

Association Between Bone Mineral Density and Pancreatic β -Cell Function in Elderly Men and Postmenopausal Women

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Purpose: To explore the association between bone mineral density (BMD) and β -cell function.

Methods: A cross-sectional study was performed in Fujian, China, from 2011 to 2012. The study included 572 elderly men older than age 60 years and 1558 postmenopausal women aged 45 to 86 years, excluding those with diabetes and insulin resistance. Fasting glucose and insulin concentrations were measured. Pancreatic β -cell function was estimated by using the homeostasis model assessment (HOMA- β). Calcaneus BMD was measured by using quantitative ultrasonography. Multiple regression analyses were applied to explore the association.

Results: Participants with decreased BMD had lower fasting glucose ($P < 0.001$ in postmenopausal women; $P = 0.007$ in elderly men) and greater HOMA- β ($P = 0.001$ in postmenopausal women; $P = 0.008$ in elderly men) than those with normal BMD, whereas no statistical differences in insulin were seen among categories of BMD. After adjustment for all confounders, HOMA- β was still significantly negatively related to BMD in both groups (all $P < 0.001$), and remarkable positive relationships were found between BMD and fasting glucose. Furthermore, binary logistic regression presented fully adjusted odds ratios for diabetes in those with osteoporosis vs those with normal BMD: 0.60 [95% confidence interval (CI), 0.38 to 0.94] and 0.66 (95% CI, 0.49 to 0.91) in the original selected population of elderly men ($n = 1070$) and postmenopausal women ($n = 2825$), respectively.

Conclusions: BMD was independently inversely associated with HOMA- β and positively associated with fasting glucose in both elderly men and postmenopausal women, suggesting that bone mass may be a predictor of glucose metabolism. Further research is needed to verify the associations and determine the exact mechanism underlying them.

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Freeform/Key Words: bone mineral density, β -cell function

Impaired β -cell function and insulin resistance are both involved in the pathogenesis of type 2 diabetes mellitus. It has been reported that, compared with insulin resistance, β -cell dysfunction plays the predominant role in the development of diabetes in some individuals [1, 2].

Abbreviations: BMD, bone mineral density; BMI, body mass index; CI, confidence interval; DBP, diastolic blood pressure; FBG, fasting blood glucose; FIns, fasting insulin; HOMA- β , homeostasis model assessment-estimated β -cell function; MET, metabolic equivalent; OR, odds ratio; OST-PTP, osteostesticular tyrosine phosphatase; QUS, quantitative ultrasound; REACTION, Risk Evaluation of cAncers in Chinese diabeTic Individuals, a lONgitudinal [study]; WHR, waist-to-hip ratio.

Most previous studies have shown that insulin resistance is associated with bone mineral density (BMD), and the conclusions are inconsistent. Arikian *et al.* [3] reported that in patients with type 2 diabetes, remarkable insulin resistance had a negative effect on BMD. Similarly, a cross-sectional study conducted in Korean men demonstrated that bone mass was inversely related to insulin resistance [4]. In contrast, a few studies found that insulin resistance could be a protective factor for bone health [5, 6]. However, to our knowledge, no study to date has examined the association between BMD and pancreatic β -cell function [homeostasis model assessment–estimated β -cell function (HOMA- β)], especially in Asian populations, which have an increasingly higher prevalence of osteoporosis and diabetes compared with Western populations [7, 8].

Therefore, the aim of the current study was to investigate the relationship between BMD and β -cell function by using data from the baseline survey of the REACTION study on Chinese persons in Fujian, China. We restricted our participants to elderly men and postmenopausal women in order to eliminate the effects of sex and hormonal change with different ages and menstrual status.

