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Association between percent body fat and the risk of prediabetes among Chinese adults: a 5-years longitudinal cohort study

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This study aimed to explore the influence of percent body fat (PBF) on the risk of developing prediabetes among Chinese individuals, given the limited evidence on this relationship. We conducted a retrospective cohort study involving 185,586 Chinese adults. We applied Cox proportional hazards regression models, cubic spline functions, and smooth curve fitting to analyze the relationship between initial PBF and the likelihood of prediabetes, focusing on its nonlinear connection. We conducted various sensitivity and subgroup analyses to strengthen our results. After adjusting for covariates, we found a positive correlation between PBF and the risk of prediabetes (HR = 1.13, 95% CI: 1.12–1.15, p < 0.0001). Moreover, a nonlinear correlation was identified between PBF and the likelihood of prediabetes, with a turning point at 29.5. On the left side of the turning point, the hazard ratio was 1.01 (95% CI: 0.99–1.03, p = 0.4128), while on the right side, it was 1.52 (95% CI: 1.45–1.59, p < 0.0001). Furthermore, sensitivity and subgroup analyses reaffirmed the robustness of these findings. Our research identified a nonlinear relationship between PBF and the development of prediabetes in the Chinese population, marked by a turning point at 29.5. Lowering PBF below 29.5 may reduce the risk of developing prediabetes.

Keywords Risk of prediabetes, Percent body fat, Chinese adults, Cohort study

Background

Prediabetes is a condition characterized by blood glucose levels that are elevated above normal but not high enough for a diabetes diagnosis. It is defined by impaired fasting glucose, impaired glucose tolerance, or a combination of both[1–3]. In 2017, an estimated 374 million adults worldwide were found to have prediabetes, accounting for 7.7% of the global population[4]. By 2045, this number is projected to rise to 548 million, representing 8.4% of the global adult population[5]. In China, the prevalence of prediabetes among adults is estimated to be 35.7%, indicating that approximately 388 million people in the country are affected, a rate that exceeds WHO standards[6]. If not properly managed, about 5–10% of individuals with prediabetes may develop diabetes within the next 10 years and may also experience complications associated with diabetes[7].

Compared to individuals with normal blood glucose levels, those with prediabetes face an increased risk of cardiovascular diseases, even if the onset of diabetes is prevented or delayed. Additionally, prediabetes can lead to dyslipidemia and heighten the likelihood of metabolic syndrome, which includes hypertension, abdominal obesity, and hyperlipidemia[8]. A meta-analysis of 53 studies involving 1.6 million individuals and a median follow-up of 9.5 years found that prediabetes is linked to a higher risk of cardiovascular disease and increased mortality from all causes[9]. A cohort study has indicated that prediabetes is significantly associated with an increased all-cause mortality rate, and compared to participants with normal blood glucose, individuals with prediabetes have an average life expectancy reduced by 0.7 years[10]. Prior research has demonstrated that lifestyle

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modifications can halt the progression of prediabetes to diabetes and improve the chances of returning to healthy blood glucose levels[11]. Despite this, many individuals, particularly the youth, often overlook this metabolic issue and underestimate its significance[12]. Therefore, it is essential to screen for prediabetes risk factors and promptly identify high-risk patients for intervention.

Percent body fat (PBF) is influenced by factors such as height, weight, age, and gender, serving as a crucial measure for evaluating body composition[13]. Compared to weight or BMI alone, PBF provides a more accurate representation of an individual's overall health[14]. It is closely associated with the likelihood of developing conditions like cardiovascular diseases, making it a significant factor in their prevention and management[15–17]. Initial findings from past epidemiological research indicate a strong link between elevated PBF and the risk of diabetes[18]. However, there is a lack of studies exploring the relationship between PBF and the potential development of prediabetes.

Thus, we propose that there may be a direct connection between PBF and the risk of developing prediabetes in the future, and this relationship could be non-linear. To test this hypothesis, we conducted a retrospective cohort study using previously published data from the Chinese population[19].

Methods

Study design

The research utilized a retrospective cohort study format, gathering information from computer databases in China and the NAGALA database created by Chinese researchers (Chen et al.)[19]. The primary independent variable was the PBF at baseline. The future result was the development of prediabetes.

