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Article *in* Journal of Pediatric Endocrinology and Metabolism · May 2015

DOI: 10.1515/jpem-2013-0495 · Source: PubMed

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Vitamin D status and its associations with components of metabolic syndrome in healthy children

Abstract

Aim: High prevalence of vitamin D insufficiency/deficiency has been reported in populations of different countries. The aim of this cross-sectional study was to determine the prevalence and association of vitamin D status with components of metabolic syndrome.

Methods: Lipid profile indices, anthropometric indices [body mass index and waist circumference (WC)], insulin resistance index (HOMA-IR), systolic blood pressure (SBP), diastolic blood pressure (DBP), C-reactive protein, intact parathyroid hormone (iPTH), and serum 25-hydroxyvitamin D [25(OH)D] concentration were evaluated in 297 healthy schoolchildren aged 7–11 years. Multivariate linear regression was used to determine independent predictors associated with low serum 25(OH)D concentrations.

Results: The mean serum 25(OH)D concentration was 14.12±8.20 ng/mL (35.3±20.5 nmol/L); 96% of children had low serum 25(OH)D levels, 31.0% were deficient, and 65.0% had insufficient levels of 25(OH)D. Vitamin D deficiency was higher in girls (χ^2=13.66; p=0.00); 25(OH)D level was negatively associated with WC, HOMA-IR, SBP, DBP, and iPTH. In the multivariate model, WC, DBP, and HOMA-IR were significant independent predictor of low 25(OH)D concentrations.

Conclusion: The prevalence of low vitamin D level in the studied healthy children was high and it is correlated with some components of metabolic syndrome. Outdoor activity for optimum sun exposure and additional studies are needed to evaluate the underlying metabolic syndrome components and hypovitaminosis D complications.

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Keywords: children; metabolic syndrome; prevalence; vitamin D deficiency and insufficiency.

DOI 10.1515/jpem-2013-0495
Received December 23, 2013; accepted August 14, 2014

Introduction

Vitamin D deficiency is a common worldwide problem (1, 2) and a prevalent disorder in developing countries such as Iran (3–5). Vitamin D deficiency is closely correlated with osteomalacia and skeletal deformities in children and osteoporosis and fracture risk in adults (6). Less severe vitamin D deficiency, also called vitamin D insufficiency, causes secondary hyperparathyroidism. It has recently been linked to many chronic conditions, such as diabetes, hypertension, insulin resistance, cardiovascular diseases, autoimmune disease, and cancer (7–9).

Serum 25-hydroxyvitamin D [25(OH)D] concentration is the best functional indicator of overall vitamin D status (1, 2). Some studies have investigated the same threshold level of serum 25(OH)D for children, adolescents, and adults (1, 10), however, most literatures recommended 11–15 ng/mL (25–37.5 nmol/L) of 25(OH)D as a cutoff point for vitamin D deficiency for children (2, 3, 11), which is lower than values reported for adults.

In accordance to defined cutoff values used within specific regions and different populations, prevalence ranges from 20% to 90% have been investigated (12–15). In addition, high prevalence of vitamin D deficiency and insufficiency have been reported in healthy growing children and adolescents in different countries (2, 14). Up to 50% of children are reported to have 25(OH)D levels <12.5 nmol/L and children in the Middle East have serum 25(OH) D levels <25 nmol/L, according to studies published in the past decade (5, 14, 16).

Recent epidemiological, observational, and interventional studies of adults (5, 7, 17, 18) and children (19–21) indicate that low levels of serum 25(OH) D) are linked

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with some components of metabolic syndrome (MetS), such as obesity, insulin resistance, dyslipidemia, and hypertension. MetS predisposes an increased risk for diabetes, cardiovascular events, and some other chronic diseases.

