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RESEARCH ARTICLE

The optimal vitamin D cut-off value associated with hyperglycemia in an Iranian population

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Abstract

Background Vitamin D deficiency may accelerate the risk of type 2 diabetes mellitus. The association of vitamin D with hyperglycemia may be influenced by lifestyle.

Objective To evaluate the relationship between vitamin D status and hyperglycemia among the workers' population.

Methods This was a medical records review of 7054 Iranian factory workers participating in an annual health check-up for employees. Of those, potential participants were included in this analysis if data for serum 25-hydroxyvitamin D [25(OH) D] levels were also available.

Results Data of 429 male participants were used for this analysis. Of those, 61.07% had serum 25(OH)D concentrations lower than the sufficient level [≥20 ng/ml]. Hyperglycemic participants had significantly lower 25(OH)D than those with normal fasting blood glucose (FBG). Regression analyses highlighted serum 25(OH)D as a significant determinant of hyperglycemia [OR: 0.943(0.901, 988); $p = 0.01$]. The association between 25(OH)D and FBG remained significant after adjustment for potential confounders ($p = 0.008$). Using the ROC analysis, the serum 25(OH)D value of 14.7 ng/ml was the optimal cut-off point to predict hyperglycemia in this population (sensitivity: 63.6%, specificity: 62.3%, $p = 0.01$).

Conclusion Our results revealed a considerable proportion of participants with serum 25(OH)D below the optimal level as well as a significant inverse association between vitamin D status and hyperglycemia among the factory workers. These findings highlight the importance of including the evaluation of vitamin D status as a part of annual health examinations for employees, and may help health policy- makers prevent or delay type 2 diabetes mellitus among the workers' population.

Keywords Vitamin D · 25-hydroxyvitamin D · 25(OH)D · Blood glucose · Hyperglycemia · Workers · Cut-off point

Introduction

Diabetes has reached epidemic proportions globally, affecting over 422 million people in the world [1]. Existing evidence shows that not only diabetes mellitus, even non-diabetic degrees of hyperglycemia are directly linked to the accelerated risks of morbidity and mortality [2]. Beside health problems, diabetes also imposes a substantial economic burden on the society through direct medical costs as well as indirect costs resulting from productivity loss, which will be increasingly significant in the working-age population [3].

Vitamin D is a fat-soluble vitamin that plays a crucial role in many physiological functions, including calcium/phosphorus homeostasis, bone metabolism, immune system, cell differentiation and replication as well as glucose hemostasis [4]. Usually, 80–90% of vitamin D in the body is synthetized in the skin upon sunlight exposure and the remainder is obtained through diet. In spite of these dual sources of attainment, vitamin D deficiency (as judged from serum 25-hydroxyvitamin D [25(OH)D] levels) is still a global health concern, estimated to affect at least 1 billion people worldwide [5]. Epidemiological studies show that even with its abundant sunshine, Middle-East region has some of the highest rates of

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vitamin D deficiency in the world [6]. In Iran, surveys from different parts of the country have reported the suboptimal levels of vitamin D in over 70% of the general population [7].

Despite the lack of consensus on vitamin D level required for the optimal health, a serum 25(OH)D concentration > 30 ng/ml is generally considered as sufficient, and the values of 21–29 ng/ml and ≤ 20 ng/ml as vitamin D insufficiency and deficiency, respectively [8]. Cumulative evidence indicates that vitamin D deficiency may accelerate the development of some chronic diseases, including diabetes [9]. On the other hand, an optimal vitamin D status has been found to be associated with a reduced risk [10]. The exact mechanism linking vitamin D status to hyperglycemia remains somewhat unclear; however, it is proposed to be in part due to a reduced activity of pancreatic beta cells leading to a decrease in insulin secretion [11].