Data source

The DATADRYAD database (http://www.datadryad.org) provided all the initial data. The dataset was sourced from a publicly accessible study published in 2018 titled "Association of body mass index and age with diabetes onset in Chinese adults: a population-based cohort study," which can be found at https://doi.org/10.1136/bmjopen-2018-021768. For those interested, the dataset can be retrieved from the following link: https://doi.org/10.5061/dryad.ft8750v[19]. The data were extracted from a computerized database established by the Rich Healthcare Group in China, which included all medical records for participants who received a health check from 2010 to 2016. The Rich Healthcare Group Review Board approved the original study. Dryad's terms of use permit additional researchers to perform secondary analyses on the data while respecting the authors' rights.

Study population

Figure 1displays flowchart of study participants. The original group of Chinese participants consisted of 685,277 individuals, with 473,744 being removed from the study, resulting in 211,833 individuals for further analysis. The study excluded 26,247 participants with FPG levels ≥ 5.6 mmol/L according to the 2021 American Diabetes Association standards[19,20]. Ultimately, 185,586 participants were included in this study. The research followed the principles outlined in the Declaration of Helsinki, and all procedures were carried out in compliance with applicable standards and rules, as outlined in the declaration. Since this was a secondary analysis conducted retrospectively, there was no need for institutional ethical review or informed consent for the study.

Data collection

Information for this research was gathered from adult individuals in China, including basic details like age and sex, body mass index (BMI), blood pressure readings (systolic and diastolic), fasting glucose levels, triglycerides, overall cholesterol, ALT levels, serum creatinine, BUN levels, and length of follow-up. Trained researchers also gathered initial information on alcohol intake, smoking habits (1 for current, 2 for past, 3 for never, and 4 for uncertain), and family diabetes history using standard surveys. The calculation of BMI involved dividing the weight in kilograms by the square of the height in meters. Blood pressure was measured using a standard mercury sphygmomanometer. Venous blood samples were obtained following a minimum of 10 h of fasting at every appointment. Covariates were selected based on clinical experience and published literature. The covariates included the following variables: continuous variables such as age, BMI, SBP, DBP, FBG, TG, TC, ALT, BUN, Scr; and categorical variables including gender, drinking status, smoking status, and family history of diabetes.

Definition

Prediabetes was characterized by elevated fasting glucose levels falling within the range of 5.6–6.9 mmol/l as per the definition (FPG 5.6–6.9 mmol/l)[21,22]. The intricate procedure for determining the PBF was explained as $PBF = 1.2 \times BMI + 0.23 \times age - 10.8 \times gender - 5.4$, where the gender was replaced by 1 for men[23].

Outcome measures

The occurrence of prediabetes was our outcome variable. Prediabetes was determined by the FPG level at the follow-up assessment, and the lack of self-reported new cases of diabetes during the follow-up period. Prediabetes was indicated by a FPG level ranging from 5.6 to 6.9 mmol/l.

Missing data processing

Missing data is inevitable in observational studies. In this research, missing data percentages were as follows: SBP at 0.00% (16 cases each), DBP at 0.00% (17 cases), TC at 2.28% (4,238 case), TG at 2.30% (4,269 case), Scr at 5.3% (9,824 case), BNU at 10.07% (18,680 case), ALT 0.83% (1,545 cases).In order to tackle this problem, we employed multiple imputation by conducting linear regression using factors such as DBP, SBP, ALT, HDL-c, LDL-c, TG, Scr, BUN, and TC. This process assumed that the missing data were Missing at Random (MAR)[24].

