No Association between Vitamin D Deficiency and Markers of Bone Health in Athletes

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ABSTRACT

ALLISON, R. J., A. FAROOQ, B. HAMILTON, G. L. CLOSE, and M. G. WILSON. No Association between Vitamin D Deficiency and Markers of Bone Health in Athletes. *Med. Sci. Sports Exerc.*, Vol. 47, No. 4, pp. 782–788, 2015. **Purpose**: Adequate vitamin D (25(OH)D) is required to maintain good bone health, yet many athletes are 25(OH)D deficient. This study sought to examine the relation between serum 25(OH)D and measures of bone health (bone mineral density (BMD) and T-score) in an ethnically diverse athletic population. **Methods**: Nine hundred and fifty male athletes presented for precompetition medical assessment in our facility. An additional 436 individuals registered with a Qatari sporting federation (such as sailing, archery, shooting, bowling) but exercising <2 h·wk^{-1} were used as control population. There were 30 Asian, 242 Black African, 235 Caucasian, 491 from Gulf Cooperation Countries, 336 Middle Eastern, and 52 Persian participants. All individuals undertook bone densitometry and body composition analysis by dual-energy x-ray absorptiometry and serum 25(OH)D evaluation. **Results**: From 950 athletes, 17.5% demonstrated severe deficiency, 39.2% demonstrated deficiency, 24.5% demonstrated insufficiency, and 18.8% demonstrated sufficiency, compared with 436 controls, 25.9% of whom demonstrated severe deficiency, 46.3% demonstrated deficiency, 19.0% demonstrated insufficiency, and 8.7% demonstrated sufficiency. No athlete presented with a T-score suggestive of osteoporosis (−2.5 SD) or osteopenia (−1.0 SD) at hip total. After adjustment for age, anthropometry, ethnicity, and athletic participation, there was no association between 25(OH)D and any BMD and T-score at any site within athletes. African and Caucasian athletes present with greater ($P < 0.05$) BMD and T-scores at the spine, neck, and hip total than those of Asian, Gulf Cooperation Countries, Middle Eastern, and Persian ethnicities. Athletes participating in high-impact sports present with higher measures ($P < 0.05$) of bone health than control participants regardless of 25(OH)D status. **Conclusions**: There is no association between 25(OH)D and BMD and T-score for any site within male athletes after adjusting for age, ethnicity, and sporting participation. **Key Words:** VITAMIN D INSUFFICIENCIES, BONE MINERAL DENSITY, T-SCORE, ATHLETE HEALTH

EPIDEMIOLOGY

Vitamin D_3 (25(OH)D), or cholecalciferol, is a secosteroid hormone generated by the basal layers of the epidermis via ultraviolet B (UVB) radiation. The bioactive metabolite, 1,25-dihydroxyvitamin D_3 ($1,25(OH)_2D_3$) exerts its biological activity by binding to and activating the vitamin D receptor to regulate numerous downstream signaling pathways in various cells and tissues (13). The major biological function of 1,25-dihydroxyvitamin D_3 is to maintain serum calcium and phosphorus homeostasis to allow essential cellular functions and promote mineralization of the skeleton (17). 25(OH)D deficiency is considered the main contributor to the etiology of osteomalacia and osteoporosis (18,23,30).

Adequate levels of both calcium and 25(OH)D are required to maintain good bone health. 25(OH)D deficiency results in reduced absorption of dietary calcium by 85%–90% in the small intestine. Current clinical ranges (18) of what constitutes 25(OH)D sufficiency (severely deficient (<10 ng·mL^{-1}), deficient (10–20 ng·mL^{-1}), insufficient (20–30 ng·mL^{-1}), or sufficient (>30 ng·mL^{-1})) are based on the association between 25(OH)D deficiency with osteomalacia and the approximate concentration at which parathyroid hormone rises abruptly (19,20,38). The cutoff for insufficiency is the approximate concentration in which calcium absorption is maximized (19). While sufficient levels of serum 25(OH)D are important in optimizing bone mineral density (BMD) in active (22,33) and inactive individuals (3), the exact 25(OH)D value to "optimize" bone health for the regulation of calcium homeostasis (calciotropic properties) (17) is still being debated. Studies suggest that a 25(OH)D value >30 ng·mL^{-1} promotes good bone health and reduces fracture risk in both healthy young and older adults (2,3),

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Submitted for publication June 2014.
Accepted for publication July 2014.