Zanjan province in the northwest of Iran has a sunny climate with four seasons, including a cold winter and a mild summer, which should favor plentiful cutaneous production of vitamin D. However, this region, like other Middle East countries is negatively impacted by urbanization, resulting in lifestyle shifts towards sedentary activities, lack of sunlight exposure, and unhealthy dietary patterns, which probably lead to a high prevalence of vitamin D deficiency. There are limited data on the prevalence of vitamin D deficiency in healthy Iranian teenagers. Therefore, the primary object of this study was to find the prevalence of vitamin D deficiency in healthy children of recruited primary schools in Zanjan province. The second objective was to determine the association of vitamin D deficiency with components of MetS in this population.

Materials and methods

Subject selection

This cross-sectional study was conducted on 297 healthy schoolchildren aged 7–11 years living in Zanjan, the provincial capital of the same name in the northwest of Iran, in the spring and summer of 2011.

All the participants were selected randomly from students of the Zanjan branch of SAMA schools, the nationwide private schools under observation of Azad Universities in Iran. The students studying in SAMA schools are representatives of the middle to top social class of people living in Zanjan.

The study was approved by the Ethical Committee of Zanjan University of Medical Sciences and informed consent was obtained from all the parents of the students.

Anthropometry and blood pressure measurements

All the participants were examined clinically by trained general practitioners for general health. The weight, height, and waist circumferences (WC) were measured by standard methods (22) and body mass index (BMI) was calculated and recorded. Blood pressures (BP) were measured three times with 10 min intervals after at least 15 min of rest and mean of the measurements were considered as BP of the subjects. For those with higher BP, the measurement was repeated on another day. NIH charts for normal population was used to define normal values of BP, weight, and BMI. Central obesity and hypertension were defined based on levels of WC and BP more than 90th percentile of normal population for each age (23).

Biochemical measurements

Venous blood samples were collected from the subjects after at least 12 h of fasting. The analysis of samples was performed using Selectra 2 autoanalyzer (Vital scientific, Spankeren, Netherlands). Total cholesterol (TC) and triglycerides (TG) levels were assayed with a sensitivity of 5 mg/dL using enzymatic colorimetric tests with cholesterol esterase and cholesterol oxidase and glycerol phosphate oxidase, respectively (Parsazmon Kits, Iran). High-density lipoprotein cholesterol (HDL-C) was measured after precipitation of the apolipoproteins with phosphotungistic acid. Low-density lipoprotein cholesterol (LDL-C) was calculated from TC, TG, and HDL-C using the Friedwald formula (24). It was calculated when TG concentrations were more than 400 mg/dL. Lipid standard (C.f.a.s., Boehringer Mannheim, Mannheim, Germany; cat.n.759350) was used to calibrate the Selectra 2 autoanalyzer for each day of the experiment. Assay performance was checked in one out of 20 test intervals using the lipid control serum perineum (normal range) and percipath (pathologic range) whenever applicable. Inter- and intra-assay coefficients of variation (CV) for the assay (TC or TG) were 3.1% and 4.6% in the lower limit and 2.4% and 1.8% for the upper limit. Sera were stored at –70°C until analysis for 25(OH) vitamin D, insulin, and intact parathyroid hormone (iPTH). All the measurements were done in one laboratory center approved by Zanjan Metabolic Diseases Research Center for quality of measurements.

Fasting blood sugar (FBS) was measured on the day of blood collection by enzymatic colorimetric method using glucose oxidase. Insulin levels were measured via an electrochemiluminescence immunoassay (ECLIA) using commercially available kits (Roche, Penzberg, Germany). The homeostasis model assessment index (HOMA index) was used to determine the level of insulin resistance (IR) and was calculated according to the following equation: [insulin (μU/L)] [fasting plasma glucose (FPG) (mmol/L)]/22.5. It was considered as a marker of insulin resistance when it was more than 2.1 (25); 25(OH)D was assayed by ELISA method using immunodiagnostic system (IDS) kits with intra-assay and inter-assay coefficient of variance (CV) with a 5.3% and 4.6% for the lower limit and 6% and 8% for the upper limit, respectively. Intact PTH (iPTH) was measured by ELISA method and Biomerica kits with sensitivity of 1.5 pg/mL. Inter- and intra-assay CV for the measurement was 4% and 5%, respectively.