1. Materials and Methods

A. Study Participants

This cross-sectional study was performed on individuals recruited from two cities (Ningde and Wuyishan) in Fujian, China, from 2011 to 2012. It was part of the baseline survey of the REACTION (Risk Evaluation of cAncers in Chinese diabeTic Individuals: a lONGitudinal) study, which investigated the association between diabetes and risk for cancer. The design and methods of the REACTION study have been described in detail elsewhere [9]. Inclusion criteria were age 60 years or older for men and, among women, cessation of menstruation for at least 1 year. A total of 5083 participants were enrolled in our study. We excluded those with diabetes ($n = 1181$) diagnosed according to the 1999 World Health Organization criteria [10]. Previous studies [11] have shown that the disposition index, calculated from the product of insulinogenic index and the Matsuda index, is a well-accepted measure of pancreatic β -cell function adjusted for insulin resistance. In contrast, for our study we selected HOMA- β for estimating β -cell function. Therefore, we also excluded persons with insulin resistance ($n = 684$), defined as homeostasis model assessment–estimated insulin resistance [fasting blood glucose (FBG) \times FIns/22.5] > 2.41 [12]. Individuals were also excluded if they met one of the following conditions: (1) self-reported history of cardiovascular, liver, or kidney diseases or malignancies ($n = 279$); (2) diseases of the thyroid, rheumatoid arthritis, or pituitary disorders ($n = 235$); and (3) incomplete informations ($n = 378$). In addition, individuals who took such medications as steroid hormones, bisphosphonates, and other drugs that affect bone and pancreas metabolism were excluded ($n = 196$). In the end, 572 elderly men and 1558 postmenopausal women were included in the present study. All participants provided written informed consent, and the Ethics Committee of Fujian Provincial Hospital approved the study.

B. Clinical and Anthropometric Measurements

Each participant underwent a face-to-face interview and completed a detailed questionnaire that solicited information on age, sex, lifestyle factors, medical histories, and other relevant social or dietary information. Cigarette smoking and alcohol consumption habits were categorized into three levels: former (those who had consumed previously but had not consumed for ≥ 1 year), current (those who had consumed in the last year), and never. Physical activity was assessed by using metabolic equivalent (MET) minutes per week according to the Global Physical Activity Questionnaire and thus was classified as low (< 600 MET), moderate (≥ 600 to < 3000 MET), and heavy (≥ 3000 MET). Body weight and height were measured with the participants not wearing shoes and wearing light clothing, to the nearest kilogram and

centimeter, respectively. Body mass index (BMI) was calculated as body weight in kilograms divided by height in meters squared. Waist circumference was measured at the middle point between the lower costal margin and the iliac crest. Hip circumference was measured at the level of the greater trochanters. Waist-to-hip ratio (WHR) was defined as the ratio of waist circumference to hip circumference. Blood pressure was measured by using a sphygmomanometer on the right upper arm after 30 minutes of rest. All the procedures were performed by experienced operators.

C. Laboratory Measurements

After an overnight fast for at least 10 hours, venous blood samples were collected. A standardized 75-g oral glucose tolerance test was given to all participants between 8:00 AM and 9:00 AM. Blood glucose and insulin levels were determined at 0 and 120 minutes after the administration of glucose. The plasma levels of fasting blood glucose were measured by using the glucose oxidase technique (Changchun Huili Biotech Co. Ltd, Changchun, China). Fasting insulin levels were measured by the radioimmunoassay method (Linco Research, St. Charles, MO). Serum total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol were tested with an autoanalyzer (Abbott Laboratories, Lake Bluff, IL). To estimate β -cell function, HOMA was used on the basis of fasting blood glucose and FIns concentrations. HOMA- β was calculated according to the following formula:

$$\text{HOMA-}\beta = [20 \times \text{Fins } (\mu\text{IU/mL})]/[\text{FBG } (\text{mmol/L}) - 3.5].$$

D. BMD Measurement

BMD was measured on the dominant calcaneus by quantitative ultrasonography (QUS) using the Achilles Express Ultrasound device (GE Healthcare, Waukesha, WI). All the measurements were performed on the same machine by the same experienced operator in strict accordance with the manufacturer's recommendations. T-scores for BMD were calculated on the basis of the database of a healthy young adult Asian population provided by the manufacturer. Because of the inapplicability of the World Health Organization criteria for QUS, participants were classified into three groups according to revised criteria for QUS [13]: normal BMD (T-score ≥ -0.5), osteopenia (T-score < -0.5 but > -1.8), osteoporosis (T-score ≤ -1.8).