0195-9131/15/4704-0782/0
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DOI: 10.1249/MSS.0000000000000457

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whereas some authors suggest that levels >40 ng·mL^{-1} may still be required (3).

Although studies have demonstrated a significant association between serum 25(OH)D and bone health (6,12,34), there are no studies examining the relation between serum 25(OH)D and measures of bone health in a large, ethnically diverse, athletic population. Athletes are a unique population, in that many are known to be severely 25(OH)D deficient (7,14,25) but have the added stimulus of loading the musculoskeletal system through high-intensity dynamic sporting activity, resulting in greater bone mass at loaded sites compared with nonathletes (29,36). Given the high prevalence of 25(OH)D insufficiency across many athletic populations (range, 67%–91%) (7) and its negative association with bone health, this study sought to examine the relation between serum 25(OH)D levels against markers of bone health (BMD and T-scores) in a large and ethnically diverse athletic population.

METHODS

Participants. Nine hundred and fifty male athletes registered with the Qatar Olympic Committee (QOC) (Asian (n = 18), Black African (n = 218), Caucasian (n = 206), those from Gulf Cooperation Countries (GCC) (n = 222), Middle Eastern (n = 251), and Persian (n = 35)) exercising ≥6 h·wk^{-1} presented for precompetition medical assessment in our facility. A further 436 individuals registered with the QOC but exercising <2 h·wk^{-1} (such as sailing, archery, shooting, and bowling) were used as control participants (Asian (n = 12), Black African (n = 24), Caucasian (n = 29), GCC (n = 269), Middle Eastern (n = 85), and Persian (n = 17). Ethical approval was obtained from the Anti-Doping Laboratory Qatar ethics board, with all athletes completing an informed consent in either Arabic or English. All athletes completed a vitamin D questionnaire in collaboration with an Arabic-, French-, or English-speaking nurse. The instrument included questions specifically related to country of origin, sporting discipline, skin type, self-reported exposure to daily sunlight (0 or <30, 30–60, 60–120, or >120 min), use of sunscreen, dietary supplements and/or medication, and a nurse's assessment of skin color (dark, olive, or fair). All athletes and controls then undertook dual-energy x-ray absorptiometry (DXA) and a blood test to assess serum 25(OH)D status. Athletes with any known comorbidities or family history of osteoporosis or bone disease were excluded from analysis. No female athletes were included in the study because of low participation rates in Qatar.

DXA. DXA (version 5.22b; Osteocore III, Pérols, France) scanning was used to assess hip and spine BMD. A certified technologist from the International Society of Clinical Densitometry performed all calibrations and measurements. The DXA machine was calibrated each morning before the test. The coefficient of variation for these records is <1.01% in our laboratory. BMD was calculated in grams per square centimeter for the spine (L2 to L4), hip–neck (neck), and hip total. In addition, the clinical age-matched and gender-specific z-score index was used to classify BMD. T-scores were calculated for those athletes and control participants older than 20 years.

Laboratory analyses. After the collection of a venous blood sample, 25(OH)D was analyzed using chemiluminescent immunoassay technology (Liaison® 25-OH Vitamin D Total Assay; Diasorin Inc., Saluggia (Vercelli), Italy). The test does not differentiate between the 25(OH)D metabolites, with sensitivity for 25(OH)D set at 7 ng·mL^{-1}. The intra- and interassay coefficients of variation were 7.6%–9.4% and 9.8%–13.4%, respectively.

Statistics. All data were coded and analyzed using SPSS (version 20.0). Descriptive statistics were presented as mean and SD, with range for continuous variables. For categorical variables, frequency and percentage were reported. 25(OH)D data were skewed; therefore, a natural log transformation was applied. Anthropometric comparisons between athletes and controls were performed using a Student's t-test. A one-way ANOVA was performed to assess anthropometric differences between the four 25(OH)D groups (<10, 10–20, 20–30, and >30 ng·mL^{-1}). A *post hoc* analysis with Bonferroni correction was used for further comparisons in the event of significance. To compare osteopenia and osteoporosis DXA scores between athletes and controls, nonparametric exact tests were performed because the expected count in cells was <5. To determine the relation of bone health parameters with 25 (OH)D, multiple linear regression analysis was performed, with bone health as a dependent variable, athletic participation as a fixed factor of interest, and age, body, composition, and ethnicity as covariates. Beta coefficients (β ± SE) were reported. A P value <0.05 was used as the cutoff for statistical significance.

