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Original Article

Association between serum 25-hydroxyvitamin D and adiposity measurements in Chinese young men with normal glucose tolerance

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Abstract: This study aimed to evaluate serum 25-hydroxyvitamin D [25(OH)D] levels in young Chinese men with normal glucose tolerance (NGT) and its relationship with adiposity measures. Method: A total of 159 normal-NGT men aged 25 to 40 years were enrolled, including 89 vitamin D (VD) deficiency men (25(OH)D < 50.0 nmol/l) and 70 non-VD deficiency men (25(OH)D ≥ 50.0 nmol/L). Body composition: total body fat mass (BF), truncal fat mass (TrF) were measured by using Dual-energy X-ray absorptionmetry (DEXA), BF% and TrF/BF % were calculated. TrF% and waist circumference (WC) were significantly higher in the VD deficiency group than in the non-VD deficiency group (P ≤ 0.001). In VD deficient patients, WC and TrF% correlated negatively with changes in 25(OH)D levels. In multiple linear regression (stepwise) models, higher TrF% (β = -0.768, 95% CI: -0.995 to -0.540, P = 0.000) and WC (β = -0.577, 95% CI: -0.792 to -0.362, P = 0.000) were associated with decreased 25(OH)D level. The area under the Operating characteristic curve (AUC) was 0.776 (95% CI: 0.700 to 0.853) of WC for prediction of vitamin D deficiency. The optimal cut off value of WC was 89.5 cm. Vitamin D deficiency is common among young Chinese young men with NGT, serum 25(OH)D level and adiposity are independently associated. WC may be a simple and efficient index to identify the risk of vitamin D deficiency in this population.

Keywords: Obesity, Vitamin D, waist circumference, body fat content

Introduction

Obesity and vitamin D (VD) deficiency have become two global public health problems. Obesity is widely recognized as the largest and fastest growing public health problem in the world. It is estimated that, by the year 2035, over 370 million people will be obese [1], and VD deficiency is also present in epidemic proportions globally, it is reported that about 10 million people suffer from this disease [2]. What's more, the prevalence of VD deficiency is increased with aging, long-term use of sunscreen products, sedentary lifestyle, inadequate exposure to sunlight, low consumption of food containing ergocalciferol, malabsorption of calcium and increase of latitude [3-5]. Besides acting as a regulatory hormone in calcium metabolism, VD is essential for noncalciotropic effects such as cellular differentiation and replication in many organs, adjusting immunity, reduce inflammation, as well as prevent tumors [6, 7]. Together, deficiencies in VD and calcium have been shown to negatively influence glycemia and studies consistently show associations with obesity, type 2 diabetes mellitus, insulin resistance (IR), and metabolic syndrome [8-10].

Previous studies have reported that 25-hydroxyvitamin D, or 25(OH)D, the commonly accepted indicator of vitamin D status, is inversely associated with adiposity and IR [11, 12]. In the study of Dasarathy et al., plasma VD concentration was significantly lower in 148 consecutive biopsy-proven patients with nonalcoholic fatty liver disease (NAFLD) (53.0 ± 26.0 mmol/L) compared with 39 healthy con-

trols (89.3 ± 15.0 mmol/L), and increased body fat mass (BMI) and higher NAFLD activity scores were all associated with lower plasma concentration of VD [13]. Another study also displays the similar result, the fatter patients has lower vitamin D levels [14]. However, results of other studies failed to confirm this [15-17]. For example, A cross-sectional study included 158 consecutive adults (121 females) with obesity (body mass index (BMI) 35.15 ± 2.8 kg/m^2), they did not detect any difference in 25(OH)D from moderate obesity patients [17]. Arunabh *et al*. reported that serum VD level was negatively associated with percentage of body fat, but it was not correlated with BMI in healthy black and white women [18]. These inconsistent results may because adiposity measures have been limited to indirect anthropometric measures as BMI. As we know, BMI is used to assess obesity, but it does not account for the distribution or composition of excess weight, it fails to distinguish increased weight related to muscle mass from adiposity. Thus, using indirect approaches to measure body fat may more accurately estimate the relationship between VD status and obesity. Other reasons for these discrepancies in study results may be ascribed to different study populations with varying ethnicity, sex, and age, or other variables. For example, a study included 435 residents (132 males) from St. Petersburg, Russia found VD insufficiency was found in 314 (72.2%) subjects, and serum 25(OH)D level inversely correlated with body weight, waist circumference (WC) and BMI in females but not in males [19].

In China, a cross-sectional study involved 1105 adults aged 20-70 years living in Tianjin, and all subjects use the bioelectrical impedance analysis method to estimate the Visceral fat area (VFA), they found higher VFA increases the risk of VD deficiency in men and pre-menopausal women, VD level is also negative associated with VFA in this population [20]. However, there still lack studies on VD and direct adiposity measurements in young Chinese men with NGT. Thus, a direct measuring method of body fat by using Dual-energy X-ray absorptiometry (DEXA), is required to reveal the association between serum 25(OH)D level and obesity. Therefore, this study was to examine serum 25(OH)D concentration and its association with adiposity in young Chinese men with NGT.