As a result of confirming association between vitamin D status and the occurrence of diabetes in several studies, attentions are now attracted toward the possibility of using serum 25(OH)D levels as a predictive factor in hyperglycemia [12, 13]. Moreover, many recent studies have been focused on defining the threshold for a sufficient 25(OH)D level in terms of various measures of glucose and insulin metabolism [14–16]. However, to the best of our knowledge, there has been no study reporting a cutoff value for vitamin D deficiency based on the fasting blood glucose levels. In addition, it was demonstrated that vitamin D predictive power can be greatly influenced by some underlying factors, including lifestyle (e.g. physical activity levels) [17]. Nevertheless, there have been very limited published literatures investigating the association of vitamin D status with hyperglycemia in the factory workers' populations who generally have high physical activity. Given the key role of workers in societies, the present study was designed to investigate: 1) the relationship between serum vitamin D levels and hyperglycemia in a sample of male workers, and 2) the optimal cut-off value for vitamin D deficiency based on the fasting blood glucose levels.

Material and methods

This was a medical record review study conducted in a large auto manufacturer (SAIPA group) in Karaj, Iran. Karaj is a major industrial city in northern-central Iran (20 km west of Tehran), with latitude of 35°50′ N, longitude of 51°00′ E and an average of 2947 h of sunshine per year. Data were collected on 7054 male employees who had participated in an annual health check-up between August 2014 and February 2015. Of these, potential participants were included in this analysis if data for serum 25(OH)D concentrations were also available ($n = 429$).

Clinical and biomedical data were collected from each participant by trained staffs at out-patient clinic, Baharloo Hospital in Tehran, Iran. Weight and height were measured with participants dressed in light clothing, without shoes using a Rasa scale with stadiometer. Body mass index (BMI) was calculated as weight in kilogram divided by height in meters squared. Participants' blood pressures were taken in the sitting position using an aneroid sphygmomanometer (ABN™, Premium) with a standard cuff (normal BP: SBP/DBP < 140/90 mmHg and no anti-hypertensive medication; high BP: SBP/DBP ≥ 140/90 mmHg or on anti-hypertensive medications). Fasting blood samples were collected via venipuncture into serum tubes, and were subsequently sent to the hospital laboratory (Baharloo hospital, Tehran, Iran) to be analyzed for fasting blood glucose (FBG), triglyceride, cholesterol, HDL-cholesterol, LDL-cholesterol, SGOT, and SGPT.

All participants were also asked to complete a questionnaire starting with a set of general questions, followed by asking about their medical history and smoking status (three categories: non-smoker, current smoker and ex-smoker).

Blood glucose levels were determined with enzymatic colorimetric method (normal range: FBG <100 mg/dl). Enzymatic assays were also used to determine the levels of serum triglyceride (normal range: TG <200 mg/dl), total cholesterol (normal range: cholesterol<200 mg/dl), HDL-cholesterol (normal range: HDL ≥ 35 mg/dl) and LDL-cholesterol (normal range: LDL ≤ 130 mg/dl). UV kinetic methods were used to determine SGOT (normal range: SGOT <37 IU/L) and SGPT (normal range: SGPT<41 IU/L) levels in serum samples. All analyses were performed using Pars Azmun diagnostic kits (Pars Azmun Co., Tehran, Iran) with a between- and within-run coefficient of variation <6.2%.

This study was approved by the ethics committee of Iran University of Medical Sciences (approval number: IR.IUMS.REC.1396. 32,734). As this study involved the review of existing medical records, the committee waived the requirement to obtain informed consent from the participants.

Statistical analysis

Statistical analyses were carried out using IBM SPSS Statistics for Windows (Version 20.0 IBM Corp. Released 2011. Armonk, NY: IBM Corp). Descriptive statistics for variables of interest were obtained and normality was assessed using the Kolmogorov-Smirnov test. Unless otherwise stated, results were presented as the mean value ± standard deviation, or as median with inter quartile range for skewed data. Comparison of demographic and clinical characteristics between groups was undertaken using χ^2 test, independent sample t-test or Mann-Whitney U test as appropriate (Table 1).