RESULTS

25(OH)D status and anthropometry. From 950 athletes, 17.5% (n = 166) demonstrated severe deficiency, 39.2% (n = 372) demonstrated deficiency, 24.5% (n = 233) demonstrated insufficiency, and 18.8% (n = 179) demonstrated sufficiency, compared with 436 controls, 25.9% (n = 113) of whom demonstrated severe deficiency, 46.3% (n = 202) demonstrated deficiency, 19.0% (n = 83) demonstrated insufficiency, and 8.7% (n = 38) demonstrated sufficiency. Compared with control participants, athletes were significantly (P < 0.003) younger (24.3 ± 4.6 vs 25.9 ± 7.3 yr), taller (183.2 ± 10.6 vs 173.5 ± 6.6 cm), and heavier (81.2 ± 14.3 vs 74.1 ± 15.6 kg) and had reduced body mass index (24.0 ± 2.9 vs 24.6 ± 4.7 kg·m^{-2}), larger body surface area (2.0 ± 0.2 vs 1.9 ± 0.2 m^2), lower percentile body fat (16.3% ± 5.5% vs 22.2% ± 8.4%), increased lean mass (64.5 ± 10.3 vs 54.5 ± 8.9 kg), and reduced fat mass (13.0 ± 6.5 vs 16.5 ± 9.6 kg). The majority of these anthropometric parameters remained significant when athletes and controls were compared in their respective 25(OH)D group (Table 1). In both athletes and controls, a significant association was observed

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TABLE 1. Physical characteristics of athletes and controls based on 25(OH)D status.

		Vitamin D category (ng·mL^{-1})			
		<10	10–20	20–30	>30
Variables		(*n* = 279)	(*n* = 574)	(*n* = 316)	(*n* = 217)
Age (yr)	A	23.9 ± 5.0	23.9 ± 4.4*	24.6 ± 4.6*	25.2 ± 4.6**,***
		15.7–38.0	18.0–36.0	16.9–38.0	16.9–39.0
	C	24.2 ± 5.8	25.8 ± 7.6	28.2 ± 8.1**	25.8 ± 6.0
		13.7–46.0	13.9–53.0	18.0–57.0	25.5 (18.0–42.0)
Height (cm)	A	179.8 ± 10.3*	182.2 ± 10.3*	184.5 ± 10.6*,**	186.5 ± 10.1*,**,***
		143.3–210.2	158.0–211.5	162.0–216.8	157.0–214.3
	C	173.3 ± 6.6	173.1 ± 6.5	174.4 ± 7.3	173.7 ± 6.0
		158.8–197.0	151.2–190.6	161.2–208.0	164.0–186.0
Body mass (kg)	A	77.9 ± 15.3	80.1 ± 14.9*	82.8 ± 14.1*,**	84.6 ± 11.4*,**,***
		50.8–129.8	46.0–158.0	52.8–156.1	56.0–113.3
	C	75.5 ± 20.0	74.1 ± 14.9	74.2 ± 12.1	69.6 ± 9.2
		42.7–157.7	31.5–141.0	52.6–115.2	51.1–88.5
Body mass index (kg·m^{-2})	A	23.8 ± 3.2 *	23.9 ± 3.2*	24.1 ± 2.5	24.1 ± 2.3
		17.0–37.6	15.4–53.8	17.7–33.1	18.0–29.5
	C	25.1 ± 5.9	24.7 ± 4.7	24.3 ± 3.1	22.9 ± 2.4
		15.8–47.0	13.8–41.0	17.8–33.0	18.5–27.5
Body surface area (m^2)	A	1.96 ± 0.2*	2.01 ± 0.2*	2.06 ± 0.2*,**,***	2.09 ± 0.2*,**,***
		1.2–2.7	1.5–2.6	1.6–3.0	1.6–2.6
	C	1.88 ± 0.2	1.87 ± 0.2	1.89 ± 0.2	1.83 ± 0.1
		1.4–2.7	1.2–2.6	1.6–2.5	1.6–2.1
Tissue % fat (%)	A	17.2 ± 6.1*	16.4 ± 5.9*	16.1 ± 5.1*	15.6 ± 4.7*
		7.6–38.9	6.9–52.5	7.0–36.7	6.9–33.8
	C	24.5 ± 9.0****,*****	22.1 ± 8.3	20.7 ± 7.9	18.6 ± 6.4
		10.2–49.7	9.0–46.2	5.7–39.4	9.2–38.1
Lean mass (kg)	A	61.1 ± 11.3*	63.5 ± 10.3*	66.0 ± 9.7*,**,***	67.8 ± 8.4*,**,***
		18.9–102.6	38.9–92.9	42.3–95.3	41.7–91.0
	C	53.7 ± 10.5	54.6 ± 8.6	55.8 ± 7.7	53.9 ± 7.1
		34.7–99.2	23.7–90.8	41.7–77.1	41.3–71.4
Fat mass (kg)	A	13.3 ± 7.1*	13.0 ± 7.1*	13.0 ± 6.1*	12.8 ± 4.9
		5.2–46.2	4.3–81.3	4.6–55.3	4.5–35.9
	C	18.9 ± 12.0****,*****	16.5 ± 9.2	15.2 ± 7.6	12.6 ± 5.2
		5.7–76.6	4.8–58.4	4.1–38.2	5.2–28.2