Subjects and methods

Study design

This prospective, case-control study enrolled 159 consecutive men who had received routine health examinations at the First Hospital of Qinhuangdao between December 2011 and February 2012 and who met the inclusion criteria (below). The study protocol was approved by the ethics committee of the Hebei Medical University. All subjects provided signed informed consent prior to study initiation.

Study subjects

Vitamin D was classified according 25(OH)D level as follows [2]: VD deficiency [25(OH)D < 50 nmol/L] and non-VD deficiency [25(OH)D ≥ 50 nmol/L]. Subjects included 89 VD deficient men (mean age 33.4 ± 4.7 years, 25(OH)D 39.0 ± 6.8 nmol/L) and 70 non-VD deficient men (mean age 33.4 ± 4.4 years, 25(OH)D 57.7 ± 5.5 nmol/L). Blood samples were drawn from all subjects between December 2011 and February 2012. All subjects were of Han ethnicity, and each underwent an oral glucose tolerance test (OGTT) initiated at 8:00 am with administration of 75 g of oral anhydrous glucose. Peripheral venous blood samples were taken at 0 and 120 min after glucose loading. Inclusion criteria were: (i) subjects were clinically stable with no previous medical history of diabetes, hypertension, dyslipidemia, coronary artery diseases, or cerebral stroke; (ii) subjects were obese with no clinical evidence of endocrinopathy; (iii) subjects had fasting plasma glucose (FPG) levels < 5.6 mmol/l and 2-h plasma glucose (2-h PG) levels < 7.8 mmol/L after a 75 g OGTT, based on the 2008 diagnostic criteria of the American Diabetes Association; and (iv) subjects were not taking medications known to affect glucose metabolism, including glucocorticoids, thyroid hormones, or thiazide diuretics. Exclusion criteria were: (i) subjects with hepatic or renal dysfunction, (ii) subjects taking vitamin and/or mineral supplements, and (iii) subjects with acute and chronic inflammation.

Measurements

A detailed history was taken for all enrolled subjects, including lifestyle behaviors (e.g., smoking, alcohol intake), and a physical examination was performed, including body weight (BW, kg),

Table 1. Subjects' baseline demographic and clinical characteristics

	VD deficiency (n = 89)	Non-VD deficiency (n = 70)	*T*	P
Age, year	33.4 ± 4.7	33.4 ± 4.4	0.048	0.962
SBP, mmHg	135.3 ± 9.4	128.7 ± 9.4*	4.411	0.000
DBP, mmHg	86.5 ± 8.4	82. 0 ± 6.7*	3.676	0.000
Scr, mmol/L	71.5 ± 8.7	73.2 ± 7.9	-1.280	0.202
Urea, umol/L	5.5 ± 1.3	5.6 ± 1.5	-0.653	0.515
UA, umol/L	327.7 ± 87.8	325.6 ± 86.2	0.151	0.880
TG, mmol/L	3.2 ± 1.5	1.8 ± 1.2*	6.296	0.000
TC, mmol/L	4.4 ± 1.3	3.7 ± 0.9*	4.075	0.000
HDL-C, mmol/L	1.2 ± 0.4	1.3 ± 0.4	-1.438	0.153
LDL-C, mmol/L	3.5 ± 1.2	2.8 ± 1.0*	4.039	0.000
FPG, mmol/L	5.0 ± 0.6	4.7 ± 0.6*	3.288	0.001
Plasma glucose 2-h OGTT, mmol/L	6.1 ± 1.2	5.5 ± 1.2*	3.585	0.000
HbA_{1c}, %	5.4 ± 0.6	5.0 ± 0.5*	4.214	0.000
Fasting insulin OGTT , uIU/mL	9.3 ± 3.8	5.4 ± 2.5*	6.815	0.000
HOMA-IR	2.1 ± 0.9	1.1 ± 0.6*	7.507	0.000
Calcium, mmol/L	2.3 ± 0.1	2.3 ± 0.1	-0.702	0.484
Phosphorus, mmol/L	1.2 ± 0.1	1.2 ± 0.1	0.350	0.727
PTH, pg/ml	38.4 ± 10.4	37.9 ± 9.1	0.316	0.752
25(OH)D, nmol/L	39.0 ± 6.8	57.7 ± 5.5*	-18.687	0.000
Smoking	41 (46.1)	30 (42.9)	0.660	0.416
Drinking	49 (55.1)	34 (48.6)	0.163	0.686

Data are presented as mean ± standard deviation, Categorical data are shown as frequency (%). Variables are tested by independent sample *t* test. Abbreviations: BW, body weight; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Scr, serum creatinine; UA, uric acid; TG, triglyceride; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; FPG, fasting plasma glucose; OGTT, oral glucose tolerance test; HOMA-IR, homeostasis model assessment insulin resistance; PTH, parathyroid hormone; 25(OH)D, 25-Hydroxyvitamin D. *Indicates significantly different from non-VD deficiency group, $P < 0.001$.

height (cm), and body mass index (BMI) calculated as kilograms per square meter ($kg \cdot m^{-2}$), waist circumference (WC), hip circumference (HC), waist/hip ratio (WHR, WC/HC), waist/height ratio (WHRT, WC/height), and blood pressure (BP). Weight in light clothing was measured, height was measured to the nearest 0.05 cm, WC and HC was evaluated to the nearest 0.01 cm. Diastolic blood pressure (DBP) and systolic blood pressure (SBP) measurements were recorded twice on the right arm using a standard mercury sphygmomanometer after 15 min rest while subjects were seated, and the average of the two measurements was used for analysis.