Table 1 Demographic and clinical characteristics of participants by fasting blood glucose (FBG)

	Normoglycemic group (FBG < 100 mg/dl) $n = 396$ (92.3%)	Hyperglycemic group (FBG ≥ 100 mg/dl) $n = 33$ (7.7%)	*P* value
Age (y)	37 (35–40)	38 (36–44)	0.30
BMI (kg/m^2)	26.8 (25.1–28.7)	28.4 (24.8–29.9)	0.22
Waist circumference (cm)	93.5 (89.0–99.0)	97.0 (90.0–101.5)	0.23
Triglyceride (mg/dl)	150.0 (105.0–205.0)	179.0 (111.5–268.5)	0.17
Cholesterol (mg/dl)	189.0(160.3–209.0)	186.0 (162.0–225.5)	0.48
Systolic blood pressure (mmHg)	110.0 (105.0–120.0)	120.0 (110.0–120.0)	0.23
Diastolic blood pressure (mmHg)	80.0 (70.0–80.0)	80.0(70.0–80.0)	0.42
SGOT (IU/L)*	23.0 (19.0–28.0)	21.5 (19.8–26.25)	0.64
SGPT (IU/L)*	31.5 (23.0–41.0)	32.0 (26.7–41.0)	0.52
Serum 25(OH)D (ng/ml)	17.0 (12.0–25.2)	13.3 (7.8–20.9)	0.01
25(OH)D quartiles			0.24
1st quartile (≤11.70 ng/ml)	96 (88.9%)	12 (11.1%)	
2nd quartile (11.80–16.60 ng/ml)	100 (91.7%)	9 (8.3%)	
3rd quartile (16.70–24.85 ng/ml)	97 (92.4%)	8 (7.6%)	
4th quartile (>24.85 ng/ml)	103 (96.3%)	4 (3.7%)	
25(OH)D status[1]			0.20
Deficient [25(OH)D < 12 ng/ml]	94 (88.7%)	12 (11.3%)	
Insufficient [12 ≤ 25(OH)D < 20 ng/ml]	144 (92.3%)	12 (7.7%)	
Optimal (≥20 ng/ml)	158 (94.6%)	9 (5.4%)	
25(OH)D status[2]			0.03
Deficient [25(OH)D ≤ 20 ng/ml]	243 (90.7%)	25 (9.3%)	
Insufficient [21 ≤ 25(O)HD ≤ 29 ng/ml]	85 (91.4%)	8 (8.6%)	
Optimal (≥30 ng/ml)	68 (100%)	0 (0%)	
Smoking status			0.10
Current smoker[3]	72 (91.1%)	7 (8.9%)	
Ex-smoker[4]	5 (71.4%)	2 (28.6%)	
Non-smoker	319 (93.0%)	24 (7.0%)	
Physical activity level			0.52
Low	56 (14.1%)	4 (12.1%)	
Moderate	116 (29.3%)	7 (21.2%)	
High	224 (56.6%)	22 (66.7%)	

* In a sub group of study population ($n = 330$), including 304 individuals with FBG <100, and 26 hyperglycemic subjects; [1] categorized based on US Institute of Medicine (IOM) recommendation; [2] categorized based on Endocrine Society recommendation; [3] Current smoker, an adult who has smoked at least 100 cigarettes in his/her lifetime and currently smokes cigarettes; [4] Ex-smoker, an individual who was previous current smoker, but has been smoke-free for at least 28 days

In the present study, participants were classified as normoglycemic (FBG < 100 mg/dl) or hyperglycemic (FBG ≥ 100 mg/dl) based on the American Diabetes Association (ADA) cut-offs for normal FBG [18]. Also, they were categorized as normal, overweight and obese according to the standard cutoffs (BMI < 25 kg/m^2, 25 ≤ BMI < 30 and BMI ≥ 30, respectively). Physical activity levels were categorized as low: < 150 min/week of moderate-intensity activity, moderate: 150–300 min/week of moderate-intensity activity or 75–150 min/week of vigorous-intensity physical activity, high: > 300 min/week of moderate-intensity activity or > 150 min/week of vigorous-intensity physical activity.