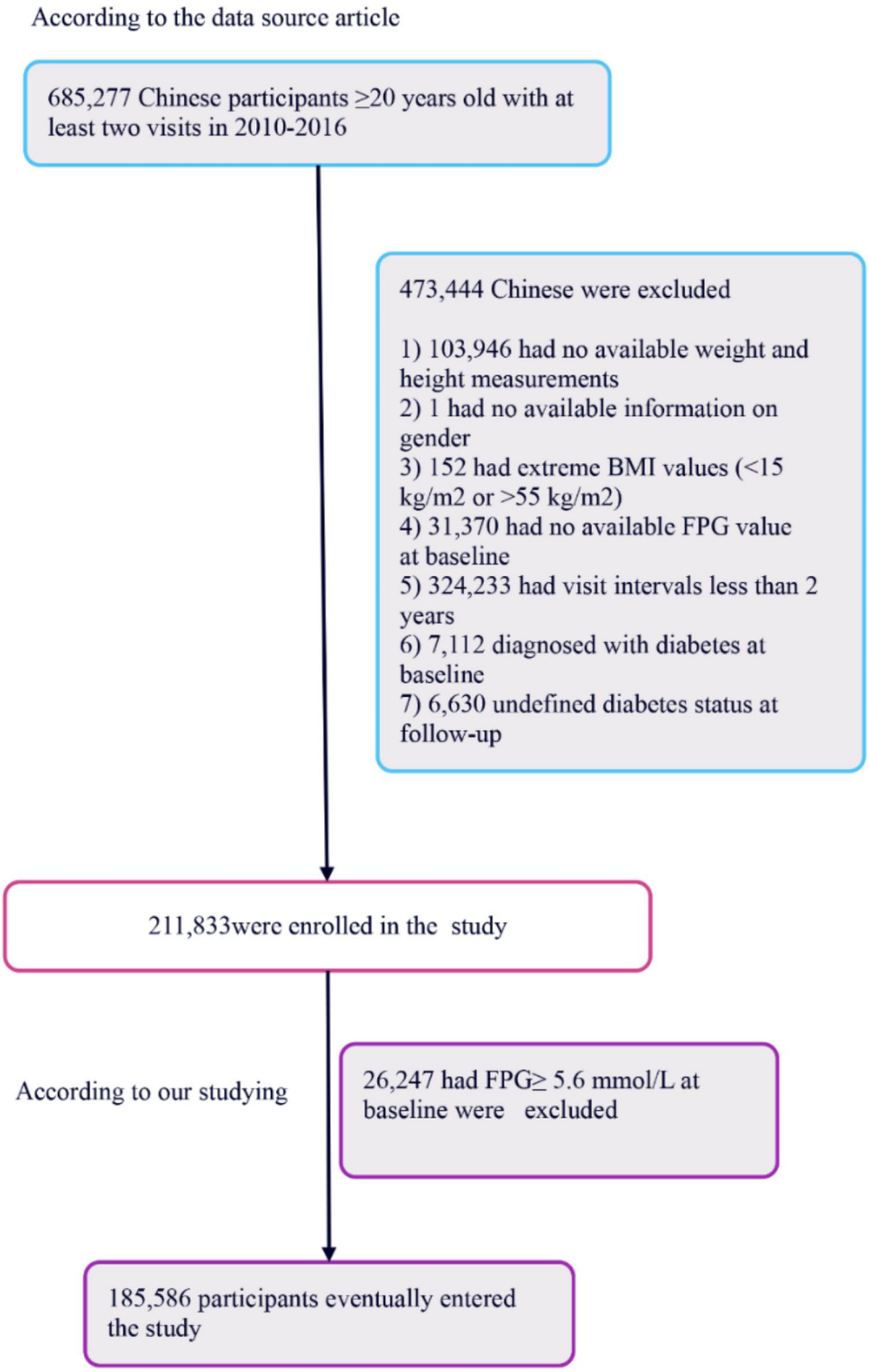

Fig. 1. Flowchart of study participants.

Statistical analysis

Participants were stratified into quartiles based on their PBF. Continuous variables were summarized as means and standard deviations for normally distributed data, and as medians with interquartile ranges for skewed distributions. Categorical variables were reported as frequencies and percentages. We used χ^2 tests to analyze categorical variables, and either one-way ANOVA (for normally distributed data) or the Kruskal-Wallis H test (for skewed data) to compare differences among PBF groups. Survival probabilities and event timing were

determined using the Kaplan-Meier technique, with comparisons of diabetes-free survival between PBF groups performed using the log-rank test.

To investigate the relationship between PBF and the risk of prediabetes, we performed univariate and multivariate Cox proportional hazards regression analyses. The analysis models included an unadjusted model, a minimally adjusted model (Model I, which adjusted for systolic blood pressure and diastolic blood pressure), and a fully adjusted model (Model II, which adjusted for systolic blood pressure, diastolic blood pressure, alanine aminotransferase, total cholesterol, triglycerides, blood urea nitrogen, creatinine, smoking and drinking status, family history of diabetes, and fasting plasma glucose at baseline). The hazard ratio (HR) and its corresponding 95% confidence interval (CI) were computed. To assess the robustness of our results, we conducted multiple sensitivity analyses.

During the follow-up period, the onset of diabetes may obscure the condition of prediabetes or influence the likelihood of its occurrence. Therefore, we performed a competing risks multivariate Cox proportional hazards regression analysis to validate the association between PBF and prediabetes. This method treats the risk of diabetes as a competing event to prediabetes. Initially, participants aged 60 years or older were excluded. Additional sensitivity analyses were conducted by excluding individuals with systolic blood pressure greater than or equal to 140 mmHg or diastolic blood pressure greater than or equal to 90 mmHg.