Plasma glucose levels were measured using the glucose oxidase method. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), and triglycerides (TG) were measured, as well as hepatic and renal function, using enzymatic assays with a Hitachi autoanalyzer (Hitachi, Tokyo, Japan). Serum 25(OH)D levels were estimated by radioimmunoassay procedure. The intra- and inter-assay coefficients of variation were 5.3% and 4.6%, respectively (IDS Corporation, Hampshire, UK). Plasma concentrations of insulin were measured by radioimmunological assay using a commercially available kit (North Institute of Biological Technology, Beijing, China). Insulin resistance was estimated from fasting plasma measurements using HOMA-IR (insulin (mU/L) × glucose (mmol/L)/22.5).

Body composition, total body fat mass (BF), body muscle mass (BM), truncal fat mass (TrF), upper limb fat mass (UF), limb fat mass (LF), android (A)% fat and gynoid (G)% fat were measured and determined by DEXA, (GE LUNAR company, America), BF% (BF/BW), TrF% (TrF/BF) and A/G were calculated. Subjects were made to lie supine on the system table, absent

Table 2. Subjects' anthropometric characters and body fat distribution

	VD deficiency (n = 89)	Non-VD deficiency (n = 70)	*t*	P
BMI, kg/m^2	28.1 ± 4.7	25.1 ± 4.1*	4.264	0.000
WC, cm	94.3 ± 7.7	86.0 ± 8.5*	6.408	0.000
HC, cm	96.1 ± 4.7	94.6 ± 4.7†	2.046	0.042
WHR	1.0 ± 0.1	0.9 ± 0.1*	8.049	0.000
WHTR	0.5 ± 0.0	0.5 ± 0.0*	6.103	0.000
Android, %	42.3 ± 7.3	35.2 ± 7.6*	5.471	0.000
Gynoid, %	32.2 ± 7.0	32.7 ± 7.6	-0.463	0.644
A/G	1.3 ± 0.2	1.1 ± 0.2*	8.460	0.000
BF, kg	25.9 ± 0.9	20.6 ± 0.9*	3.558	0.000
BF, %	30.2 ± 6.7	26.8 ± 7.5*	2.985	0.003
BM, kg	52.9 ± 6.3	52.2 ± 9.5	0.552	0.581
UF, kg	2.3 ± 0.1	2.2 ± 0.3	0.111	0.912
LF, kg	6.5 ± 2.9	6.4 ± 3.2	0.277	0.782
TrF, kg	16.5 ± 6.0	11.6 ± 5.4*	5.331	0.000
TrF, %	63.2 ± 4.4	55.8 ± 3.6*	11.548	0.000

Data are presented as mean ± standard deviation, Variables are tested by independent sample *t* test. Abbreviations: BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist hip ratio; WHTR, waist height ratio; A, android fat %; G, gynoid fat %; A/G, android gynoid ratio; BF, total body fat mass; BF%, total body fat mass percent; BM, body muscle mass; UF, upper limb fat mass; LF, limb fat mass; TrF, truncal fat mass; TrF%, truncal fat mass percent. *Indicates significantly different from non-VD deficient group, P < 0.001. †Indicates significantly different from non-VD deficient group, P < 0.05.

of any metal objects. Scanning condition is 77 kv and 0.75 mA, X ray efficient energy is 38 kev and 70 kev respectively, scan width is 52 cm, scan length is 195 cm, and scan rate is 8 cm/s (medium speed) (Supplementary Data).

Statistical analysis

Statistical analyses were performed using the SPSS 13.0. Continuous variables are reported as mean ± standard deviation. Categorical data are shown as frequency (%). Comparisons were done between the two groups using independent sample *t* test for continuous variables and chi-square test for categorical variables. Spearman's rank correlation was used to identify effectors of 25(OH)D level in univariable analysis. Multiple linear regression (stepwise) analyses were performed to examine the relationship between 25(OH)D level and other variables. The receiver operating characteristic (ROC) curve was used to identify the optimal cut off value of WC for the prediction of vitamin D deficient risk in this population. P < 0.05 was considered as statistically significant. Significance level was adjusted to 0.006 (0.05/8) as appropriate.

Results

Baseline characteristics of 159 subjects

A total of 159 males with age between 25 and 40 years old were enrolled in this study and classified into two groups based on VD levels, including VD deficient group of 89 men, and non-VD deficient group of 70 men. Subjects' baseline characteristics are summarized in **Table 1**. The two groups of patients shared similarities in age, serum creatinine, urea, UA, calcium, phosphorus, PTH, and proportions of patients who reported smoking and drinking.