E. Statistical Analysis

Statistical analyses were performed by using SPSS software, version 17.0 (IBM, Chicago, IL). Variables were presented as means (standard deviations) for normal distribution, as median (interquartile ranges) for nonnormal distribution, or as percentages for categorical variables. Differences in participants among three categories of BMD were examined with one-way analysis of variance, the Kruskal–Wallis test, or the χ^2 test as appropriate. *Post hoc* comparisons were carried by using Bonferroni correction. Multiple linear regression was applied to test for a linear trend, using the natural logarithm–transformed β -cell function (HOMA- β) as the dependent variable. In the multivariate analysis, we used stepwise regression to avoid the multicollinearity within covariates (the criteria of $P = 0.10$ for a variable to enter and $P = 0.15$ for a variable to be removed). Two regression models were established to adjust for the confounding factors. In model 1, we adjusted for age and body mass index. In model 2, we further adjusted for low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, serum total cholesterol, triglycerides, WHR, years since menopause (postmenopausal women), smoking status, alcohol consumption, and physical activity. Results were presented as standardized β coefficients and P values.

In addition, for the sake of verifying our results, we used a binary logistic regression model in the original selected population covering those with diabetes and those with insulin resistance ($n = 1070$ elderly men and $n = 2825$ postmenopausal women) to evaluate the adjusted

odds ratios (ORs) and 95% confidence intervals (CIs), including all confounders in model 2 for the incidence of diabetes across categories of BMD, with normal BMD as the reference. All *P* values reported were two tailed, and *P* < 0.05 was considered to represent a statistically significant difference.

2. Results

The baseline characteristics of 572 elderly men and 1558 postmenopausal women with normal BMD, osteopenia, and osteoporosis are shown in [Tables 1 and 2](#), respectively. Both male and female participants with lower BMD were more likely to be older (*P* < 0.001) and to have lower BMI (*P* < 0.001) and fasting glucose levels (*P* < 0.01). With bone mass decreased, individuals were predisposed to have greater pancreatic β -cell function (HOMA- β) (all *P* values for trend < 0.01). Compared with women who had normal BMD, those with bone loss had lower high-density lipoprotein cholesterol (*P* = 0.001) and diastolic blood pressure (*P* = 0.002); this relationship was not seen among men. In addition, alcohol consumption was associated with increased BMD (*P* < 0.05) in all participants. However, there were no significant differences in fasting insulin, WHR, triglycerides, total cholesterol, low-density lipoprotein cholesterol, and other lifestyle variables (such as smoking and physical activity) among different categories of BMD (all *P* > 0.05).

As presented in [Table 3](#), there were significant negative associations between BMD and logarithm-transformed HOMA- β in both elderly men and postmenopausal women before and

Table 1. Characteristics of Elderly Men With Normal BMD, Osteopenia, and Osteoporosis

Characteristic	Normal BMD	Osteopenia	Osteoporosis	<i>P</i> Value
Participants, n	275	182	115	
Age, y	65.5 (4.6)	67.1 (5.4) ^a	68.9 (6.1) ^{a,b}	<0.001
BMI, kg/m ²	23.4 (2.8)	22.8 (2.9) ^a	22.1 (2.8) ^{a,b}	<0.001
HDL cholesterol, mmol/L	1.34 (1.15–1.65)	1.33 (1.09–1.86)	1.40 (1.20–1.61)	0.528
LDL cholesterol, mmol/L	2.94 (0.75)	2.86 (0.81)	2.89 (0.93)	0.590
TC, mmol/L	5.08 (0.93)	4.92 (1.03)	4.93 (1.13)	0.184
TG, mmol/L	1.17 (0.87–1.74)	1.13 (0.80–1.58)	1.07 (0.85–1.58)	0.086
FBG, mmol/L	5.53 (5.20–5.99)	5.49 (5.05–5.90) ^a	5.32 (4.95–5.71) ^{a,b}	0.007
FIns, μ U/mL	7.36 (2.24)	7.65 (2.12)	7.46 (2.29)	0.389
HOMA- β	78.51 (64.31–96.28)	83.82 (69.56–108.34) ^a	88.17 (74.88–114.50) ^{a,b}	0.008
Smoker, %				0.358
Never	47.3	56.1	55.6	
Former	34.5	28.0	29.6	
Current	18.2	15.9	14.8	
Alcohol consumption, %				0.007
Never	66.9	73.6 ^a	73.9 ^a	
Former	28.7	16.5	20.9	
Current	4.4	9.9	5.2	
Physical activity, %				0.212
Low	80.7	87.4	87.8	
Moderate	8.0	3.8	5.2	
High	11.3	8.8	7.0	
SBP, mmHg	141.0 (19.6)	141.5 (21.7)	136.5 (18.6)	0.086
DBP, mmHg	77.6 (10.8)	76.3 (10.9)	75.4 (10.2)	0.153
WHR	0.89 (0.06)	0.88 (0.06)	0.88 (0.06)	0.677