The associations between serum 25(OH)D and having higher fasting blood glucose were examined using multiple logistic regression analyses, and the results were presented as odds ratio (OR) and 95% CI (Table 2). Serum 25(OH)D was entered into the regression analysis as: 1) a continuous variable (ng/ml) and 2) a categorical variable based on

Table 2 The results of logistic regression models evaluating the association between serum 25(OH)D and high FBG levels

	Model 1	Model 2	Model 3
	Crude OR (95% CI)	AOR (95% CI)	AOR (95% CI)
25(OH)D continuous (ng/ml)	0.943 (0.901, 988)	0.942 (0.900, 0.986)	0.918 (0.861,0.978)
p value	**0.013**	**0.011**	**0.008**
	Model 1	Model 2	Model 3
25(OH)D quartiles	Crude OR (95% CI)	AOR (95% CI)	AOR (95% CI)
1st Q (8.60 ng/ml)§	1.0	1.0	1.0
2nd Q (14.30 ng/ml)§	0.720 (0.290,1.786)	0.777 (0.305,1.983)	0.803 (0.273, 2.366)
3rd Q (29.97 ng/ml)§	0.660 (0.258,1.686)	0.648 (0.245,1.714)	0.230(0.050,1.060)
4th Q (31.00 ng/ml)§	**0.311(0.097,0.996)***	**0.296 (0.089,0.987)***	0.284 (0.072, 1.121)
p for trend	0.088	0.078	**0.015**

Model 1: With no adjustment; Model 2: Adjusted for age, BMI, examination season, physical activity, smoking status; Model 3: Model 2 plus WC, cholesterol, triglyceride, systolic blood pressure, diastolic blood pressure, liver function test (SGOT, SGPT); *, significant in comparison to the reference group (1st Q) at 5% significance level;§, median of the quartile group; Values in bold denote statistical significance at the $p < 0.05$ level.

25(OH)D quartiles, with lowest category as the reference. *P*-values for trend were calculated by the category median values [19].

The best cut-off value of serum 25(OH)D to predict hyperglycemia was determined using receiver operating characteristic (ROC) analysis (Fig. 1). All tests were two-tailed and a $p < 0.05$ was considered as statistically significant.

Results

Participants included in this analysis were 429 Iranian men aged between 28 to 68 years, with a median BMI of 26.81 kg/m^2. Of those, 92.3% had a normal FBG (<100 mg/dl) and 7.7% had a FBG level higher than the normal range (≥ 100 mg/dl). Statistics describing the demographic and clinical