A Cox proportional hazards regression model with cubic splines and smoothing curve fits was employed to examine the nonlinear association between PBF and the risk of developing prediabetes. Additionally, a segmented Cox proportional hazards regression model was utilized to further elucidate the nonlinear relationship between PBF and the likelihood of prediabetes. The log-likelihood ratio test was performed to identify the model that best explains the connection between PBF and the risk in prediabetes patients.

Various subgroup analyses were conducted using a stratified Cox proportional hazards regression model, stratifying by gender, age, BMI, SBP, and DBP. Age, BMI, DBP, and SBP were categorized based on clinical thresholds: Age categories were <30, 30–40, 40–50, 50–60, 60–70, 70–80 and ≥ 80 years; BMI categories were <25 and ≥ 25; SBP categories were <90 and ≥ 90 mmHg; and additional SBP categories were <140 and ≥ 140 mmHg. Furthermore, apart from the stratification variables, we accounted for various other factors (including SBP, DBP, alanine aminotransferase, total cholesterol, triglycerides, blood urea nitrogen, serum creatinine, smoking habits, alcohol consumption, family history of diabetes, and baseline fasting plasma glucose) within each subgroup. The model did not adjust for the categorical stratification variable in each instance.

The R statistical software package (http://www.r-project.org, R Foundation) and Empower Stats (X&Y Solutions, Inc., Boston, MA, http://www.empowerstats.com) were utilized for all analyses. Statistical significance was determined by a two-tailed P-value below 0.05.

Results

Characteristics of participants

Table 1 displays the demographic and clinical features of the individuals involved in the study. The average age was 41.11 ± 12.15 years, with 98,699 participants (53.18%) being male. The average duration of follow-up was 3.15 years, during which 20,827 participants (11.22%) were diagnosed with prediabetes. The distribution of the PBF ranged from 0.47 to 58.46, with a mean of 22.10 (see Fig. 2). As the PBF quartiles rose, there was a notable rise in age, BMI, SBP, DBP, FBG, TC and ALT levels, along with an increase in the percentage of females and a decrease in Scr and BUN levels, as well as the percentage of males (all p-values <0.001).As PBF quartile increased, variations were noted in smoking habits, alcohol consumption, and family history of diabetes (all p-values <0.001).(Table 1).

Continuous variables were summarized as mean (SD) or medians (quartile interval); categorical variables were displayed as percentage (%). Abbreviations: PBF, percent body fat; BMI, body mass index; SBP, systolic blood pressure; DBP; diastolic blood pressure; TC, total cholesterol; TG triglyceride; HDL-c, high-density lipoprotein cholesterol; ALT, alanine aminotransferase; BUN, blood urea nitrogen; Scr, serum creatinine; FBG, fasting plasma glucose; DM, diabetes mellitus.

Investigating the relationship between PBF quartiles and prediabetes incidence

Table 2 examined prediabetes incidence across PBF quartiles, showing a trend of increasing incidence with higher quartiles ($P<0.001$). Overall, the incidence was 11.22% (95% CI: 11.08–11.37), or 356.19 events/10,000 person-years. Incidence in Q1 was 8.70% (95% CI: 8.45–8.96), or 277.96 events/10,000 person-years, increasing in Q2 to 15.99% (95% CI: 15.65–16.32), or 504.42 events/10,000 person-years, in Q3 to 7.46% (95% CI: 7.22–7.70), or 237.58 events/10,000 person-years, and in Q4 to 12.73% (95% CI: 12.43–13.03), or 405.41 events/10,000 person-years.

Figure 3 depicts prediabetes incidence stratified by age and gender, showing higher rates in males across all age groups. Moreover, prevalence increases with age in both genders. Kaplan-Meier curves in Fig. 4 demonstrate prediabetes probability across PBF quartiles, revealing significant differences in transition rates (log-rank test, $p<0.001$). In the higher PBF quartiles (Q2 and Q4), Chinese individuals have an elevated risk of prediabetes.

Univariate Cox proportional hazards regression was used to analyze the factors that impact the risk of developing prediabetes

Univariate analysis revealed significant positive correlations between prediabetes risk and various factors including age, BMI, baseline SBP, DBP, FPG, ALT, TG, PBF, TC, Scr, and BNU (all $P<0.05$). Additionally, smoking and alcohol consumption also influenced prediabetes risk (all $P<0.05$; see (Supplementary Table 1 for details).