*Significant difference between A and C, $P < 0.05$.
**Significantly greater than values from <10 ng·mL^{-1}.
***Significantly greater than values from 10–20 ng·mL^{-1}.
****Significantly greater than values from 20–30 ng·mL^{-1}.
*****Significantly greater than values from >30 ng·mL^{-1}.
A, athletes; C, controls.

between serum 25(OH)D and skin exposure ($P < 0.05$), sun exposure ($P < 0.002$), and ethnic origin ($P < 0.001$).

25(OH)D and markers of bone health. Caucasian athletes demonstrated significantly greater ($P < 0.001$) serum 25(OH)D values than all other ethnicities. Athletes of GCC origin presented significantly lower serum 25(OH)D values than Middle Eastern, African, and Caucasian athletes. African and Caucasian athletes demonstrated significantly greater ($P < 0.05$) BMD and T-scores across all sites (spine, neck, and hip total) compared with those of Asian, GCC, Middle Eastern, and Persian athletes (Table 2).

Athletes had significantly greater ($P < 0.05$) BMD and T-scores across all sites (spine, neck, and hip total) compared with those of control participants. Athletes presenting with either 25(OH)D insufficiency (20–30 ng·mL^{-1}) or sufficiency (>30 ng·mL^{-1}) demonstrated significantly greater ($P < 0.05$) BMD scores for the spine, neck, and hip total than athletes severely deficient (<10 ng·mL^{-1}) in 25(OH)D (Table 3).

TABLE 2. Bone health parameters and serum 25(OH)D by ethnicity.

	Asian	GCC	Middle East	Persian	African	Caucasian
Spine BMD	1.30 ± 0.2*	1.27 ± 0.2*	1.37 ± 0.2*	1.31 ± 0.1*	1.46 ± 0.2	1.48 ± 0.1
	1.0 to 1.7	0.7 to 1.7	0.8 to 1.8	1.0 to 1.5	0.9 to 2.0	1.1 to 1.9
Neck BMD	1.29 ± 0.2*	1.24 ± 0.2*	1.34 ± 0.2*	1.35 ± 0.2*	1.46 ± 0.2	1.42 ± 0.2
	0.9 to 1.6	0.7 to 1.8	0.7 to 1.9	1.0 to 1.8	0.9 to 2.0	0.9 to 1.9
Hip total BMD	1.28 ± 0.2*	1.25 ± 0.2*	1.34 ± 0.2*	1.35 ± 0.1*	1.45 ± 0.2	1.41 ± 0.1
	0.9 to 1.6	0.4 to 1.8	0.7 to 1.9	1.1 to 1.6	1.0 to 1.9	1.0 to 1.8
Spine T-score	0.55 ± 1.4*	0.51 ± 1.4*	1.28 ± 1.3*	0.69 ± 1.2*	2.03 ± 1.3	2.14 ± 1.2
	−1.8 to 3.3	−2.9 to 4.2	−3.7 to 5.0	−1.9 to 2.6	−2.7 to 6.3	−1.0 to 5.7
Neck T-score	1.59 ± 1.9*	1.32 ± 1.5*	2.11 ± 1.3*	2.02 ± 1.1*	2.92 ± 1.3	2.72 ± 1.2
	−1.5 to 4.1	−2.5 to 5.3	−3.2 to 6.0	−0.2 to 4.3	−1.4 to 7.0	−1.0 to 5.9
Hip T-score	1.11 ± 1.5*	1.09 ± 1.3*	1.66 ± 1.1*	1.58 ± 0.9*	2.38 ± 1.1	2.14 ± 1.0
	−1.4 to 3.3	−2.4 to 4.9	−3.1 to 5.2	−0.3 to 3.4	−0.7 to 5.5	−0.5 to 4.9
Mean serum 25(OH)D value	17.84 ± 9.7	13.82 ± 7.6***	20.44 ± 10.1	17.19 ± 11.0	19.85 ± 10.7	28.16 ± 12.3**
	6.4 to 55.6	4.0 to 53.0	4.0 to 74.9	4.0 to 54.2	4.0 to 62.7	5.7 to 87.4