Data are presented as means (standard deviation), median (interquartile ranges), or percentages. One-way analysis of variance or Kruskal–Wallis test was used for continuous data and Pearson χ^2 test was used for categorical data. *Post hoc* tests were performed by using Bonferroni correction.

Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TC, total cholesterol; TG, triglycerides; SBP, systolic blood pressure.

^a*P* < 0.05 compared with normal BMD group.

^b*P* < 0.05 compared with osteopenia group.

Table 2. Characteristics of Postmenopausal Women With Normal BMD, Osteopenia, and Osteoporosis

Characteristic	Normal BMD	Osteopenia	Osteoporosis	P Value
Participants, n	732	491	335	
Age, y	56.0 (6.5)	59.0 (6.7) ^a	62.3 (8.5) ^{a,b}	<0.001
BMI, kg/m ²	24.0 (2.9)	23.5 (3.1) ^a	23.0 (2.9) ^{a,b}	<0.001
HDL cholesterol, mmol/L	1.46 (0.34)	1.39 (0.33) ^a	1.41 (0.35) ^a	0.001
LDL cholesterol, mmol/L	3.20 (0.81)	3.15 (0.82)	3.09 (0.90)	0.138
TC, mmol/L	5.43 (1.04)	5.31 (1.06)	5.29 (1.17)	0.065
TG, mmol/L	1.31 (0.95–1.79)	1.18 (0.92–1.73)	1.21 (0.92–1.69)	0.066
FBG, mmol/L	5.40 (5.03–5.80)	5.28 (4.87–5.70) ^a	5.22 (4.78–5.61) ^{a,b}	<0.001
FIns, μ U/mL	7.82 (2.99)	7.89 (3.07)	7.85 (3.15)	0.843
HOMA- β	80.08 (62.86–102.42)	84.41 (64.73–116.83) ^a	88.38 (67.15–118.95) ^{a,b}	0.001
Smoker, %				0.554
Never	99.3	99.8	99.1	
Former	0.5	0.2	0.9	
Current	0.1	0	0	
Alcohol consumption, %				0.045
Never	95.8	97.6 ^a	97.9 ^a	
Former	4.2	2.0	1.8	
Current	0	0.4	0.3	
Physical activity, %				0.793
Low	91.4	92.7	92.5	
Moderate	5.3	4.5	5.4	
High	3.3	2.8	2.1	
SBP, mmHg	133.1 (19.3)	133.7 (19.7)	133.7 (19.7)	0.849
DBP, mmHg	76.5 (10.6)	75.3 (10.5) ^a	74.1 (10.1) ^a	0.002
YSM, y	34.0 (31.0–36.0)	33.0 (31.0–36.0) ^a	33.0 (30.0–35.0) ^a	0.003
WHR	0.86 (0.06)	0.86 (0.06)	0.87 (0.06)	0.483

Data are presented as means (standard deviation), median (interquartile ranges), or percentages. One-way analysis of variance or Kruskal–Wallis test was used for continuous data and Pearson χ^2 test was used for categorical data. *Post hoc* tests were performed by using Bonferroni correction.

Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TC, total cholesterol; TG, triglycerides; SBP, systolic blood pressure; YSM, years since menopause.

^a $P < 0.05$ compared with normal BMD group.

^b $P < 0.05$ compared with osteopenia group.

after controlling for the effects of various confounding factors (all $P < 0.01$). Compared with model 1, the correlation results remained significant after adjustment for all potential confounders in model 2, although the strength of the correlations diminished somewhat. In addition, markedly positive associations between BMD and fully adjusted fasting glucose in model 2 were observed ($\beta = 0.199$ and $P = 0.019$ in elderly men; $\beta = 0.140$ and $P < 0.001$ in postmenopausal women).

Table 4 shows adjusted ORs of diabetes across categories of BMD in the original selected population of elderly men ($n = 1070$) and postmenopausal women ($n = 2825$) separately, including those with diabetes and insulin resistance. Low BMD was associated with a decreased risk for developing diabetes in both men and women after adjustment for all covariates (for both, P for trend < 0.001). Compared with individuals with normal BMD, individuals with osteoporosis had 40% and 34% lower risk for diabetes development [OR, 0.60 (95% CI, 0.38 to 0.94), P for trend = 0.028 in elderly men; OR, 0.66 (95% CI, 0.49 to 0.91), P for trend = 0.011 in postmenopausal women].

3. Discussion

A bone–islet axis exists in the body. Insulin receptor signaling in the osteoblast promotes osteoblast differentiation and simultaneously increases osteocalcin secretion, which leads to regulation of insulin secretion in the pancreas and controls glucose homeostasis [14]. *In vivo*

Table 3. Multiple Linear Regression Results of Associations Between BMD and HOMA- β

Variable	LN (HOMA- β) ^a			
	Elderly Men		Postmenopausal Women	
	Standardized β	P Value	Standardized β	P Value
BMD unadjusted	-0.131	0.002	-0.115	<0.001
Model 1 ^b	-0.152	<0.001	-0.163	<0.001
Model 2 ^c	-0.150	<0.001	-0.154	<0.001

^aValues converted to natural logarithmic scale were used as the dependent variables.

^bModel 1 was adjusted for age and body mass index.

^cModel 2 was adjusted for low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, total cholesterol, triglycerides, WHR ratio, years since menopause (postmenopausal women), smoking status, alcohol consumption, and physical activity in addition to factors included in model 1.

and *in vitro* studies demonstrated that Gprc6a, the osteocalcin-sensing receptor, is highly expressed in pancreatic β cells and helps regulate the response of circulating osteocalcin in the bone-pancreas endocrine loop [15]; deletion of this receptor was associated with bone loss, glucose intolerance, and insulin resistance [16]. Moreover, a US study by Pi and Quarles [17] also showed that Gprc6a defined a molecular mechanism linking bone metabolism with metabolic regulation of β cells. All of the previous findings support the existence of the bone-pancreas loop (Fig. 1). However, to date, most studies regarding the connection in the bone-pancreas loop have been conducted in animal models; similar evidence in humans is scarce.

In the current study, we observed that BMD were associated inversely with pancreatic β -cell function (HOMA- β) and positively with fasting plasma glucose levels in both elderly men and postmenopausal women, independent of insulin resistance and diabetes. The results were consistent even after adjustment for other potential confounding factors, such as age, BMI, WHR, lipid profiles, and lifestyle change. These findings suggest that bone mass seems to be a predictor of glucose metabolism.