PBF (quartile)	Q1 (7.67–21.41)	Q2 (21.41–25.73)	Q3 (25.73–30.02)	Q4 (30.02–58.46)	*P*-value
participants	46,360	46,425	46,356	46,445	
Age (years)	34.20 ± 6.90	44.91 ± 11.92	37.50 ± 11.73	47.80 ± 11.98	< 0.001
BMI (kg/m2)	21.74 ± 2.07	25.15 ± 2.80	21.29 ± 3.62	23.86 ± 2.81	< 0.001
SBP (mmHg)	118.11 ± 13.01	123.10 ± 15.34	112.01 ± 15.69	118.42 ± 17.16	< 0.001
DBP (mmHg)	72.96 ± 9.09	77.82 ± 10.65	70.24 ± 10.55	73.29 ± 10.73	< 0.001
FBG (mg/dL)	4.74 ± 0.49	4.83 ± 0.48	4.69 ± 0.50	4.81 ± 0.47	< 0.001
TC (mmol/L)	4.47 ± 0.83	4.85 ± 0.88	4.50 ± 0.82	4.86 ± 0.93	< 0.001
TG (mmol/L)	1.19 ± 0.74	1.73 ± 1.21	0.98 ± 0.78	1.21 ± 0.79	< 0.001
ALT (U/L)	24.42 ± 21.00	32.25 ± 25.96	17.47 ± 17.53	19.10 ± 18.75	< 0.001
BUN (mmol/L)	4.79 ± 1.12	4.91 ± 1.17	4.28 ± 1.14	4.47 ± 1.15	< 0.001
Scr (μmol/L)	79.39 ± 10.99	79.25 ± 13.44	61.13 ± 13.95	58.71 ± 10.73	< 0.001
Gender, n (%)					< 0.001
Female	0 (0.00%)	1796 (3.87%)	38,970 (84.07%)	46,121 (99.30%)	
Male	46,360 (100.00%)	44,629 (96.13%)	7386 (15.93%)	324 (0.70%)	
Smoking status, n (%)					< 0.001
Current smoker	3664 (7.90%)	5205 (11.21%)	908 (1.96%)	37 (0.08%)	
Ever smoker	1065 (2.30%)	916 (1.97%)	141 (0.30%)	19 (0.04%)	
Never	11,253 (24.27%)	8505 (18.32%)	10,328 (22.28%)	9344 (20.12%)	
Unknown	30,378 (65.53%)	31,799 (68.50%)	34,979 (75.46%)	37,045 (79.76%)	
Drinking status, n (%)					< 0.001
Current drinker	275 (0.59%)	573 (1.23%)	124 (0.27%)	20 (0.04%)	
Ever drinker	3268 (7.05%)	3235 (6.97%)	601 (1.30%)	311 (0.67%)	
Never	12,439 (26.83%)	10,818 (23.30%)	10,652 (22.98%)	9069 (19.53%)	
Unknown	30,378 (65.53%)	31,799 (68.50%)	34,979 (75.46%)	37,045 (79.76%)	
Family history of diabetes, n (%)			< 0.001		< 0.001
No	45,800 (98.79%)	45,700 (98.44%)	45,362 (97.86%)	45,022 (96.94%)	
Yes	560 (1.21%)	725 (1.56%)	994 (2.14%)	1423 (3.06%)	
Follow-up (year)	3.13 ± 0.93	3.17 ± 0.95	3.14 ± 0.94	3.14 ± 0.94	< 0.001

Table 1. The baseline characteristics of participants.

A multivariate Cox proportional hazards regression model was utilized to examine the association between the PBF and the likelihood of developing prediabetes

Three Cox proportional hazards regression models were constructed to assess the association between PBF and prediabetes risk. The initial model revealed a 11% increased risk of prediabetes per 10-unit rise in PBF (HR 1.11, 95% CI 1.10–1.13, $P < 0.0001$). A minimally adjusted model including only SBP and DBP showed a 9% higher risk (HR 1.09, 95% CI 1.08–1.11, $P < 0.0001$). Lastly, a fully adjusted model indicated a 13% higher risk (HR 1.13, 95% CI 1.12–1.15, $P < 0.0001$) per 10-unit increase in PBF. Confidence intervals support the robustness of the PBF-prediabetes risk association (see Table 3).

