was categorised as either endurance (race distances at or above 800 m) or sprint/power (all other track and field disciplines). The month of testing was recorded and categorised as either winter (December - March) or summer (April - August).

Blood sampling

Blood samples were collected by standard venepuncture using a 10 ml syringe and 23 gauge needle. There was no standard fasting procedure before testing. For athletes tested in Birmingham (*n*=5) blood samples were analysed at the Birmingham Heartlands Hospital, those in Loughborough (*n*=17) at the Leicester Royal Infirmary and those in London (*n*=41) were tested at the Hospital of St John and St Elizabeth. Laboratories in London and Loughborough used the same manufacturer's chemiluminescent immunoassay (Liason, Diasorin® Dartford, Kent). No repeated sampling with the same athlete was done to compare different laboratories. The Birmingham Heartlands

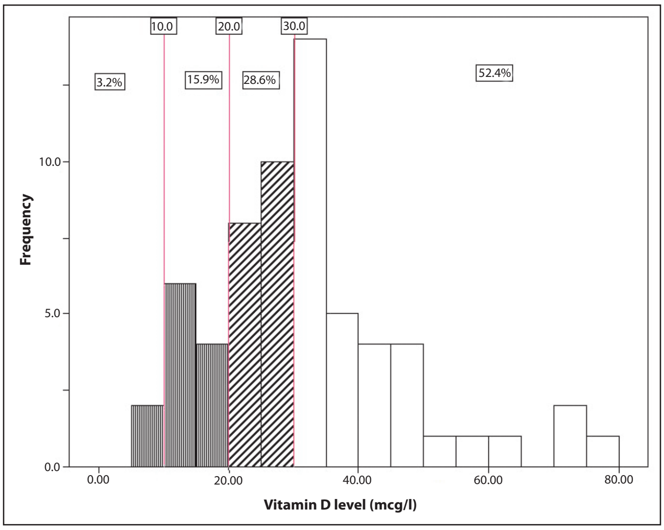

Fig. 1. Distribution of 25(OH)D level: deficient (vertical lines); insufficient (diagonal hash); sufficient (white).

Hospital laboratory used an HPLC tandem mass spectrometer (Applied Biosystems, Warrington Cheshire).

Insufficient 25(OH)D levels were defined as <30 mcg/l and deficiency as <20 mcg/l.[10]

Statistical *t*-tests were performed to assess differences between the variables noted above, under data collection, and regression analysis was used to identify independent predictors for 25(OH)D deficiency. A *p*-value of <0.05 was considered significant. The independent predictors assessed by regression analysis were location, sex, training environment, skin colour, athletic event and season of testing. The study was granted ethical approval by Queen Mary's University of London Ethics panel (QMREC2010/84).

Results

Demographics

Sixty-three subjects (mean ± standard deviation (SD) age 24.9±4.2 years) had their serum 25(OH)D level measured. Their socio-demographic and other characteristics are noted in Table 1. All dark-skinned athletes were Afro-Caribbean. Seventeen athletes eligible for the study did not take up the offer of the blood test.

25(OH)D status in elite track and field athletes

Overall analysis identified a mean (±SD) serum concentration of 25(OH)D of 31.5±15.1 mcg/l (*N*=63). An insufficient 25(OH)D status was noted in 29% (*n*=18) of athletes and a further 19% (*n*=12) were deficient with levels <20 mcg/l (Fig. 1).

Risk factors for low 25(OH)D status

Female athletes, dark-skinned athletes and those tested in the winter months were all noted to have significantly lower 25(OH)D levels (Table 2). Subsequent regression analysis, on variables of sex, age, location, season, skin colour and athletic discipline, identified female sex and dark skin as independent predictors for low 25(OH)D levels.

Table 2. Differences in 25(OH)D levels by gender, event, skin colour, training venue and season

	25(OH)D level (mean±SD), mcg/l	*p*-value
Gender		0.014
Male	36.1±15.7	
Female	26.8±13.2	
Event		0.509
Endurance	33.2±11.1	
Sprint/power	30.8±16.7	
Skin colour		<0.001
Dark	24.9±11.0	
Fair	37.9±16.0	
Training Venue		0.016
Indoors	24.2±16.6	
Outdoors	34.9±13.3	
Season		0.005
Summer	35.4±15.8	
Winter	25.2±11.8	

Analysis of the 15 dark-skinned athletes tested in the winter found 3 (20%) athletes to be insufficient and a further 8 (53%) deficient. In the summer 7 of 16 (44%) dark-skinned athletes were insufficient and 2 (13%) were deficient (Table 3). Of the tests performed on female athletes with dark skin 54% were deficient and a further 31% had insufficient levels. There were no male athletes with fair skin noted to be deficient (Table 4).