This study investigated the association between BMD and pancreatic β -cell function as assessed by HOMA- β in an Asian population. The exact mechanism responsible for the association remains unclear. It is well documented that in addition to its traditional function, the skeleton is increasingly being recognized as an endocrine organ secreting osteocalcin, an osteoblast-specific hormone. The uncarboxylated form of osteocalcin, but not the carboxylated one, can induce β -cell proliferation, improve insulin sensitivity, and regulate glucose metabolism [18]. However, other studies reported that both uncarboxylated and carboxylated osteocalcin increased glucose transport in a rat model [19] and were associated with blood glucose levels in type 2 diabetes [20]. Analogous to the latter survey, a retrospective cohort study conducted in Chinese individuals also indicated that serum osteocalcin was inversely

Table 4. Adjusted ORs (95% CIs) for Incident Diabetes According to Categories of BMD in Primary Selected Population of Elderly Men and Postmenopausal Women

Category of BMD	Elderly Men (n = 1070)		Postmenopausal Women (n = 2825)	
	Diabetes (%)	Adjusted OR (95% CI)	Diabetes (%)	Adjusted OR (95% CI)
Normal BMD	14.3	1.00 (reference)	10.5	1.00 (reference)
Osteopenia	12.5	0.72 (0.48–1.07)	11.0	0.79 (0.57–1.04)
Osteoporosis	10.1	0.60 (0.38–0.94)	11.4	0.66 (0.49–0.91)
P for trend	—	<0.001	—	<0.001

Covariates included in the model were age, BMI, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, total cholesterol, triglycerides, WHR, years since menopause (postmenopausal women), smoking status, alcohol consumption, and physical activity.

correlated with fasting plasma glucose levels and positively correlated with homeostasis model assessment of β -cell function (HOMA- β) [21].

Although these studies had inconsistent results, they implied a common viewpoint: Irrespective of its form, osteocalcin contributes to hyperglycemia control and the improvement of pancreatic β -cell function. On the other hand, it has been well established that the level of osteocalcin is significantly higher in postmenopausal osteoporotic women than in nonosteoporotic persons [22]. Similarly, in a cross-sectional study, Melton *et al.* [23] found that serum osteocalcin was inversely associated with BMD at diverse sites in postmenopausal women without estrogen treatment after adjustment for age. Additionally, other researchers have shown that elevated bone turnover markers, including osteocalcin, are associated with greater bone loss [24–26] and reduced diabetes risk [27] after adjustment for confounding variables in elderly men.

Given these findings, we speculated that the aforementioned statements may be able to primarily explain our observed negative and positive associations between BMD and β -cell function and fasting plasma glucose in our participants, respectively, despite the existence of other mechanisms underlying the associations yet to be detected. Nevertheless, because previous studies have reported that the correlations between bone turnover markers and BMD are weaker in men than in women [24, 26, 28], we therefore postulate that there may exist another main mechanism accounting for the associations between BMD and β -cell function and fasting glucose in elderly men, different from that in postmenopausal women. For example, besides osteocalcin, it is possible that the skeleton extensively produces other hormones that help improve pancreatic β -cell function and glucose homeostasis in elderly bone loss among men. This possibility should be researched further.

In addition, osteotesticular tyrosine phosphatase (OST-PTP), a novel receptor-like protein tyrosine phosphatase, is expressed specifically in bone and testes. *In vivo* and *in vitro*