Sensitivity analysis

To validate our findings, multiple sensitivity analyses were conducted. Initially, individuals aged ≥ 60 years (18,341 participants) were excluded. After adjusting for confounders, results consistently indicated a positive correlation between PBF and prediabetes risk (HR = 1.12, 95% CI: 1.10–1.14, $P < 0.0001$). Furthermore, sensitivity analyses excluding participants with systolic blood pressure ≥ 140 mmHg ($N = 15,822$) or diastolic blood pressure ≥ 90 mmHg ($N = 13,247$) confirmed a strong association between PBF and prediabetes risk (HR = 1.13, 95% CI 1.11–1.15 and HR = 1.14, 95% CI 1.12–1.15, respectively, both $P < 0.0001$). These comprehensive analyses support the reliability of our findings (Table 4). Through an in-depth analysis of the original data, we reached the same conclusion (Supplementary Table S2). In addition, considering the risk of diabetes as a competing event for prediabetes risk, the results of the competing risk analysis are presented in Supplementary Table S3. In Model I, PBF was positively correlated with the risk of prediabetes (SHR = 1.12, 95% CI 1.09–1.15). In Model II, after adjusting for SBP and DBP, the SHR was 1.21 (95% CI 1.21–1.21). In the fully adjusted Model III, which accounted for confounders such as SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, drinking status, family history of diabetes, and baseline FPG, the association between PBF and prediabetes risk remained positive (SHR = 1.13, 95% CI 1.11–1.14).

Using the Cox proportional hazards regression model with cubic splines to adjust for non-linear relationships

Using the Cox proportional hazards regression model with cubic splines, we identified a non-linear relationship between PBF and prediabetes risk (Fig. 5; Table 5). Initially, a Cox proportional hazards regression model with cubic splines assessed this relationship, revealing a non-linear association with future prediabetes risk.