Based on the suggested cutoffs for children (26, 27), measured serum 25(OH)D concentrations in our sample population were categorized into tertiles as follows: tertile I was considered vitamin D deficiency (sever deficiency), ≤10 ng/mL (≤25 nmol/L); tertile II was considered vitamin D insufficiency (mild deficiency), 10–30 ng/mL (25–75 nmol/L); and tertile III was considered vitamin D sufficiency, ≥30 ng/mL (≥75 nmol/L).

Statistical analysis

Normality of the data distribution was assessed with the Kolmogrov-Smirnov test. Data are given as means±standard deviation or number and percentage. Differences in continuous variables between two groups were tested by using Student t-test or nonparametric Mann-Whitney U-test, depending on data distribution. The association between serum 25(OH)D and the different study variables were examined by bivariate Pearson. Multivariate linear regression analysis was used to determine the independent predictor of 25(OH)D. Differences were considered statistically significant when p value was <0.05.

Statistical analysis was conducted with the Statistical Package for the Social Sciences (version 13; SPSS, Chicago, IL, USA).

Results

Baseline characteristics

The demographic, clinical, and biochemical characteristics of the study population are summarized in Table 1. Two hundred and ninety-seven healthy children [134 (45.1%) boys and 163 (54.9%) girls] aged 7–11 years old were enrolled. As shown, no significant differences were found for variables FBS, serum concentration of insulin, HOMA-IR, LDL-C and HDL-C between the boys and girls. The mean height, weight, WC, SBP, DBP, TG, and iPTH were significantly higher in girls than in boys, whereas the BMI and the mean serum 25(OH)D concentrations were significantly higher in boys than girls (Table 1). The mean serum 25(OH)D and iPTH concentrations were 14.12±8.20 ng/mL and 42.67±37.34 pg/mL, respectively.

Prevalence of vitamin D deficiency

Distribution of our total sample of 297 individuals according to their serum 25(OH)D levels showed that 31.0% (n=92) had 25(OH)D deficiency (≤10 ng/mL; severe vitamin D deficiency status), 65.0% (n=193) had 25(OH)D insufficiency (10–30 ng/mL; mild vitamin D deficiency status), and only 4.0% (n=12) had sufficient concentration (≥30 ng/mL) of serum 25(OD)D. In these categories, 74.6% (n=100) of boys and 57.1% (n=93) of girls showed vitamin D insufficiency and 16.4% (n=22) of boys and 42.9% (n=70) of girls showed vitamin D deficiency (Table 2). Between tertiles groups, vitamin D deficiency was higher in girls (χ^2=13.66; p=0.00).

The relation between serum 25(OH)D levels and other variables

The distribution of PTH relative to 25(OH)D concentrations is shown in Figure 1. Intact PTH was shown to be inversely associated with 25(OH)D (r=−0.155, p=0.007). Pearson's correlation results are listed in Table 3. Serum 25(OH)D level was negatively and significantly associated with WC (r=−0.121, p=0.037), SBP (r=−0.176, p=0.002), DBP (r=−0.190, p=0.001), iPTH (r=−0.155, p=0.007), insulin (r=−0.107, p=0.065), and borderline negative correlation with HOMA-IR (r=−0.107, p=0.065) and positively associated with BMI (r=0.117, p=0.042). Multivariate linear regression analysis for the association of serum 25(OH)D with other variables are presented

Table 1 Demographic and biochemical features of 297 study subjects.