*Significantly lower compared with values for Africans and Caucasians.
**Significantly higher than Asians, GCC, Middle Easterns, Persians, and Africans.
***Significantly lower than Middle Easterns, Africans, and Caucasians.

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TABLE 3. BMD and T-scores for athletes and controls based on 25(OH)D status.

Variables		Vitamin D Category (ng·mL^{-1})			
		<10	10–20	20–30	>30
Spine BMD	A	1.38 ± 0.2*	1.42 ± 0.2*	1.44 ± 0.1*,**	1.46 ± 0.1*,**,***
		1.0 to 1.7	0.8 to 1.8	1.0 to 2.0	1.2 to 1.9
	C	1.20 ± 0.2	1.23 ± 0.2	1.25 ± 0.2	1.27 ± 0.2
		0.7 to 1.6	0.7 to 1.7	0.8 to 1.6	0.9 to 1.5
Neck BMD	A	1.37 ± 0.2*	1.40 ± 0.2*	1.43 ± 0.2*,**	1.42 ± 0.2*,**
		1.0 to 1.8	0.8 to 2.0	1.0 to 2.0	1.1 to 1.9
	C	1.17 ± 0.2	1.20 ± 0.2	1.19 ± 0.2	1.23 ± 0.2
		0.8 to 1.6	0.8 to 1.8	0.7 to 1.8	0.9 to 1.7
Hip total BMD	A	1.36 ± 0.2*	1.40 ± 0.2*,**	1.41 ± 0.2*,**	1.41 ± 0.1*,**
		0.4 to 1.8	0.8 to 1.9	1.1 to 1.8	1.1 to 1.8
	C	1.17 ± 0.2	1.21 ± 0.2	1.20 ± 0.2	1.24 ± 0.2
		0.8 to 1.5	0.8 to 1.6	0.7 to 1.6	0.9 to 1.6
Spine T-score	A	1.54 ± 1.2*	1.78 ± 1.2*	1.85 ± 1.2*	1.93 ± 1.1*,**
		−1.5 to 4.2	−1.3 to 5.2	−1.5 to 6.3	−0.6 to 5.1
	C	0.02 ± 1.3	0.17 ± 1.3	0.14 ± 1.4	0.45 ± 1.4
		−2.8 to 3.1	−2.9 to 3.8	−3.7 to 3.2	−2.9 to 2.5
Neck T-score	A	2.44 ± 1.2*	2.60 ± 1.3*	2.75 ± 1.2*	2.60 ± 1.2*
		−0.4 to 5.5	−1.2 to 6.8	−0.3 to 7.0	0.1 to 5.7
	C	0.82 ± 1.4	0.94 ± 1.4	0.90 ± 1.4	1.31 ± 1.5
		−2.3 to 3.7	−2.1 to 4.6	−3.2 to 5.2	−1.4 to 4.5
Hip T-score	A	1.93 ± 1.0*	2.14 ± 1.1*	2.19 ± 1.1*	2.09 ± 0.9*
		−0.5 to 4.7	−0.8 to 5.5	−0.3 to 5.0	0.1 to 4.3
	C	0.61 ± 1.3	0.72 ± 1.1	0.67 ± 1.2	1.12 ± 1.4
		−2.1 to 3.0	−1.8 to 3.8	−3.1 to 3.1	−1.6 to 3.4

*Significant difference between A and C, $P < 0.05$.
**Significantly greater than values from <10 ng·mL^{-1}.
***Significantly greater than values from 10–20 ng·mL^{-1}.
A, athletes; C, controls.