Table 3. Proportion of vitamin D deficient and insufficient athletes by skin colour and season

Skin colour	Season	*n*	Insufficient, *n* (%)	Deficient, *n* (%)
Dark-skinned	Winter	15	3 (20)	8 (53)
Dark-skinned	Summer	16	2 (13)	7 (44)
Fair-skinned	Winter	9	3 (33)	
Fair-skinned	Summer	23	5 (22)	1 (4)

Table 4. Proportion of vitamin D deficient and insufficient athletes by skin colour and gender

Skin colour	Gender	*n*	Insufficient, *n* (%)	Deficient, *n* (%)
Dark-skinned	Female	13	4 (31)	7 (54)
Dark-skinned	Male	18	6 (33)	3 (17)
Fair-skinned	Female	18	5 (28)	2 (11)
Fair-skinned	Male	14	2 (14)	0 (0)

Discussion

This is the largest published study on 25(OH)D levels in elite international athletes. It provides clear evidence of a notable prevalence of 25(OH)D insufficiency and deficiency in elite UK track and field athletes. It should be recognised that this study is an observational cross-sectional study in our elite athlete group and there are no control group data from non-elite athletes or the general population. However, the aim of the study was to determine the prevalence of 25(OH)D deficiency in athletes. Two differing laboratory techniques were used to determine 25(OH)D levels with the resultant possibility of inter-laboratory variability. There were no significant differences between the mean 25(OH)D levels in the groups assessed by each laboratory.

The prevalence rates reported here support those of previous studies in young adults.[17,39] In the USA and Canada 36% of young adults have been noted to be deficient in the winter,[39] which compares with our winter figure of 38%. Our overall prevalence of deficiency throughout the year of 19%, with insufficiency in a further 28%, is similar to published work relating to adolescents in northern USA (latitude 42°N) which noted 24% of subjects with levels <15 mcg/l.[17] By comparison, in Middle Eastern national level athletes from a variety of sports, 93% of athletes were noted to be deficient and, in Australia, 33% of 18 gymnasts were found to be deficient.[18,19]

In our cohort, skin colour and sex were noted to be significant independent predictors of vitamin D status. The remarkable prevalence of deficiency in 54% of dark-skinned female athletes (and insufficient levels in a further 31%) is still comparable with some published literature in older groups of a similar skin colour.[29] Skin colour is well recognised as a predictor of vitamin D status with numerous studies identifying lower levels in individuals with dark skin.[29-31] This is primarily due to increased melanin content reducing UVB absorption and subsequent vitamin D production.[32] It has also been reported that seasonal differences in 25(OH)D levels, while apparent in other populations, is less evident in dark-skinned individuals.[40] This is supported by our study, which found that season was not an independent predictor and prevalence of deficiency and insufficiency were similar in the dark-skinned group throughout the year, presumably because of persisting reduction in cutaneous production.

However, in addition to melanin content, social behaviour such as sun exposure and clothing should also be considered when reviewing an athlete's risk of developing vitamin D deficiency. In studies of Middle Eastern groups, females have been reported to have significantly lower 25(OH)D levels.[41-43] This has been attributed to required clothing covering all skin and thereby reducing exposure to UVB.[44]

In South Africa, the Mediterranean and other sun-rich areas the relevance of this study may not be immediately apparent. While individuals in sun-rich areas may be at less risk,[45] a number of studies have shown a significant prevalence of vitamin D deficiency in these areas,[46,47] potentially due to an individual's skin colour or sunscreen application. Our study findings would support the assertion that clinicians in any geographical area with a significant population of dark-skinned individuals should be mindful of the possibility of vitamin D deficiency.

Sex was a significant independent predictor in our study and this has been noted in other populations.[48] It is possible that the application of sunscreen, UVB-blocking moisturiser or make-up may enhance the risk of developing deficiency. Unfortunately we do not have information on hours of sun exposure or the use of sun-protection agents; further work is required. However, if these findings were corroborated in a larger athlete group, vitamin D deficiency may be an additional aetiological risk factor for the increased incidence of stress fractures in female athletes.[49-54]

Insufficient serum concentration of 25(OH)D is known to increase parathyroid hormone (PTH) secretion, increasing bone turnover and bone resorption.[55] In a prospective study, in Finnish army recruits, high serum PTH levels were identified as a risk factor for stress fracture development.[56] In two studies lower 25(OH)D levels have been found to be associated with a significantly increased risk of stress fracture in young Finnish men[57] and with high-grade stress fractures, in a large army cohort.[58] One randomised controlled trial of more than 5 000 army recruits reports a reduction in stress fractures after daily supplementation of 2 g calcium and 800 IU vitamin D.[59]