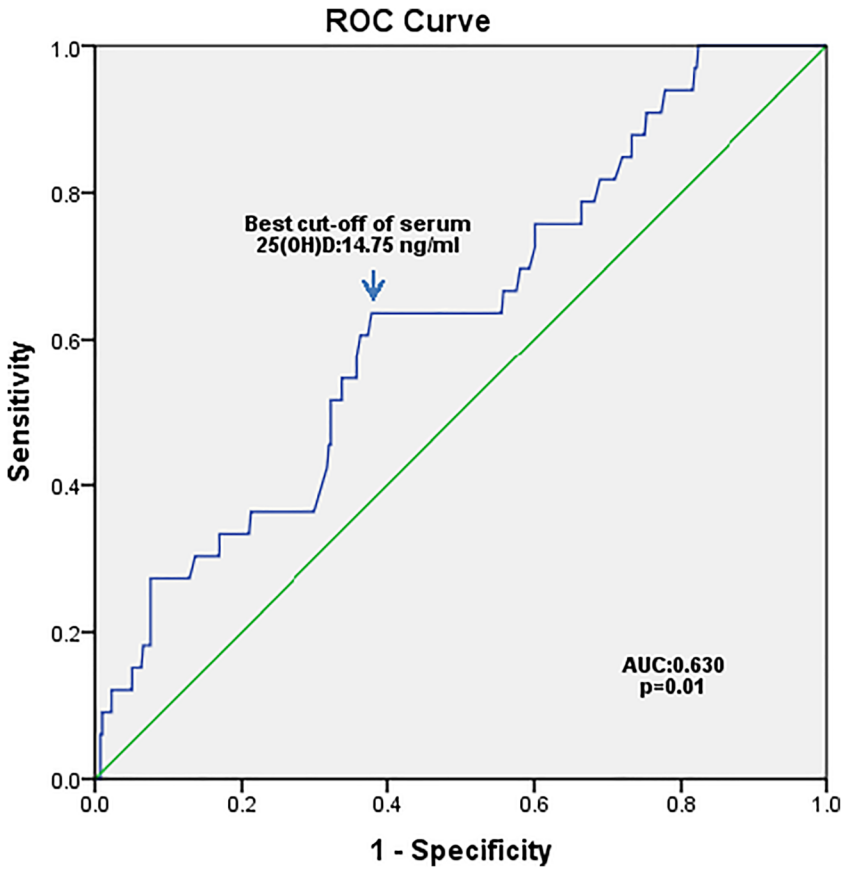

Fig. 1 Receiving operating characteristic (ROC) curve and the best cut-off of serum 25(OH)D associated with hyperglycemia

characteristics of normal and hyperglycemic groups are presented in Table 1. While, the two groups of participants were comparable in terms of different characteristics including age, BMI, waist circumference (WC), systolic and diastolic blood pressure, serum triglyceride, cholesterol, SGOT, SGPT, smoking status and physical activity levels; the hyperglycemic group had a significantly lower serum 25(OH)D than the group with normal FBG [median 13.3 ng/ml, IQR 7.8–20.9 vs. 17.0 ng/ml, 12.0–25.2; $p = 0.01$]. Hyperglycemia was recognized in 9.3% (25/268) of participants who were vitamin D deficient (serum 25(OH)D ≤ 20 ng/ml, $n = 268$) and 8.6% (8/93) of those being vitamin D insufficient [21 ≤ 25(OH)D ≤ 29 ng/ml, $n = 93$], according to the Endocrine Society guideline [8]. However, all participants with a serum 25(OH)D levels of 30 ng/ml or greater ($n = 68$) had a FBG at the normal range [Table 1].

Using the IOM classification for vitamin D status [20], vitamin D deficiency [serum 25(OH)D < 12 ng/ml (30 nmol/l)] was more prevalent among participants with hyperglycemia than those having normal FBG [36.4% (12/33) vs. 23.7% (94/396)]. In addition, 72.7% (24/33) of participants with hyperglycemia had a serum 25(OH)D lower than the optimal level [< 20 ng/ml (50 nmol/l)] compared to 60.1% (238/396) in the normoglycemic group.

There was no significant difference between serum 25(OH)D levels measured in normal, overweight, and obese participants, in either normoglycemic [normal: 16.6 (12.4–23.0) ng/ml, overweight: 17.4 (11.6–26.8) ng/ml, obese: 16.0 (12.0–23.6) ng/ml; $p = 0.70$] or hyperglycemic groups [normal: 13.0 (7.1–26.7) ng/ml, overweight:13.2 (8.8–18.5) ng/ml, obese:19.3 (7.0–23.0) ng/ml; $p = 0.69$). There was also no significant difference between serum 25(OH)D measured in participants with and without central obesity [16.85 (11.4–24.27) vs. 16.26 (12.30–25.87) ng/ml, $p = 0.71$) categorized in terms of waist circumference (WC ≥ 94 cm or WC < 94 cm, respectively). Furthermore, no significant association was found between serum levels of 25(OH)D and age, BMI, WC, lipid profile, blood pressure or liver function tests (all p values >0.05).

Logistic regression analyses highlighted serum 25(OH)D as a significant determinant of having a high FBG (Table 2). When the serum 25(OH)D was entered into the regression model as a continuous variable, every unit increase in serum 25(OH)D significantly reduced the odds of hyperglycemia by 5.7% (OR: 0.943 (0.901, 988); $p = 0.01$) in model 1. The association between serum 25(OH)D and FBG remained significant after adjustment for age, BMI, season, smoking status and physical activity in model 2 (AOR 0.942 (0.900, 0.986); $p = 0.01$) and additional adjustment for WC, systolic and diastolic blood pressures, lipid profile and liver function test in model 3 (AOR 0.918 (0.861,0.978); $p = 0.008$). Analysis by quartiles of serum 25(OH)D showed that the people in the highest quartile [serum 25(OH)D > 24.85 ng/ml] had a significantly reduced odds of having hyperglycemia compared to those in the lowest quartile [serum 25(OH)D ≤ 11.70 ng/ml, $p < 0.05$ in models 1 & 2], although this association attenuated after final adjustment for CVD risk factors in model 3 ($p = 0.07$ for Q4 vs. Q1; $p = 0.01$ for trend).