For control subjects, there were no significant differences in any BMD or T-scores across the four 25(OH)D categories.

No athlete had a T-score consistent with osteoporosis (−2.5 SD) at any of the three sites (neck, spine, and hip total). Osteopenia (−1 to −2.5 SD) was noted in one (0.1%) athlete at the neck and in six (0.8%) athletes at the spine (Table 4). No athlete presented with a T-score suggestive of osteopenia at the hip total.

Control participants, however, presented with significantly ($P < 0.001$) lower "normal" T-scores than athletes across all sites (neck, spine, and hip total). Furthermore, control participants presented a significantly ($P < 0.001$) higher prevalence of osteopenia at all sites compared with that of athletes. Although the prevalence of control participants who presented with T-scores consistent with osteoporosis was higher than the prevalence among athletes in all sites, the difference was not significant.

Indoor versus outdoor sports. Outdoor athletes presenting with severe 25(OH)D deficiency demonstrated significantly ($P < 0.05$) lower spine, neck, and hip total BMD than 25(OH)D-insufficient (20–30 ng·mL^{-1}) and 25(OH)D-sufficient (>30 ng·mL^{-1}) outdoor athletes, with outdoor athletes presenting with severe 25(OH)D deficiency and also demonstrating significantly ($P < 0.05$) lower neck and hip total T-scores than those of outdoor athletes with sufficient 25(OH)D (>30 ng·mL^{-1}). For athletes who compete indoors, there was no significant difference in any BMD or T-score at either the neck, spine, or hip total across all four 25(OH) D categories (Fig. 1).

Logarithmic transformation. After logarithmic transformation adjusting 25(OH)D for age, anthropometry, and ethnicity, athletes had a significantly greater BMD and T-scores across all sites than control participants. However, after further adjustment for athletic participation, there was no association between 25(OH)D and any BMD and T-score for any site within athletes.

DISCUSSION

There is a high prevalence of 25(OH)D deficiency across many athletic populations (5,7,15), and we found 57% of male athletes presenting with either 25(OH)D deficiency or severe deficiency. We observed, however, that despite this inadequate level of 25(OH)D, after adjusting 25(OH)D for

TABLE 4. Prevalence of osteopenia and osteoporosis based on T-scores for athletes and controls.

Variables	Status	Athletes	Controls	*P* Value	Athletes, Mean 25(OH)D	Controls, Mean 25(OH)D
Neck T-score	Normal	750 (99.9)	317 (90.1)	<0.001	21.0 ± 11.7	16.6 ± 9.1
	Osteopenia	1 (0.1)	33 (9.4)		11.5 ± 0.0	17.2 ± 12.2
	Osteoporosis	0 (0.0)	2 (0.6)		0.0 ± 0.0	23.5 ± 1.3
Hip T-score	Normal	751 (100.0)	323 (91.5)	<0.001	21.0 ± 11.7	16.7 ± 9.1
	Osteopenia	0 (0.0)	29 (8.2)		—	17.0 ± 12.6
	Osteoporosis	0 (0.0)	1 (0.3)		—	
Spine T-score	Normal	745 (99.2)	283 (80.6)	<0.001	21.0 ± 11.7	17.0 ± 9.6
	Osteopenia	6 (0.8)	59 (16.8)		13.9 ± 6.3	15.0 ± 7.6
	Osteoporosis	0 (0.0)	9 (2.6)		—	20.7 ± 13.3

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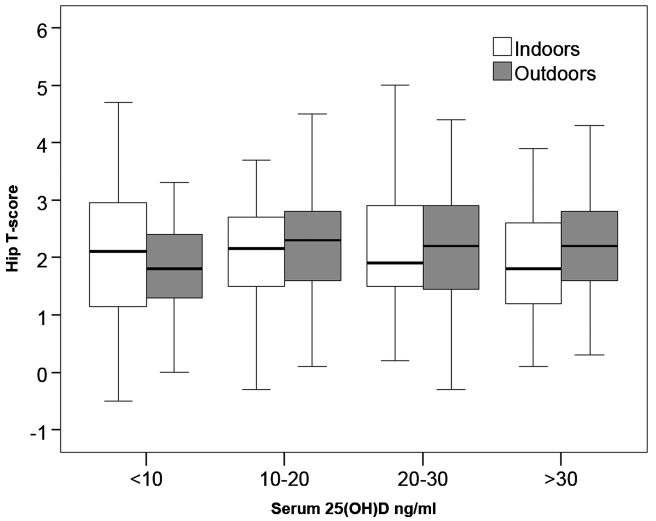

FIGURE 1—BMD and T-scores for indoor versus outdoor athletes based on 25(OH)D status.

age, anthropometry, ethnicity, and athletic participation, there was no association between 25(OH)D and BMD and any T-score (spine, neck, or hip total). Control participants exercising less than 2 $h \cdot wk^{-1}$ presented with significantly ($P < 0.001$) lower "normal" T-scores than athletes across all sites (spine, neck, or hip total).