In addition to the classic role in bone metabolism, vitamin D deficiency may directly impact on athletic performance through other physiological mechanisms including muscle function,[38,60-62] immunity and the potential mediation of exercise-induced inflammation.[63,64] The vitamin D receptor and intracellular vitamin D regulation have also been identified in heart muscle, liver, lung and adrenal systems, all of which are determinants of athletic performance.[13,65,66]

However, the direct evidence for treatment of vitamin D deficiency to improve performance is extremely weak. High-quality trials in athletic populations are required to determine the effects of correcting vitamin D deficiency and insufficiency on athletic, and particularly muscular, performance. There remain many questions regarding optimal 25(OH)D serum concentration and management of deficiency in athletes, and indeed the wider population. Management may include increased sunlight exposure, dietary fortification and medication such as cholecalciferol. However, as this study reveals, athletes are at risk of 25(OH)D deficiency and, for the reasons discussed above, we recommend that sport and exercise medicine physicians are mindful of assessing vitamin D status.

REFERENCES

1. DeLuca HF. Overview of general physiologic features and functions of vitamin D. Am J Clin Nutr 2004;80:1689S-1696S.
2. Autier P, Gandini S. Vitamin D supplementation and total mortality: a meta-analysis of randomized controlled trials. Arch Intern Med 2007;167:1730-1737.
3. Giovannucci E. Calcium plus vitamin D and the risk of colorectal cancer. N Engl J Med 2006;354:2287-2288; author reply 2288.
4. Holick MF. Vitamin D: its role in cancer prevention and treatment. Prog Biophys Mol Biol 2006;92:49-59.
5. Hypponen E, Laara E, Reunanen A, Jarvelin MR, Virtanen SM. Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. Lancet 2001;358:1500-1503.
6. Holick MF. Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease, and osteoporosis. Am J Clin Nutr 2004;79:362-371.
7. Garland CF, Garland FC, Gorham ED, et al. The role of vitamin D in cancer prevention. Am J Public Health 2006;96:252-261.
8. Lappe JM, Travers-Gustafson D, Davies KM, Recker RR, Heaney RP. Vitamin D and calcium supplementation reduces cancer risk: results of a randomized trial. Am J Clin Nutr 2007;85:1586-1591.
9. Pittas AG, Lau J, Hu FB, Dawson-Hughes B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. J Clin Endocrinol Metab 2007;92:2017-2029.
10. Holick MF. Vitamin D deficiency. N Engl J Med 2007;357:266-281.
11. Kamen DL, Cooper GS, Bouali H, Shaftman SR, Hollis BW, Gilkeson GS. Vitamin D deficiency in systemic lupus erythematosus. Autoimmun Rev 2006;5:114-117.
12. Tavera-Mendoza LE, White JH. Cell defenses and the sunshine vitamin. Sci Am 2007;297:62-65,68-70,72.
13. Dusso AS, Brown AJ, Slatopolsky E. Vitamin D. Am J Physiol Renal Physiol 2005;289:F8-28.
14. Calvo MS, Whiting SJ. Prevalence of vitamin D insufficiency in Canada and the United States: importance to health status and efficacy of current food fortification and dietary supplement use. Nutr Rev 2003;61:107-113.
15. Lips P. Vitamin D status and nutrition in Europe and Asia. J Steroid Biochem Mol Biol 2007;103:620-625.
16. Calvo MS, Whiting SJ, Barton CN. Vitamin D intake: a global perspective of current status. J Nutr 2005;135:310-316.
17. Gordon CM, DePeter KC, Feldman HA, Grace E, Emans SJ. Prevalence of vitamin D deficiency among healthy adolescents. Arch Pediatr Adolesc Med 2004;158:531-537.
18. Hamilton B, Grantham J, Racinais S, Chalabi H. Vitamin D deficiency is endemic in Middle Eastern sportsmen. Public Health Nutr 2010:13(10):1528-1534.
19. Lovell G. Vitamin D status of females in an elite gymnastics program. Clin J Sport Med 2008;18:159-161.
20. Lehtonen-Veromaa M, Mottonen T, Irjala K, et al. Vitamin D intake is low and hypovitaminosis D common in healthy 9- to 15-year-old Finnish girls. Eur J Clin Nutr 1999;53:746-751.
21. Schmidt-Gayk H, Bouillon R, Roth HJ. Measurement of vitamin D and its metabolites (calcidiol and calcitriol) and their clinical significance. Scand J Clin Lab Invest Suppl 1997;227:35-45.
22. Holick MF. High prevalence of vitamin D inadequacy and implications for health. Mayo Clin Proc 2006;81:353-373.
23. Kimlin MG, Olds WJ, Moore MR. Location and vitamin D synthesis: is the hypothesis validated by geophysical data? J Photochem Photobiol B 2007;86:234-249.
24. Webb AR. Who, what, where and when - influences on cutaneous vitamin D synthesis. Prog Biophys Mol Biol 2006;92:17-25.
25. Pal BR, Marshall T, James C, Shaw NJ. Distribution analysis of vitamin D highlights differences in population subgroups: preliminary observations from a pilot study in UK adults. J Endocrinol 2003;179:119-129.
26. Serhan E, Newton P, Ali HA, Walford S, Singh BM. Prevalence of hypovitaminosis D in Indo-Asian patients attending a rheumatology clinic. Bone 1999;25:609-611.
27. Macdonald HM, Mavroeidi A, Barr RJ, Black AJ, Fraser WD, Reid DM. Vitamin D status in postmenopausal women living at higher latitudes in the UK in relation to bone health, overweight, sunlight exposure and dietary vitamin D. Bone 2008;42:996-1003.
28. Glass D, Lens M, Swaminathan R, Spector TD, Bataille V. Pigmentation and vitamin D metabolism in Caucasians: low vitamin D serum levels in fair skin types in the UK. PLoS One 2009;4:e6477.
29. Nesby-O'Dell S, Scanlon KS, Cogswell ME, et al. Hypovitaminosis D prevalence and determinants among African American and white women of reproductive age: third National Health and Nutrition Examination Survey, 1988-1994. Am J Clin Nutr 2002;76:187-192.
30. Meier DE, Luckey MM, Wallenstein S, Clemens TL, Orwoll ES, Waslien CI. Calcium, vitamin D, and parathyroid hormone status in young white and black women: association with racial differences in bone mass. J Clin Endocrinol Metab 1991;72:703-710.