SBP, DBP, TG, TC, LDL-C, FPG, 2-h G-OGTT, HbA_{1c}, fasting insulin OGTT and HOMA-IR were all significantly higher in the VD deficient group than in the non-VD deficient group (P ≤ 0.001). 25(OH)D level was significantly lower in the VD deficient group than in the non-VD deficient (P < 0.05).

The anthropometric parameters and body fat distribution measurements of two groups were compared. BMI, WC, HC, WHR, WHTR, Android %, A/G, BF, BF%, TrF and TrF% in VD deficient group were higher than in non-VD deficient group (all P < 0.05). No significant difference between the two groups in other variables, including Gynoid %, BM, UF, and LF (P > 0.05) (**Table 2**).

Effectors of VD

The correlation coefficients among 25(OH)D concentration, anthropometric parameters and body fat distribution measurements for all of the subjects were also shown in **Table 3**. 25(OH)D was negatively corrected with all anthropometric parameters investigated, including BMI (r = -0.556, P < 0.001), WC (r = -0.666, P < 0.001), HC (r = -0.336, P < 0.001), WHR (r = -0.695, P < 0.001), WHRT (r = -0.653, P < 0.001), and most adiposity distribution measures, such as android % (r = -0.607, P < 0.001), A/G (r = -0.657, P < 0.001), BF (r = -0.521, P <

Table 3. Univariate analysis of 25(OH)D in two groups subjects and all subjects by 25(OH)D levels (spearman)

	VD deficiency (n = 89)	Non-VD deficiency (n = 70)	All subjects (n = 159)
	γ_s (P)	γ_s (P)	γ_s (P)
BMI, kg/m^2	-0.592 (**0.000**)	-0.475 (**0.000**)	-0.556 (**0.000**)
WC, cm	-0.646 (**0.000**)	-0.469 (**0.000**)	-0.666 (**0.000**)
HC, cm	-0.463 (**0.000**)	-0.305 (**0.010**)	-0.336 (**0.000**)
WHR	-0.610 (**0.000**)	-0.439 (**0.000**)	-0.695 (**0.000**)
WHRT	-0.616 (**0.000**)	-0.498 (**0.000**)	-0.653 (**0.000**)
Android, %	-0.483 (**0.000**)	-0.475 (**0.000**)	-0.607 (**0.000**)
Gynoid, %	-0.142 (0.185)	-0.075 (0.535)	-0.019 (0.809)
A/G	-0.350 (**0.001**)	-0.473 (**0.000**)	-0.657 (**0.000**)
BF, kg	-0.513 (**0.000**)	-0.444 (**0.000**)	-0.521 (**0.000**)
BF%	-0.397 (**0.000**)	-0.393 (**0.001**)	-0.442 (**0.000**)
BM, kg	-0.241 (**0.023**)	0.043 (0.724)	-0.072 (0.365)
UF, kg	-0.336 (**0.001**)	-0.400 (**0.001**)	-0.364 (**0.000**)
LF, kg	-0.239 (**0.024**)	-0.317 (**0.008**)	-0.189 (**0.017**)
TrF, kg	-0.605 (**0.000**)	-0.484 (**0.000**)	-0.634 (**0.000**)
TrF, %	-0.705 (**0.000**)	-0.388 (**0.001**)	-0.817 (**0.000**)

Abbreviations: BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist hip ratio; WHTR, waist height ratio; A, android fat %; G, gynoid fat %; A/G, android gynoid ratio; BF, total body fat mass; BF%, total body fat mass percent; BM, body muscle mass; UF, upper limb fat mass; LF, limb fat mass; TrF, truncal fat mass; TrF%, truncal fat mass percent. Bold values indicate statistical significance, $P < 0.05$.

0.001), BF% (r = -0.442, P < 0.001), UF (r = -0.364, P < 0.001); LF (r = -0.189, P = 0.017), TrF (r = -0.634, P < 0.001) and TrF% (r = -0.817, P < 0.001), among these variables, the negative correlation between TrF and 25(OH)D_3 level was the strongest. When we separately analyzed VD deficiency and non-VD deficient subjects, 25(OH)D levels still negatively correlated with BMI, WC, HC, WHR, WHRT, android %, A/G, BF, BF%, UF, LF, TrF and TrF% in two groups. In subjects with VD deficiency, 25(OH)D levels also negatively related with BM levels (**Table 3**).

When 25(OH)D level was considered as the dependent variable in multiple regress analysis with BMI, WC, HC, Android %, UF, LF, BF% and TrF%, after adjusting age, drinking and smoking, the TrF% (β = -1.119, 95% CI: -1.361 to -0.877, P = 0.000) and WC (β = -0.521, 95% CI: -0.690 to -0.352, P = 0.000) maintained an independent association with 25(OH)D levels in all subjects, the TrF% (β = -0.768, 95% CI: -0.995 to -0.540, P = 0.000) and WC (β = -0.577, 95% CI: -0.792 to -0.362, P = 0.000) maintained independent association with 25(OH)D levels in VD deficiency. Higher TrF% and WC were associated with decreased in 25(OH)D levels (**Table 4**).