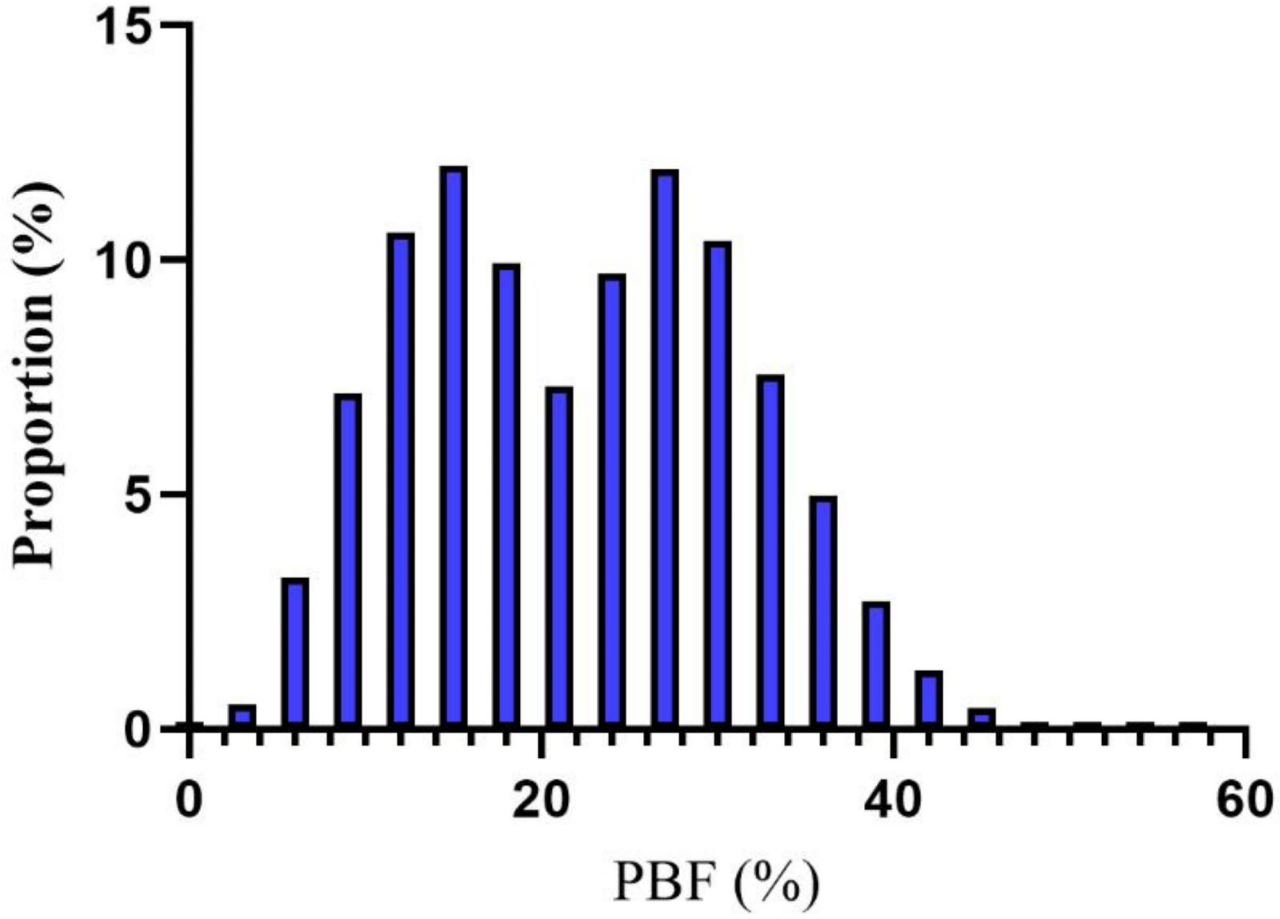

Fig. 2. Distribution of PBF (%). It presented a normal distribution, ranging from 0.47 to 58.46, with a mean of 22.10.

PBF quartile	Participants (*n*)	prediabetes events (*n*)	Incidence rate (95% CI) (%)	Per 10,000 person-year
Total	185,586	20,827	11.22 (11.08–11.37)	356.19
Q1	46,360	4,035	8.70(8.45–8.96)	277.96
Q2	46,425	7,422	15.99 (15.65–16.32)	504.42
Q3	46,356	3,458	7.46 (7.22–7.70)	237.58
Q4	46,445	5,912	12.73 (12.43–13.03)	405.41
P for trend				< 0.001

Table 2. The incidence rate of diabetes (% or per 10000 person-year).

Subsequently, a segmented Cox model was used to explore PBF's impact on prediabetes likelihood, showing an HR of 1.13 (95% CI 1.12–1.15, $P < 0.0001$). Additionally, a threshold effect was observed at 29.5 units of PBF, where the HR increased to 1.01 (95% CI 0.99–1.03, $P = 0.4128$) below this level and to 1.52 (95% CI 1.45–1.59, $P < 0.0001$) at or above this threshold.

Subgroup analysis

Figure 6 delineates the stratified associations between PBF and prediabetes risk across various subgroups. PBF is significantly associated with prediabetes risk, particularly in older adults. The age group 50–60 years shows an HR of 1.07 (95% CI: 1.03–1.11, $p = 0.0002$), increasing to 1.09 (95% CI: 1.05–1.14, $p < 0.0001$) for those aged 60–70 years. Participants aged ≥ 80 years have an HR of 1.15 (95% CI: 1.02–1.30, $p = 0.0200$). Stratification by BMI reveals that individuals with a BMI ≥ 25 kg/m² have an HR of 1.17 (95% CI: 1.14–1.20, $p < 0.0001$), compared to 1.08 (95% CI: 1.06–1.10, $p < 0.0001$) for those with a BMI < 25 kg/m². Women exhibit a higher risk, with an HR of 2.07 (95% CI: 1.99–2.16, $p < 0.0001$) versus 1.50 (95% CI: 1.44–1.55, $p < 0.0001$) for men. No significant differences were observed in other subgroups.