Variables	Totals (n=297)	Boys (n=134)	Girls (n=163)	p-Value
	Means±SD	Means±SD	Means±SD	
Age, years	7.86±1.32	7.64±1.33	8.04±1.29	0.010
Height, cm	127.04±9.92	121.79±8.05	131.37±9.20	0.000
Weight, kg	28.99±7.82	28.00±7.61	29.80±7.92	0.049
BMI, kg/m^2	17.81±3.39	18.70±3.60	17.08±3.02	0.000
WC, cm	60.47±8.83	59.13±8.81	61.57±8.71	0.017
SBP, mm Hg	99.50±12.51	95.93±10.68	102.44±13.16	0.000
DBP, mm Hg	63.91±11.53	61.72±11.11	65.72±11.58	0.003
FBS, mg/dL	82.79±10.93	82.45±12.12	83.08±9.88	0.621
Insulin, μU/L	8.96±6.91	8.63±7.48	9.23±6.41	0.457
HOMA-IR	1.83±1.37	1.75±1.37	1.91±1.36	0.319
TC, mg/dL	167.22±25.57	165.52±25.29	168.61±25.7	0.300
TG, mg/dL	87.21±19.69	84.15±17.48	89.73±21.07	0.015
LDL-C, mg/dL	93.27±16.25	92.69±15.47	93.74±16.89	0.583
HDL-C, mg/dL	51.52±10.90	52.69±10.79	50.55±10.92	0.093
25(OH)D, ng/mL	14.12±8.20	17.62±9.55	11.24±5.42	0.000
iPTH, pg/mL	42.67±37.34	33.23±19.97	50.44±45.67	0.000
CRP, mg/L	1.69±3.89	1.22±1.96	2.08±4.90	0.058

n, number; SD, standard deviation; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBS, fasting blood sugar; HOMA-IR, homeostatic model assessment for insulin resistance; TC, total cholesterol; TG, triglyceride; LDL-C, cholesterol joined low-density lipoprotein; HDL-C, cholesterol joined high-density lipoprotein; 25(OH)D, 25-hydroxy vitamin D; iPTH, intact parathyroid hormone; CRP, C-reactive protein; p-value was calculated by the independent t-test. A p-value <0.05 was considered significant.

Table 2 Anthropometric and biochemical characteristics of studied children (n=297) based on serum 25-hydroxy vitamin D [25(OH)D] tertiles.

	Deficient	Insufficient	Sufficient	p-Value
	≤10 ng/mL	10–30 ng/mL	≥30 ng/mL	
n, %	92 (31.0)	193 (65.0)	12 (4.0)	0.00
Gender [n, %]				
Boys [n=126 (44.4)]	22 (16.4)	100 (74.6)	12 (9.0)	0.00
Girls [n=158 (55.6)]	70 (42.9)	93 (57.1)	0.0 (0.0)	0.00
Age, years (means±SD)	7.87±1.29	7.84±1.36	8.00±0.60	0.92
BMI, kg/m^2 (mean±SD)	17.09±2.98	18.12±3.59	17.81±1.94	0.047
WC, cm	61.36±8.94	60.25±8.92	57.17±5.24	0.255
SBP, mm Hg	102.66±12.34	98.19±12.40	96.33±12.05	0.012
DBP, mm Hg	66.69±10.92	62.84±11.70	59.83±9.78	0.014
25(OH)D, ng/mL (mean±SD)	6.74±2.51	16.00±4.73	40.50±9.42	0.000
iPTH, pg/mL (mean±SD)	57.79±51.55	35.59±26.14	40.72±28.47	0.000
FBS, mg/dL (mean±SD)	83.00±11.29	82.44±10.94	86.92±7.30	0.380
Insulin, µU/L (mean±SD)	10.50±8.09	8.12±5.72	10.65±11.65	0.017
HOMA-IR, (mean±SD)	2.11±1.52	1.68±1.21	2.19±2.10	0.032
TG, mg/dL (mean±SD)	90.21±21.21	86.13±19.04	81.67±16.02	0.160
TC, mg/dL (mean±SD)	167.24±23.11	167.08±26.75	169.33±26.16	0.957
LDL-C, mg/dL (mean±SD)	92.85±15.76	93.32±16.55	95.58±16.19	0.858
HDL-C, mg/dL (mean±SD)	51.22±10.37	51.23±10.86	58.33±13.94	0.086
CRP, mg/L (mean±SD)	2.13±5.95	1.50±2.51	1.45±1.62	0.438