Operating characteristic (ROC) curves to VD deficient risk

The ROC curves were generated to identify the optimal point cut off WC for the prediction of vitamin D deficient risk in this population. The area under the ROC curve (AUC) was 0.776 (95% CI: 0.700 to 0.853) of WC for prediction of vitamin D deficient risk. We found the optimal cut off value of WC was 89.5 cm, and the sensitivity and specifity are 0.753 and 0.771 respectively (**Table 5**; **Figure 1**).

Discussion

In the present study, We evaluated serum 25(OH)D levels in young Chinese young men with NGT, its relationship between 25(OH)D with adiposity measurements and whether independent associations existed between serum vitamin D levels and parameters of adiposity. About 25(OH)D average level was 47.3 nmol/L, 56.0% (89/159) of subjects had deficient vitamin D levels. And the abnormalities of glucose and lipid metabolism of VD deficient men were more severe than non-VD deficient men. We also reported the indexes of obesity were negatively associated with plasma 25(OH)D levels. Moreover, higher TRF% and WC are the independent risk factors to vitamin D deficiency after adjusting for age, drinking and smoking in VD deficiency and all subjects. But BMI, the indicator often used to estimate adiposity, was not independently associated with 25(OH)D concentration.

Previous study have showed the prevalence of vitamin D deficiency as high as 64% in the general population [21], which is a litter higher than our result (56.0%), the reason of this difference may associated with age, gender and other factors. But our result of the prevalence of vitamin D deficiency was consisted by a study from the Fourth Korean National Health Nutrition and Examination Survey (KNHANES IV-2), which was conducted in 2008, the preva-

Table 4. Multiple linear regression analysis for 25(OH)D in young Chinese men with NGT (Stepwise Method)

	VD deficiency (n = 89)				All subjects (n = 159)			
	Unstandardized coefficients β	Std.error	P	95% CI	Unstandardized coefficients β	Std.error	P	95% CI
Constant	131.981	8.348	**0.000**	115.383 to 148.578	154.783	6.145	**0.000**	142.644 to 166.922
WC	-0.577	0.108	**0.000**	-0.792 to -0.362	-0.521	0.086	**0.000**	-0.690 to -0.352
TrF%	-0.768	0.114	**0.000**	-0.995 to -0.540	-1.119	0.123	**0.000**	-1.361 to -0.877

Dependent variable: 25(OH)D. Abbreviations: WC, waist circumference; TrF%, truncal fat mass percent. Bold values indicate statistical significance, P < 0.006.

Table 5. AUC and cutoff point of WC for prediction of vitamin D deficiency

	AUC	Cut-off point (cm)	Sensitivity	Specificity	95% CI
WC	0.776	89.5	0.753	0.771	0.700-0.853

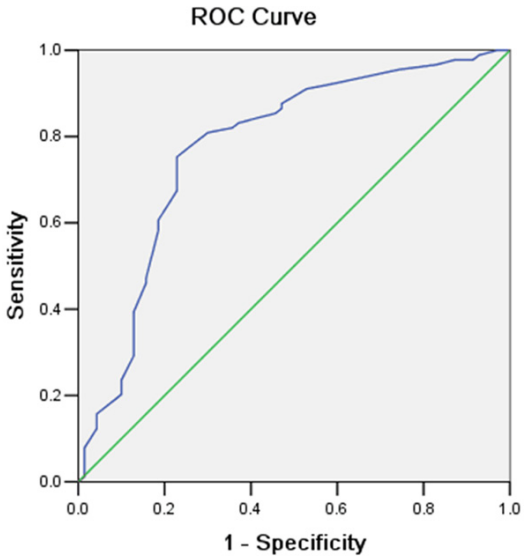

Figure 1. ROC curves of indicators for prediction of vitamin D deficiency.

lence of vitamin D insufficiency (< 50 nmol/L) was 56% in Korean adults over 19 years of age [22]. Poor vitamin D status was even shown in earlier studies of middle-aged and older Chinese individuals in Beijing [23] and Shenyang [24], respectively. Although little is known regarding to what extent the high prevalence of vitamin D deficiency in our population may be explained by certain environmental and/or genetic factors, geographic location and season may be factors in vitamin D status [25]. St. Petersburg, a north-western region of Russia, and many other regions of Russia, are located higher than 42° North latitude and have approximately 62 sunny days per year, which may deprive the population of sufficient sunlight and predispose people to vitamin D deficiencies. In Spain, vitamin D levels were measured in older women (72 ± 1.6 years) in winter and summer, with significantly lower levels shown for winter than for summer, specifically severely deficient levels (< 25 nmol/L) were found in roughly 28% of subjects, about twice the percentage found in summer [26]. In the present study, participants were selected during winter months, December 2011 to February 2012, from Qinhuangdao, a northern city in China with a relatively high latitude (39° North latitude). In that region, compared to low latitude cities, daily sun exposure time is short, and ultraviolet light is weak. Therefore, this may help to explain why 56% subjects had defecient 25(OH) D levels, it maybe more serious in other older populations. However, this was a small study and the study population may not be suitable for evaluating the prevalence of vitamin D deficiency. A large-scale population study must be done for this purpose. At least, our results do indicate that greater attention should be paid to improving vitamin D nutritional status in the Chinese population.