Effect of ethnicity on 25(OH)D and markers of bone health. Our data demonstrate that across three key skeletal sites (spine, neck, and hip total), African and Caucasian athletes present with significantly greater ($P < 0.05$) BMD and T-scores than those of Asian, GCC, Middle Eastern, and Persian ethnicity. However, for the vast majority of athletes regardless of ethnicity, BMD and T-scores were within the normal clinical range set out by the World Health Organization (21). Caucasians present significantly greater mean serum 25(OH)D values compared with all other ethnicities, with participants originating from the GCC presenting the lowest mean serum 25(OH)D score.

Ethnicity is an important factor when considering serum 25(OH)D status and its potential effect on BMD. Previous research has shown that among Caucasian adults, BMD significantly decreases ($P < 0.01$) as serum 25(OH)D declines, but this is not observed in Black adults ($P = 0.2$) (12). This may be explained by the photoprotective effect of darker skin pigmentation (26), reducing the capacity of the skin to synthesize vitamin D_3 (4).

Vitamin D–binding protein provides insight into why certain ethnic groups may have distinct 25(OH)D and BMD relations (31). Vitamin D–binding protein is the primary vitamin D carrier, binding 85%–90% of total circulating 25 (OH)D with the remaining nonbinding 25(OH)D considered to be bioavailable (1). Vitamin D–binding protein is therefore believed to inhibit certain actions of vitamin D because the bound fraction is unavailable to act on target cells. Polymorphisms in the vitamin D–binding protein, however, produce proteins that differ in affinity for 25(OH)D, and it is these polymorphisms that are known to differ between ethnic groups (9). Consequently, genotyping of common single-nucleotide polymorphisms in the coding region of the vitamin D–binding protein gene (rs4588 and rs7041) demonstrates variation between race and is in turn linked to vitamin D–binding protein function (31). Although controversy surrounding the role of vitamin D–binding protein and its effects on bioavailable 25(OH)D exists (37), our data demonstrate higher BMD and lower 25(OH)D scores in Black athletes compared with those in Caucasians, similar to results observed in previous studies (31).

Lifestyle. Numerous lifestyle factors contribute to 25(OH)D deficiency, including sunlight exposure, sun block use, insufficient dietary vitamin D consumption, refraction and/or nonabsorption of UVB light due to atmospheric dust particles, and wearing concealing clothing, particularly relevant in the Middle East (10,14,16). Our data oppose previous research by Hamilton et al. (14) who observed no association between serum 25(OH)D concentrations in male athletes and the use of sunscreen, sunlight exposure, or skin exposure. Furthermore, there was no significant difference in mean serum 25(OH)D between athletes that competed and trained indoors (volleyball, basketball, and handball) and athletes that competed outdoors (football). This may be explained by the fact that the majority of football training and competition in Qatar is performed after sunset because of several cultural, social, and environmental factors.

Sports participation and bone loading. Exercise is associated with an increase in BMD (29,32). Physical loading contributes to the process of bone remodeling, forming mechanically appropriate bone structure (28). Indeed, numerous studies on adolescent female (27,36) and male athletes (8,35), adult athletes (28,29,32), and senior athletes (24) have all demonstrated the beneficial effect of load-bearing physical activity on bone health. Athletes engaged in high-impact sports present significantly greater total BMD scores than athletes engaged in low-impact sports (11).

Future research. Sport type is an important contributing factor to high peak bone mass. Nikander et al. (28) observed that after adjustment for anthropometry, the sport (or loading modality) was the determinant of the structure and strength of the femoral neck, observing that athletes competing in high-impact (basketball and volleyball) and odd-impact sports (football) demonstrated greater BMD scores than athletes competing in low-impact sports such as swimming and cycling. Although unlike the current study, these studies did not factor serum 25(OH)D status or other lifestyle factors such as UVB exposure into their analysis.