Using the ROC analysis for the entire group, the serum 25(OH)D value of 14.7 ng/ml was the best cut-off point to predict hyperglycemia in the population (sensitivity: 63.6%, specificity: 62.3%, Yoden's index: 0.259, $p = 0.01$), (Fig. 1). Considering this cut-off, 39.4% of participants (37.4% in normoglycemic group & 63.6% in hyperglycemic group) were vitamin D deficient.

Discussion

In the present study the lower levels of serum 25(OH)D were significantly associated with the higher odds of having hyperglycemia in a sample of Iranian male workers. This association was independent of potential confounders including age, BMI, WC, season, smoking status, physical activity and biomedical factors. These findings are in agreement with several observations revealing an inverse relationship between vitamin D status and disturbances in glucose metabolism [12, 21–23]. For instance, the cohort study by Forouhi et al. [12] provides strong evidence showing that the baseline measures of serum 25(OH)D are inversely related to 10-year risk of hyperglycemia. In another prospective study with an average follow-up of 2.7 years, after adjusting for confounders, higher plasma 25(OH)D was significantly associated with a reduced risk of incident diabetes in patients with pre-diabetes [22]. Similarly, in a recent cross-sectional population-based study, Pannu et al. [21] found that, independent of metabolic syndrome components, individuals in the high tertile of 25(OH)D had a 39% reduced odds of hyperglycemia compared to those in the low 25(OH)D tertile. However, our results do not support the study conducted by Haslacher et al. [17] suggesting that low levels of serum vitamin D may not predict hyperglycemia in individuals with intense physical activity.

Despite robust evidence in observational studies, clinical trials has somewhat failed to favour the notion that vitamin D supplementation can help prevent and/or control diabetes mellitus in people at high risk. While a meta-analysis showed that vitamin D supplementation may reduce fasting plasma glucose and HbA_{1C} in pre-diabetes [24], it had no significant effect on glucose and insulin metabolism in patients with type 2 diabetes or those who were overweight and obese in two other systematic reviews [25, 26]. This discrepancy could hypothetically be due to differences in the population under study, small sample sizes or inappropriate study design [27, 28]. In fact, the majority of intervention studies on this topic had fewer than 100 participates [29] and administrated

vitamin D for a relatively short period [30] or with an inadequate dose [31].

Several potential mechanisms have been proposed to explain the association of vitamin D deficiency with hyperglycemia and insulin resistance [11]. The expression of vitamin D receptors in pancreatic islets highlights the possible role of vitamin D in the β-cell secretory function [32]. Vitamin D is also involved in the regulation of intracellular calcium hemostasis, which in turn is crucial for the action of insulin on various peripheral tissues such as skeletal muscle and adipose tissue [33, 34]. Another possible mechanism linking vitamin D and insulin sensitivity involves the vitamin D-parathyroid (PTH) axis [35]. Finally, adequate levels of 25(OH)D are required to modulate inflammatory pathways in the body [36]. Hypovitaminosis D may accordingly promote insulin resistance and the subsequent risk of diabetes mellitus through amplifying inflammatory responses [37].