n, number; SD, standard deviation; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; iPTH, intact parathyroid hormone; FBS, fasting blood sugar; HOMA-IR, homeostatic model assessment for insulin resistance; TG, triglyceride; TC, total cholesterol; LDL-C, cholesterol joined low-density lipoprotein; HDL-C, cholesterol joined high-density lipoprotein; CRP, C-reactive protein.

in Table 4. WC, DBP, iPTH, and HOMA-IR were significant negative independent predictors and BMI significant positive independent predictors of low serum 25(OH)D concentrations.

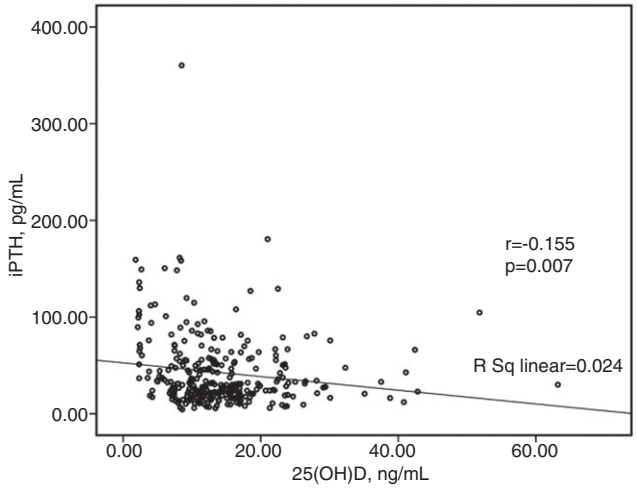

Figure 1 Association between serum 25-hydroxy vitamin D levels (Pearson's r=−0.155, p=0.007) in healthy participant study children (n=297).

Table 3 Correlations of serum 25-hydroxy vitamin D concentrations with biochemical and anthropometric variables in bivariate analysis.

Variables	r	p-Value
Age, years	0.001	0.988
Weight	−0.104	0.073
BMI	0.118	0.042
WC	−0.121	0.037
SBP	−0.176	0.002
DBP	−0.190	0.001
iPTH	−0.155	0.007
FBS	0.056	0.332
TC	0.009	0.871
TG	−0.080	0.171
LDL-C	0.025	0.665
HDL-C	0.107	0.066
Insulin	−0.114	0.051
HOMA-IR	−0.107	0.065
CRP	−0.058	0.322

BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; iPTH, intact parathyroid hormone; FBS, fasting blood sugar; TC, total cholesterol; TG, triglyceride; LDL-C, cholesterol joined low-density lipoprotein; HDL-C, cholesterol joined high-density lipoprotein; HOMA-IR, homeostatic model assessment for insulin resistance; CRP, C-reactive protein. A p-value <0.05 was considered significant; r, Pearson correlation.

Table 4 Multivariate linear regression model for plasma 25-hydroxy vitamin D concentrations.

Variables	Regression coefficient, β	SE	p-Value
Constant	25.54	3.47	<0.001
BMI	1.26	0.206	<0.001
WC	−0.410	0.082	<0.001
DBP	−0.107	0.039	0.007
iPTH	−0.024	0.012	0.049
HOMA-IR	−0.688	0.329	0.037

SE, standard error; BMI, body mass index; WC, waist circumference; DBP, diastolic blood pressure; iPTH, intact parathyroid hormone; HOMA-IR, homeostatic model assessment for insulin resistance.