We found that 25(OH)D-insufficient or 25(OH)D-sufficient athletes present significantly greater ($P < 0.05$) BMD scores than severely deficient athletes. However, after adjusting 25(OH)D for age, anthropometry, ethnicity, and athletic participation, there was no association between 25(OH)D and BMD and any T-score (spine, neck, or hip total). The fact that no athlete presented a T-score suggestive of osteoporosis despite a prevalence rate of 57% for 25(OH)D

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deficiency and severe deficiency suggests that in this cohort, the osteogenic effect of impactful weight-bearing exercise is sufficient to maintain markers of bone health, irrespective of 25(OH)D status.

Indoor versus outdoor athletes. All athletes recruited in the present study competed in weight-bearing sports (football, basketball, volleyball, and handball), yet an association between serum 25(OH)D and bone health (BMD and T-scores) was only observed for the outdoor and not the indoor athlete. Sports such as basketball, volleyball, and handball seem to produce more site-specific osteogenic effects with greater BMD scores compared with "odd-impact" sports such as football (11). The rapid accelerating and decelerating movements associated with football are often in directions that the body and hip region are not accustomed to, therefore eliciting a different effect on bone remodeling (28).

Do athletes require 25(OH)D supplementation? This study demonstrates that after adjusting 25(OH)D for age, anthropometry, ethnicity, and athletic participation, there was no association between 25(OH)D and BMD and any T-score and thus questions the notion that sports medicine physicians should be supplementing athletes insufficient in 25(OH)D on the basis of bone health. Recent evidence suggested that correcting serum 25(OH)D in deficient individuals may in turn improve athletic performance (5); however, this is not universally supported (15). Ethical rules dictate that the physician should "do no harm," yet several ethical and methodological issues regarding 25(OH)D supplementation remain unanswered. For athletes, these may include potential supplement contamination, the effects of toxicity such as hypercalcemia, and what exactly is an "optimal" 25(OH)D status.

Limitations. We acknowledge that training volume and intensity were not recorded; athletes were only included in the study if they were registered with the QOC, competed at national- or international-level and trained for more than 6 $h \cdot wk^{-1}$. We also acknowledge that serum 25(OH)D measurement only offered a snapshot of current status. Thus, it is possible for severely deficient (<10 $ng \cdot mL^{-1}$) and deficient (10–20 $ng \cdot mL^{-1}$) athletes in the present study to have spent many years having sufficient (>30 $ng \cdot mL^{-1}$) 25(OH)D before recruitment. Finally, our data are only applicable to male athletes; future work examining a large and ethnically diverse cohort of female athletes requires exploration.

CONCLUSIONS

In summary, our data question 25(OH)D supplementation programs for 25(OH)D-deficient athletes purely on the basis of bone health. Despite 57% of male athletes presenting with either 25(OH)D deficiency or severe deficiency, after adjusting 25(OH)D for age, anthropometry, ethnicity, and athletic participation, there was no association between 25(OH)D and BMD and any T-score (spine, neck, or hip total). We conclude that no male athlete presented a T-score indicative of osteoporosis (−2.5 SD) in the neck, spine, or hip total or a T-score indicative of osteopenia (−1 to −2.5 SD) in the hip total, indicating that the osteogenic effect of impactful weight-bearing exercise is sufficient to maintain markers of bone health, irrespective of 25(OH)D status. Finally, African and Caucasian athletes present with significantly greater ($P < 0.05$) BMD and T-scores across all sites (spine, neck, and hip total) than those of Asian, GCC, Middle Eastern, and Persian ethnicity. Sports medicine physicians working with male athletes who are presented with a 25(OH)D-deficient athlete must factor in ethnicity, alongside other factors such as diet and UVB exposure, in the decision to supplement that athlete.

We acknowledge the sterling efforts of Aspetar's athlete screening team in the data collection; Caroline Buckler, Nelly Khalil, Pascal Tahtouh, Farah Demachkieh, Nisrine Sawaya, Ezzoubair Moustaati, and Diana El Chamaa.

The preceding study was funded solely by the Aspetar Orthopaedic and Sports Medicine Hospital, Doha, Qatar.

None of the authors has any relevant conflicts of interest. No payments or services from a third party were received for any aspect of the submitted work.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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