31. Cosman F, Nieves J, Dempster D, Lindsay R. Vitamin D economy in blacks. J Bone Miner Res 2007;22 Suppl 2:V34-38.
32. Clemens TL, Adams JS, Henderson SL, Holick MF. Increased skin pigment reduces the capacity of skin to synthesise vitamin D3. Lancet 1982;1:74-76.
33. Maimoun L, Manetta J, Couret I, et al. The intensity level of physical exercise and the bone metabolism response. Int J Sports Med 2006;27:105-111.
34. Lehtonen-Veromaa MK, Mottonen TT, Nuotio IO, Irjala KM, Leino AE, Viikari JS. Vitamin D and attainment of peak bone mass among peripubertal Finnish girls: a 3-y prospective study. Am J Clin Nutr 2002;76:1446-1453.
35. Bischoff-Ferrari HA, Willett WC, Wong JB, Giovannucci E, Dietrich T, Dawson-Hughes B. Fracture prevention with vitamin D supplementation: a meta-analysis of randomized controlled trials. JAMA 2005;293:2257-2264.
36. Pasco JA, Henry MJ, Kotowicz MA, et al. Seasonal periodicity of serum vitamin D and parathyroid hormone, bone resorption, and fractures: the Geelong Osteoporosis Study. J Bone Miner Res 2004;19:752-758.
37. Hamilton B. Vitamin D and human skeletal muscle. Scand J Med Sci Sports 2010;20(2):182-190.
38. Ceglia L. Vitamin D and skeletal muscle tissue and function. Mol Aspects Med 2008;29:407-414.
39. Tangpricha V, Pearce EN, Chen TC, Holick MF. Vitamin D insufficiency among free-living healthy young adults. Am J Med 2002;112:659-662.
40. Harris SS, Dawson-Hughes B. Seasonal changes in plasma 25-hydroxyvitamin D concentrations of young American black and white women. Am J Clin Nutr 1998;67:1232-1236.
41. Allali F, El Aichaoui S, Saoud B, Maaroufi H, Abouqal R, Hajjaj-Hassouni N. The impact of clothing style on bone mineral density among post menopausal women in Morocco: a case-control study. BMC Public Health 2006;6:135.
42. Fonseca V, Tongia R, el-Hazmi M, Abu-Aisha H. Exposure to sunlight and vitamin D deficiency in Saudi Arabian women. Postgrad Med J 1984;60:589-591.
43. Hatun S, Islam O, Cizmecioglu F, et al. Subclinical vitamin D deficiency is increased in adolescent girls who wear concealing clothing. J Nutr 2005;135:218-222.
44. Batieha A, Khader Y, Jaddou H, et al. Vitamin D status in Jordan: dress style and gender discrepancies. Ann Nutr Metab 2011;58:10-18.
45. Poopedi MA, Norris SA, Pettifor JM. Factors influencing the vitamin D status of 10-year-old urban South African children. Public Health Nutr 2011;14:334-339.
46. Linos E, Keiser E, Kanzler M, et al. Sun protective behaviors and vitamin D levels in the US population: NHANES 2003-2006. Cancer Causes Control 2012;23:133-140.
47. Bener A, Al-Ali M, Hoffmann GF. Vitamin D deficiency in healthy children in a sunny country: associated factors. Int J Food Sci Nutr 2009;60 Suppl 5:60-70.
48. Daly RM, Gagnon C, Lu ZX, et al. Prevalence of vitamin D deficiency and its determinants in Australian adults aged 25 years and older: a national, population-based study. Clin Endocrinol (Oxf) 2011. [http://dx.doi.org/10.1111/j.1365-2265.2011.04320.x.]