Body composition can reflect human nutrition status and level of body fat, fat is the major part of the body composition, fat accumulating too much can cause obesity. Various kinds of methods have been used in previous studies on the determination of body composition, BMI, WC, WHR, skinfold measurement, bioelectric impedance analysis etc. But the accuracy of these methods for estimating total body fat percentage is limited. Few studies have reported an inverse correlation between vitamin D and direct measurements of body fat distribu-

tion, especially, among young Chinese men with NGT. BMI is used to assess obesity, but it fail to distinguish increased weight related to muscle mass from adiposity. Actually, many patients with abdominal obesity suffer from hypertetion, hyperlipidemia or diabetes, their weight is normal and the BMI also in a normal range [27], So BMI abnormal or not can not accurately represent the body's fat metabolism and composition. There were inconstant results about VD level and the indirect anthropometric measures such as BMI and WC. For example, A study of 191 obese patients reported a high prevalence of vitamin D deficiency (48.7%) and vitamin D insufficiency (33.0%), with correlations shown between 25(OH)D levels and the degree of adiposity, and 25(OH)D levels were also correlated inversely with WC [28]. On the other hand, a Asian study enrolled 4,771 Korean adults, total body fat content was determined by DEXA, they found serum 25(OH)D concentration was independently associated with the total body fat content, but it may be not associated with WC and BMI [29]. DEXA is routinely used to asses bone mineral density, it systems generate X-rays at two energies and estimate the bone mineral content and the soft tissue composition. It is a noninvasive method with a low radiation dose. DXEA is now well used to measure body composition, and it provides valid and reliable information about fat tissue. According to the different position of adipose tissue distribution, obesity can be divided into two groups, which are central obesity and peripheral obesity. Many studies already have confirmed, the higher risk of metabolic disturbance induced central obesity than caused by peripheral obesity [30-33]. In our study, we use DEXA to measure to body fat distribution. As we know, WC is a index to evaluate the degree of central obesity, WC $\geq$ 90 cm in men is defined as central obesity [33]. TrF% is another parameter to reflect internal fat content. Our research found the fatter patients had lower vitamin D levels, and higher TrF% and WC were associated with decreased in 25(OH)D level, suggesting obesity especially central obesity is closely with 25(OH)D, which was supported by previous studies. The incidence of vitamin D deficiency was increased in obese individuals [34-36]. Tzotzas et al. found that 25(OH)D levels were lower in obese subjects compared with control subjects, and vitamin D levels increased after a 10% non-bariatric surgery-induced weight loss [36]. And another study about 25(OH)D level and obesity showed the plasma 25(OH)D concentration in obese group was significantly lower than non-obese group, and it was also negatively correlated with DEXA fat mass in Caucasian and African-American adults [37]. A China research included 3,890 nondiabetic individuals, subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) volumes were measured by multidetector computed tomography, they found he prevalence of vitamin D deficiency was threefold higher in those with high SAT and high VAT than in those with low SAT and low VAT, Vitamin D status is strongly associated with variation in subcutaneous and especially visceral adiposity [35]. In our study, we also didn't find the independent association between 25(OH)D and BMI, which was same as Kim's study [29], but it did to WC. This result was consisted with a prior study, in this study, they reported the risk of VD deficiency was 3.3 times than normal subject [38].

The adverse association between 25(OH)D level and obesity could be explained by the following points [39, 40]. First, vitamin D is the lipid-soluble vitamin induce increased storage of 25(OH)D in the adipose tissue and lower circulating levels of 25(OH)D in obese patients; Second, the bioavailability of vitamin D is decreased because of its storage in the adipose tissue; Third, 25(OH)D has also shown effects on lipogenesis and adipogenesis in adipose tissue, Whereas increasing adipose tissue can augment deposition of vitamin D in fat tissue which aggravated vitamin D deficiency. This potential mechanism is still not clear and needs further investigation.

Because 25(OH)D can't be tested by some hospital especially in rural hospital, and TrF is measured need DEXA which is also not acquired easy. However, WC is a index which can be simple measured, so we used ROC curves to identify the optimal cut off value of WC for the prediction of vitamin D deficient risk in this population. We found the optimal cut off value of WC was 89.5 cm, and the sensitivity and specifity were higher, WC maybe as a simple and efficient index to estimate the risk of vitamin D difeciency. However, due to the small sample, the further research is needed to do.

Limitations

The present study has several limitations. First, the sample was small, the study does not prove a causal relationship because the study was designed cross-sectionally; Second, we did not include the subjects' degree of smoking and drinking in the analysis of variables; Third, sunlight exposure and amount of activity were not measured. Larger prospective studies are needed to further investigate both the prevalence of vitamin D deficiency in Chinese populations and also the associations between vitamin D level and adiposity.