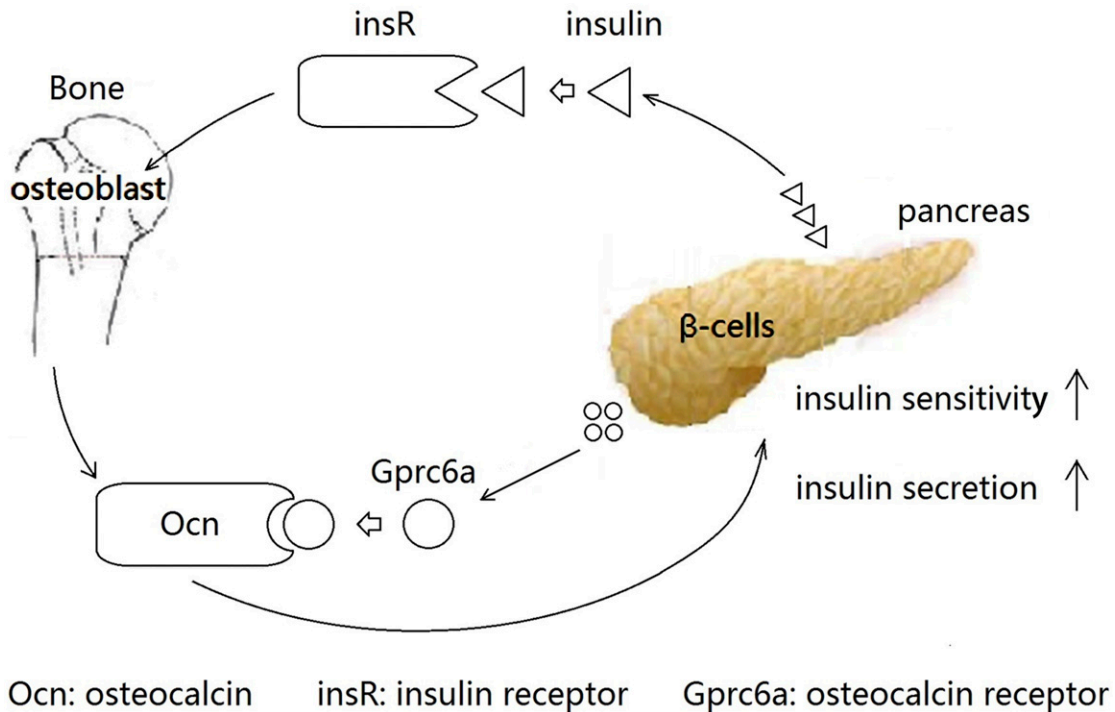


Figure 1. Interaction between bone and pancreas. Insulin released from pancreatic β -cell activates insulin receptor (insR) in the osteoblast to promote osteoblast differentiation and simultaneously increase osteocalcin (Ocn) secretion. Gprc6a, the osteocalcin-sensing receptor, is highly expressed in the pancreatic β cell. Ocn released from osteoblast targets Gprc6a to increase insulin secretion and promote insulin sensitivity.

experiments demonstrated that mice lacking OST-PTP presented elevated β -cell proliferation, insulin sensitivity, and glucose tolerance [18]. On the basis of this finding, it is hypothesized that there is a link between BMD and the expression of OST-PTP and that OST-PTP can become a mediator for the association between β -cell function and bone mass in both groups of participants, and these may extend our explanations. Future studies focusing on these aspects are warranted.

To verify our results, we also estimated the risk for prevalent diabetes using multivariable logistic regression analysis in the primary selected sample of elderly men ($n = 1070$) and postmenopausal women ($n = 2825$) separately and found that a trend toward decreased risk for diabetes was correlated with bone loss; this was analogous to the results of an Australian survey by Yeap *et al.* [27] revealing that higher bone remodeling rates were regarded as markers for osteoporosis yet were associated with reduced diabetes risk in older men. In our study, compared with participants with normal BMD, those with osteoporosis had lower risk for incident diabetes after controlling for the effects of various confounders. These observations provide better evidence for the inverse correlation between BMD with pancreatic β -cell function.

The current study had several limitations. First, our study had a cross-sectional design, and thus we were unable to ascertain the cause-and-effect relationship between BMD and pancreatic β -cell function. Second, the participants were limited to elderly men and postmenopausal women in China; therefore, the findings may not be generalizable to different ethnic populations. Third, bone status was assessed by using QUS, which is not recognized as an accepted gold standard for BMD measurement. However, it does have several advantages, such as low cost, portability, ease of use, and lack of radiation. Fourth, we did not measure osteocalcin, which might provide direct evidence on and more insight into the association between BMD with β -cell function. Finally, we did not consider other confounders, including vitamin D, adiponectin, leptin, and body composition, which should be involved in future research.

In conclusion, our study found an inverse relationship between BMD and pancreatic β -cell function (HOMA- β) and a positive relationship between BMD and fasting glucose in both elderly men and postmenopausal women. These results suggest that bone mass may be a predictor of glucose metabolism. A longitudinal study is needed to confirm our findings and explore the exact mechanisms in the future.

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Disclosure Summary: The authors have nothing to disclose.

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