49. Jones BH, Knapik JJ. Physical training and exercise-related injuries. Surveillance, research and injury prevention in military populations. Sports Med 1999;27:111-125.
50. Brudvig TJ, Gudger TD, Obermeyer L. Stress fractures in 295 trainees: a one-year study of incidence as related to age, sex, and race. Mil Med 1983;148:666-667.
51. Pester S, Smith PC. Stress fractures in the lower extremities of soldiers in basic training. Orthop Rev 1992;21:297-303.
52. Evans RK, Negus C, Antczak AJ, Yanovich R, Israeli E, Moran DS. Sex differences in parameters of bone strength in new recruits: beyond bone density. Med Sci Sports Exerc 2008;40:S645-653.
53. Finestone A, Milgrom C, Evans R, Yanovich R, Constantini N, Moran DS. Overuse injuries in female infantry recruits during low-intensity basic training. Med Sci Sports Exerc 2008;40:S630-635.
54. Popp KL, Hughes JM, Smock AJ, et al. Bone geometry, strength, and muscle size in runners with a history of stress fracture. Med Sci Sports Exerc 2009;41:2145-2150.
55. Lips P. Vitamin D physiology. Prog Biophys Mol Biol 2006;92:4-8.
56. Valimaki VV, Alfthan H, Lehmuskallio E, et al. Risk factors for clinical stress fractures in male military recruits: a prospective cohort study. Bone 2005;37:267-273.
57. Ruohola JP, Laaksi I, Ylikomi T, et al. Association between serum 25(OH)D concentrations and bone stress fractures in Finnish young men. J Bone Miner Res 2006;21:1483-1488.
58. Givon U, Friedman E, Reiner A, Vered I, Finestone A, Shemer J. Stress fractures in the Israeli defense forces from 1995 to 1996. Clin Orthop Relat Res 2000:227-232.
59. Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. Calcium and vitamin D supplementation decreases incidence of stress fractures in female navy recruits. J Bone Miner Res 2008;23:741-749.
60. Sato Y, Iwamoto J, Kanoko T, Satoh K. Low-dose vitamin D prevents muscular atrophy and reduces falls and hip fractures in women after stroke: a randomized controlled trial. Cerebrovasc Dis 2005;20:187-192.
61. Bischoff-Ferrari HA, Conzelmann M, Dick W, Theiler R, Stahelin HB. [Effect of vitamin D on muscle strength and relevance in regard to osteoporosis prevention]. Z Rheumatol 2003;62:518-521.
62. Bischoff HA, Stahelin HB, Dick W, et al. Effects of vitamin D and calcium supplementation on falls: a randomized controlled trial. J Bone Miner Res 2003;18:343-351.
63. Liu PT, Stenger S, Li H, et al. Toll-like receptor triggering of a vitamin D-mediated human antimicrobial response. Science 2006;311:1770-1773.
64. Willis KS, Peterson NJ, Larson-Meyer DE. Should we be concerned about the vitamin D status of athletes? Int J Sport Nutr Exerc Metab 2008;18:204-224.
65. Pfeifer M, Begerow B, Minne HW. Vitamin D and muscle function. Osteoporos Int 2002;13:187-194.
66. Nibbelink KA, Tishkoff DX, Hershey SD, Rahman A, Simpson RU. 1,25(OH)2-vitamin D3 actions on cell proliferation, size, gene expression, and receptor localization, in the HL-1 cardiac myocyte. J Steroid Biochem Mol Biol 2007;103:533-537.