Conclusions

In conclusion, results of this study suggest that vitamin D deficiency is common among young Chinese young men with NGT and that 25(OH)D concentration was inversely associated with TrF% and WC in this population, suggesting that vitamin D level is independently related with adiposity. What's more, WC may be used to identify the risk of vitamin D deficiency in young Chinese men with NGT. Regardless of the cause-effect relationship between vitamin D deficiency and obesity, it is clear that the relationship affects many different parameters of public health in China and that results of our study are pertinent to improving both vitamin D status and related public health policy.

Disclosure of conflict of interest

None.

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References

[1] Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract 2014; 103: 137-149.

[2] Holick MF. Vitamin D deficiency. N Engl J Med 2007; 357: 266-281.

[3] Matyjaszek-Matuszek B, Lenart-Lipinska M, Wozniakowska E. Clinical implications of vitamin D deficiency. Prz Menopauzalny 2015; 14: 75-81.

[4] Adams JS, Hewison M. Update in vitamin D. J Clin Endocrinol Metab 2010; 95: 471-478.

[5] Bikle D. Nonclassic actions of vitamin D. J Clin Endocrinol Metab 2009; 94: 26-34.

[6] Santoro D, Caccamo D, Lucisano S, Buemi M, Sebekova K, Teta D, De Nicola L. Interplay of vitamin D, erythropoiesis, and the renin-angiotensin system. Biomed Res Int 2015; 2015: 145828.

[7] Salekzamani S, Neyestani TR, Alavi-Majd H, Houshiarrad A, Kalayi A, Shariatzadeh N, Gharavi A. Is vitamin D status a determining factor for metabolic syndrome? A case-control study. Diabetes Metab Syndr Obes 2011; 4: 205-212.

[8] Pittas AG, Chung M, Trikalinos T, Mitri J, Brendel M, Patel K, Lichtenstein AH, Lau J, Balk EM. Systematic review: Vitamin D and cardiometabolic outcomes. Ann Intern Med 2010; 152: 307-314.

[9] Mitri J, Muraru MD, Pittas AG. Vitamin D and type 2 diabetes: a systematic review. Eur J Clin Nutr 2011; 65: 1005-1015.

[10] Lamendola CA, Ariel D, Feldman D, Reaven GM. Relations between obesity, insulin resistance, and 25-hydroxyvitamin D. Am J Clin Nutr 2012; 95: 1055-1059.

[11] Laway BA KS, Shah ZA. Pattern of 25 hydroxy vitamin D status in North Indian people with newly detected type 2 diabetes: a prospective case control study. Indian J Endocrinol Metab 2014; 18: 726-730.

[12] Pham NM AS, Kurotani K, Nanri A, Sato M, Hayabuchi H, Yasuda K, Mizoue T. Serum 25-hydroxyvitamin D and markers of insulin resistance in a Japanese working population. Eur J Clin Nutr 2012; 66: 1323-1328.

[13] Dasarathy J, Periyalwar P, Allampati S, Bhinder V, Hawkins C, Brandt P, Khiyami A, McCullough AJ, Dasarathy S. Hypovitaminosis D is associated with increased whole body fat mass and greater severity of non-alcoholic fatty liver disease. Liver Int 2014; 34: e118-127.

[14] Shantavasinkul PC, Phanachet P, Puchaiwattananon O, Chailurkit LO, Lepananon T, Chanprasertyotin S, Ongphiphadhanakul B, Warodomwichit D. Vitamin D status is a determinant of skeletal muscle mass in obesity according to body fat percentage. Nutrition 2015; 31: 801-806.

[15] Pilz S, van den Hurk K, Nijpels G, Stehouwer CD, Van't Riet E, Kienreich K, Tomaschitz A, Dekker JM. Vitamin D status, incident diabetes and prospective changes in glucose metabolism in older subjects: the Hoorn study. Nutr Metab Cardiovasc Dis 2012; 22: 883-889.

[16] Tao MF, Zhang Z, Ke YH, He JW, Fu WZ, Zhang CQ, Zhang ZL. Association of serum 25-hy-