Discussion

The present study is the first, to our knowledge, to evaluate vitamin D status and its relationship with components of metabolic syndrome in a population based, representative sample of healthy children in Zanjan province, northwest of Iran. Although data from children are more limited, in this cross-sectional study of otherwise healthy children aged 7–11 years, a strikingly high prevalence of vitamin D insufficiency/deficiency was observed. Ninety-six percent of the sampled population had vitamin D concentrations below what is considered optimal by the current guidelines. Of note, the prevalence of vitamin D deficiency was more common in girls (42.9%) than in boys (16.4%).

Congruent with our findings, Moussavi et al., in an assessment of 318 students between the ages of 14 and 18, showed that vitamin D deficiency (<50 nmol/L) is detected in 46.2% of the sample examined and girls are four times more likely to have inadequate vitamin D concentrations (28). More recently, Neyestani et al. demonstrated that 91.7% of the 1111 schoolchildren aged 9–12 years have vitamin D levels below 50 nmol/L (29). Contrarily, in a representative survey of US children aged 1–11 years, a significantly lower proportion of sampled individuals were diagnosed as vitamin D deficient; the proportion of children with vitamin D levels below 25, 50, and 75 nmol/L were 1%, 18%, and 69%, respectively (30). Indeed, it may be argued that the prevalence of vitamin D deficiency/insufficiency among schoolchildren in Iran, as our study and other efforts indicate, is substantially high. Yet, these findings should be interpreted with caution.

Currently set guidelines for optimal cut-off have often been derived from limited populations in developed countries, failing to take possible ethnic, regional, and genetic variations into consideration (4). When the same criterion for optimal vitamin D concentrations (≥75 nmol/L) is used, white children are three times more likely to have their vitamin D concentrations in the acceptable range compared with their black counterparts (31). A multitude of vitamin D receptor gene polymorphism has been identified that can modulate the biological functions of vitamin D in humans and result in different outcomes in individuals with comparable concentrations of vitamin D (32–34). Therefore, it is possible that the strikingly high prevalence of vitamin D deficiency results from employing cut-off points not tailored for the population under study, resulting in an overestimation of clinically significant vitamin D deficiency. The question of "whether a universal cut-off point for hypovitaminosis D is appropriate for children of all ages, of different ethnicities, and living in various regions of the world is appropriate or country/region/ethnicity specific guidelines need to be developed in order to capture the true burden of vitamin D deficiency of clinical significance?" is still much alive and debated. Future nationwide studies are needed to develop and validate specific guidelines for the definition of vitamin D deficiency among Iranian children.

In the present study, we found inverse association of serum 25(OH)D concentrations with WC, SBP, DBP, serum insulin, and HOMA-IR, and significantly positive association with BMI.

Obesity have been shown to be associated with decreased serum 25(OH)D concentrations in children (35) and adults (1, 36). In our findings, WC and BMI, two indices of obesity, have negative and positive correlations with serum 25(OH)D levels, respectively. Inverse association of WC and serum 25(OH)D in our results is in accordance with findings of most experts, suggesting association of low vitamin D status with abdominal obesity (3, 20, 37). The WC is a better predictor than BMI for low vitamin D in healthy subjects (38, 39), as WC is an indicator of visceral fat or abdominal fat distribution. Visceral fat is not only metabolically more active in comparison with peripheral fat but also contains large insulin-resistant adiposities (40, 41). BMI is not an indicator of fatness but an index of total or peripheral fat (42, 43).

Most observational studies show inverse relation of 25(OH)D levels and BMI in obese children (35, 44–47). However, this inverse relation between BMI and 25(OH)D is not consistent across ethnic groups (48). Furthermore, other studies found no association between BMI and 25(OH)D (49). In our study, we observed positive correlation between BMI and serum 25(OH)D levels. Although we do not have a definite explanation, we think it may be due to healthy subjects, ethnic/race specificity, differences in study design, and/or subject characteristics. Similar to our findings, Mansoor et al. found a positive correlation

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between BMI and serum 24(OH)D levels in a healthy population of Pakistani adults (4). Snijder et al. (50), in a large population based study, found association of 25(OH)D with total body fat percentage (BF%) was stronger than BMI. Thus, in order to evaluate association of 25(OH)D level with adiposity, in line with anthropometric assessments, the direct performance of BF% measurements is recommended.