Notwithstanding a large number of studies on vitamin D, there is still no general consensus on the optimal threshold reflecting a sufficient circulating 25(OH)D concentration, and it may vary in terms of outcomes and populations. Two main guidelines for interpreting 25(OH)D levels have been established primarily on the basis of the studies on bone health. According to the Endocrine Society, vitamin D deficiency and insufficiency are defined by serum 25(OH)D levels <20 ng/ml and 21–29 ng/ml respectively, and a serum 25(OH)D level ≥ 30 ng/ml is considered as sufficient [8]. However, the Institute of Medicine (IOM) classifies 25(OH)D concentration of <12 ng/ml as deficient and advocates a serum 25(OH)D ≥ 20 ng/ml as sufficient [20]. Furthermore, due to well-established links between vitamin D and the pathogenesis of other diseases including CVD, the guidelines for interpreting 25(OH)D levels need to consider the endpoints beyond the skeletal system as well. Accordingly, many recent studies have been focused on defining the threshold for a sufficient 25(OH)D level in terms of various non-bone related outcomes, including hypertension, body mass index, cardiovascular events as well as different measures of glucose and insulin metabolism (i.e. indices of insulin sensitivity and resistance, fasting and 2-h insulin levels, and 2-h plasma glucose concentration) [14–16]. To the best of our knowledge, this study is the first reporting a cutoff value for vitamin D deficiency based on the fasting blood glucose levels. In the present study, a serum 25(OH)D level of about 15 ng/ml was determined as the optimal cut-off point to predict hyperglycemia in the target population. Though this point was lower than the threshold of ~26 ng/ml suggested to be required for normal glucose metabolism [15], it was quite similar to the cut-off determined by Ashraf et al. [14] on the basis of an insulin sensitivity index. Indeed, our results support recent evidence revealing that the current cut-off of 20 ng/ml may be higher than required in healthy people who are not at risk for vitamin D deficiency [8, 38, 39].

In this study, serum levels of 25(OH)D were not correlated with BMI. There was also no significant difference between serum 25OH levels measured in normal, overweight and obese participants categorized on the basis of BMI. These findings are in line with some previous studies conducted on Iranian population revealing that despite a significant inverse association with fat mass, circulating 25(OH)D levels were not associated with BMI [40]. The strong correlation of vitamin D status with adiposity has been proven in several studies [41, 42]; however, the accuracy of BMI as a surrogate indicator of adiposity is still debated [43]. Besides, in studies using both body fat percentage derived from DXA, and BMI to predict 25(OH)D levels, only body fat percentage remained as an independent determinant in the final model [44]. In the present study, serum 25(OH)D levels also did not exhibit any correlation with waist circumference. Studies examining the link between vitamin D status and waist circumference have reported controversial results [40, 45, 46]. The discrepancy between these findings may be in part due to the fact that circulating levels of 25(OH)D are mostly correlated to visceral adipose tissue [47]; however, waist circumference has generally a stronger association with subcutaneous adipose tissue than with visceral fat [48]. As a result, it may misclassify individuals in respect of visceral adiposity and its related metabolic risks [49]. In this context, it should be also noted that the population included in our analysis was a sample of male factory workers, the majority with a high physical activity level. Accordingly, due to previous studies examining the association of physical activity with the body composition [50], the BMI and WC determined for this population could be assumed to reflect their lean-tissue mass rather than the fat mass.

The present study should be interpreted within the context of its limitations. First, the cross-sectional nature of study design limits the possibility of deriving causal relationships. Moreover, circulating 25(OH)D levels may vary according to polymorphic variations in several genes involved in vitamin D metabolism and signaling pathways [51]. Another limitation is the lack of data on the family history of diabetes which can be a potential confounder. The annual health checkup did not also include the employees' dietary information, although due to consumption of meals at the factory restaurant served according to a certain meal plan, the participants' dietary habits could be assumed not to differ, considerably. Lastly, considering a specific group (factory workers) may limit the generalizability of our results to the entire population.

In conclusion, our results reveal a significant inverse association between vitamin D status and hyperglycemia in the factory workers. A serum 25(OH)D level of 14.7 ng/ml was suggested as an optimal cut-off point to predict hyperglycemia in this population. These findings along with the considerable proportion of participants with serum 25(OH)D levels below this threshold highlight the importance of evaluating vitamin

D status as a part of annual health checkup for employees to diagnose individuals with vitamin D deficiency/insufficiency. Subsequent vitamin D therapy may help prevent or delay type 2 diabetes mellitus in this group of workers.

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Compliance with ethical standards

Competing interests The authors declare to have no competing interests.

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