droxyvitamin D with insulin resistance and beta-cell function in a healthy Chinese female population. Acta Pharmacol Sin 2013; 34: 1070-1074.
[17] Al Masri M, Romain AJ, Boegner C, Maimoun L, Mariano-Goulart D, Attalin V, Leprieur E, Picandet M, Avignon A, Sultan A. Vitamin D status is not related to insulin resistance in different phenotypes of moderate obesity. Appl Physiol Nutr Metab 2017; 42: 438-442.
[18] Arunabh S, Pollack S, Yeh J, Aloia JF. Body fat content and 25-hydroxyvitamin D levels in healthy women. J Clin Endocrinol Metab 2003; 88: 157-161.
[19] Karonova T, Belyaeva O, Jude EB, Tsiberkin A, Andreeva A, Grineva E, Pludowski P. Serum 25(OH)D and adipokines levels in people with abdominal obesity. J Steroid Biochem Mol Biol 2018; 175: 170-176.
[20] Zhang M, Li P, Zhu Y, Chang H, Wang X, Liu W, Zhang Y, Huang G. Higher visceral fat area increases the risk of vitamin D insufficiency and deficiency in Chinese adults. Nutr Metab (Lond) 2015; 12: 50.
[21] Anderson JL, May HT, Horne BD, Bair TL, Hall NL, Carlquist JF, Lappe DL, Muhlestein JB; Intermountain Heart Collaborative (IHC) Study Group. Relation of vitamin D deficiency to cardiovascular risk factors, disease status, and incident events in a general healthcare population. Am J Cardiol 2010; 106: 963-968.
[22] Kim J. Dairy food consumption is inversely associated with the risk of the metabolic syndrome in Korean adults. J Hum Nutr Diet 2013; 26 Suppl 1: 171-179.
[23] Xue Y. [Serum levels of 25-hydroxyvitamin D in normal Beijing subjects]. Zhonghua Yu Fang Yi Xue Za Zhi 1991; 25: 177-179.
[24] Yan L, Zhou B, Wang X, D'Ath S, Laidlaw A, Laskey MA, Prentice A. Older people in China and the United Kingdom differ in the relationships among parathyroid hormone, vitamin D, and bone mineral status. Bone 2003; 33: 620-627.
[25] Lagunova Z, Porojnicu AC, Lindberg F, Hexeberg S, Moan J. The dependency of vitamin D status on body mass index, gender, age and season. Anticancer Res 2009; 29: 3713-3720.
[26] Rodriguez Sangrador M, Beltran de Miguel B, Quintanilla Murillas L, Cuadrado Vives C, Moreiras Tuny O. The contribution of diet and sun exposure to the nutritional status of vitamin D in elderly Spanish women: the five countries study (OPTIFORD Project). Nutr Hosp 2008; 23: 567-576.
[27] Jensen MD. Role of body fat distribution and the metabolic complications of obesity. J Clin Endocrinol Metab 2008; 93: S57-63.
[28] Boonchaya-anant P, Holick MF, Apovian CM. Serum 25-hydroxyvitamin D levels and metabolic health status in extremely obese individuals. Obesity (Silver Spring) 2014; 22: 2539-2543.
[29] Kim D, Kim J. Association of serum 25-hydroxyvitamin D and parathyroid hormone with hypertension in middle-aged and older korean adults. Am J Hypertens 2016; 29: 96-103.
[30] Urquidez Romero R, Murguia Romero M, Esparza Romero J, Diaz Torres BA, Rodriguez Tadeo A, Medrano Donlucas G, Ramos Jimenez A, Wall Medrano A, Gallardo Ortiz IA, Tapia Pancardo D. Abdominal obesity is strongly associated to blood pressure in young Mexicans. Nutr Hosp 2017; 34: 357-362.
[31] Esmaillzadeh A, Mirmiran P, Moeini SH, Azizi F. Larger hip circumference independently contributed to reduced metabolic risks in Tehranian adult women. Int J Cardiol 2006; 108: 338-345.
[32] Pankow JS, Kwan DK, Duncan BB, Schmidt MI, Couper DJ, Golden S, Ballantyne CM. Cardiometabolic risk in impaired fasting glucose and impaired glucose tolerance: the atherosclerosis risk in communities study. Diabetes Care 2007; 30: 325-331.
[33] Alberti KG, Zimmet P, Shaw J; IDF Epidemiology Task Force Consensus Group. The metabolic syndrome--a new worldwide definition. Lancet 2005; 366: 1059-1062.
[34] Chacko SA, Song Y, Manson JE, Van Horn L, Eaton C, Martin LW, McTiernan A, Curb JD, Wylie-Rosett J, Phillips LS. Serum 25-hydroxyvitamin D concentrations in relation to cardiometabolic risk factors and metabolic syndrome in postmenopausal women. Am J Clin Nutr 2011; 94: 209-217.
[35] Cheng S, Massaro JM, Fox CS, Larson MG, Keyes MJ, McCabe EL, Robins SJ, O'Donnell CJ, Hoffmann U, Jacques PF. Adiposity, cardiometabolic risk, and vitamin D status: the Framingham heart study. Diabetes 2010; 59: 242-248.
[36] Tzotzas T, Papadopoulou FG, Tziomalos K, Karras S, Gastaris K, Perros P, Krassas GE. Rising serum 25-hydroxy-vitamin D levels after weight loss in obese women correlate with improvement in insulin resistance. J Clin Endocrinol Metab 2010; 95: 4251-4257.
[37] Parikh SJ, Edelman M, Uwaifo GI, Freedman RJ, Semega-Janneh M, Reynolds J, Yanovski JA. The relationship between obesity and serum 1,25-dihydroxy vitamin D concentrations in healthy adults. J Clin Endocrinol Metab 2004; 89: 1196-1199.
[38] Theuri G, Kiplamai F. Association between vitamin D levels and central adiposity in an east-

ern Africa outpatient clinical population. Dermatoendocrinol 2013; 5: 218-221.
[39] Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF. Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr 2000; 72: 690-693.
[40] Earthman CP, Beckman LM, Masodkar K, Sibley SD. The link between obesity and low circulating 25-hydroxyvitamin D concentrations: considerations and implications. Int J Obes (Lond) 2012; 36: 387-396.