In the present study, serum 25(OH)D level was not associated with some individual metabolic risk factors, such as fasting blood glucose, triglycerides, LDL-C, and C-reactive protein. However, there was positive association between 25(OH)D levels and HDL-C, which supports the results of other researchers (20, 35), suggesting that vitamin D probably plays a role in HDL-C metabolism.

Our data is in agreement with some (42, 51), but not all (20), prospective observational studies of children and adolescents that show an inverse correlation between serum 25(OH)D levels and SBP and DBP. There is accumulating evidence that vitamin D may be a suppressor of the rennin-angiotensin-aldosterone system, the system that regulates blood pressure (52). Also, 1,25-hydroxy vitamin D, the activated form of 25(OH)D, has been shown to inhibit rennin-gene expression (53).

In the present study, we found a significant inverse association of serum 25(OH)D concentrations with serum insulin levels and borderline significant association with HOMA-IR, respectively (Tables 2 and 3). This finding is in line with most observational and prospective studies in children and adolescents (51, 54, 55) but with some inconsistency (56, 57). Ganji et al. (51) studied data from NHANES 2001–2006 using 12–19-year-old children and found that low 25(OH)D levels were associated IR. However, Erdonmez et al. (56), using 305 primary and high school students in Turkey, failed to show any relationships between insulin sensitivity and vitamin D status, and suggested the euglycemic hyperinsulinemic method as the gold standard that is required to confirm the results of most reports that assess HOMA-IR as the marker of IR. The mechanisms proposed in insulin resistance in individuals with vitamin D deficiency might include alterations in glucose homeostasis, impair insulin sensitivity or β-cell function or both (9, 51, 54, 58).

In the multivariate linear regression model, WC, DBP, and HOMA-IR were negatively associated with serum 25(OH)D concentrations (Table 4). This finding is consistent with the majority of previous research in adults (4, 45, 59) and in children (3, 60). The inverse association of WC with serum 35(OH)D levels and decreased bioavailability of serum 25(OH)D is likely to be attributed to the sequestration of vitamin D within adipose tissue (59, 61, 62). This result confirms that obesity is a risk factor for vitamin D deficiency, therefore, public health interventions, such as outdoor physical activities and achieving optimal vitamin D status, should be suggested for obese children.

There are some limitations to our study. First, because of the cross-sectional design of the present study, we could not determine causal inference among low vitamin D status and metabolic risk factors. Second, there was a lack of a direct measure of total adiposities or percentage of body fat of the studied subjects. Finally, we did not obtain information regarding physical outdoor activity of studied children, sunscreen use, and socioeconomic status. However, the present study had some strengths including a relatively large sample size for statistical documentation of several important associations and blood samples were obtained during the same season.

In conclusion, we found a high prevalence (96%) of vitamin D insufficiency/deficiency in a sample of healthy schoolchildren in the Zanjan province of Iran. Based upon current results, low vitamin D levels in this schoolchildren population were significantly associated with some components of metabolic syndrome, including WC, SBP, DBP, and HOMA-IR. Moreover, WC, DBP, and HOMA-IR were strong independent predictors of low vitamin D status. Therefore, we suggest that outdoor activity for optimum sunlight exposure and vitamin D dietary supplementation is needed to avoid hypovitaminosis D complications and additional studies are required to evaluate the underlying metabolic syndrome components.

Acknowledgments: This study was financially supported by Zanjan University of Medical Sciences. The authors express appreciation to the staff of the Zanjan Metabolic Disease Research Center.

Conflict of interest statement: The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

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