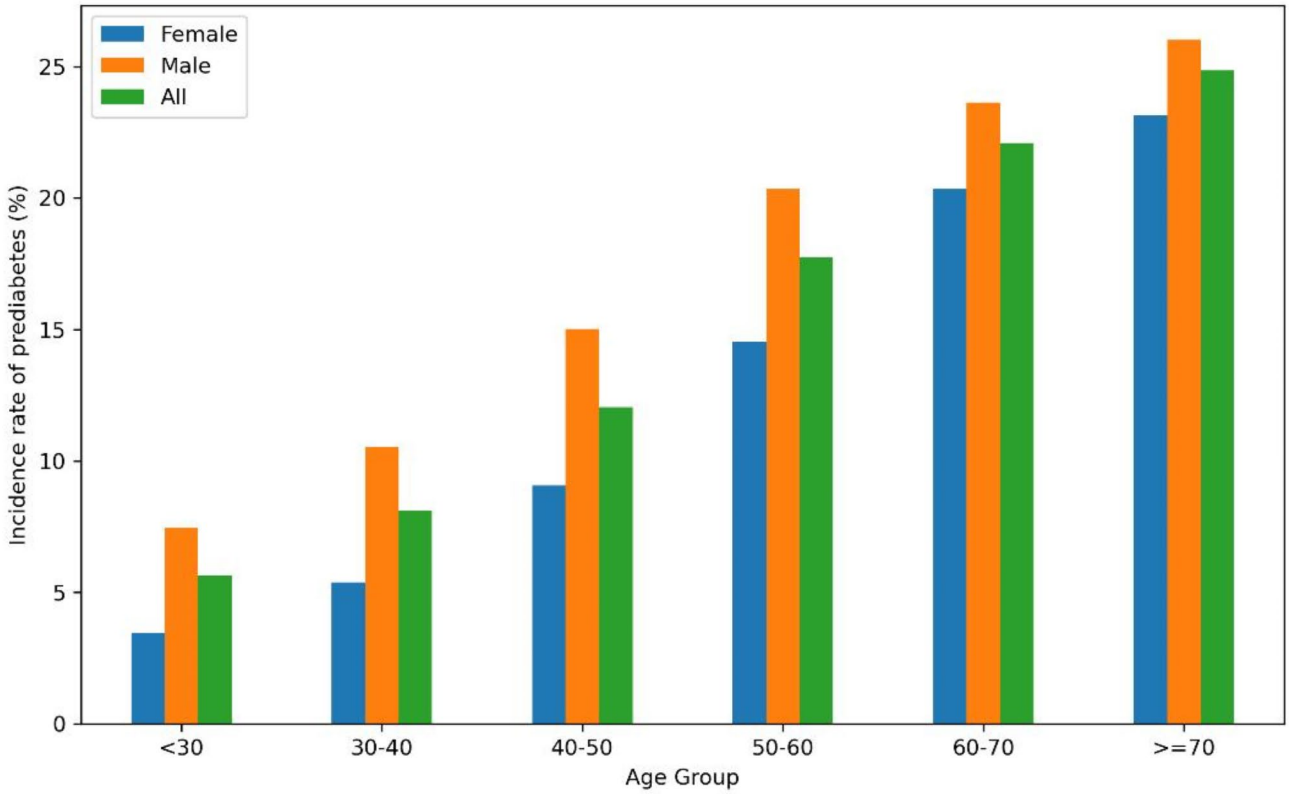

Fig. 3. The incidence of prediabetes by age stratification and gender. It shows that the incidence of prediabetes in participants was higher in male than in female, regardless of age group. It was also found that the incidence of prediabetes increased with age in both female and male.

Discussion

In our study, we investigated the association between PBF and prediabetes risk among Chinese individuals. Following a long-term follow-up of 185,586 participants, we identified a significant increase in prediabetes risk with higher PBF. Additionally, we found a non-linear relationship between PBF and incident prediabetes, particularly examining the impact of PBF around a critical threshold. Above 29.5 units of PBF, each 10-unit increase raised prediabetes risk by 52% (HR 1.52, 95% CI 1.45–1.59, $P<0.001$). Conversely, below 29.5, the increase in prediabetes risk was less pronounced (HR 1.01, 95% CI 0.99–1.03, $P=0.4128$). This suggests that maintaining PBF below 29.5 could potentially mitigate prediabetes risk in adults. Interaction analysis revealed a stronger association between PBF and prediabetes risk in female participants.

In a recent study, 12.8% of 799 initially normoglycemic individuals from the Baependi community in Brazil developed prediabetes over 5 years[25]. In our study of Chinese health check-up participants, the prediabetes incidence rate was 11.26% over an average follow-up of 3.14 years. The similar follow-up durations and age distributions in both study populations suggest that our lower prediabetes incidence rate might result from using fasting plasma glucose impairment as the sole criterion for defining prediabetes.

Recent studies have minimally explored the relationship between PBF and diabetes risk. A cross-sectional study involving 4,828 white adults aged 18–80 years from Spain suggests that PBF might more accurately predict diabetes or prediabetes risk compared to BMI[26]. This finding has significant implications for improving the prevention and treatment of obesity-related diseases. In China, a study with 5,595 participants aged 18–65 found that meeting specific PBF thresholds significantly increased the risk of type 2 diabetes in both men and women[27]. Another study in Shanghai with 1,857 individuals showed that transitioning from normal PBF to obesity raised diabetes risk[28]. A South Korean study with 5,972 participants suggested that higher PBF was linked to a greater risk of diabetes, especially when combined with other risk factors[17]. Addressing prediabetes is crucial to preventing or delaying the onset of diabetes.

However, research on the association between PBF and prediabetes remains limited. A cross-sectional study conducted in the United States involving 11,125 non-pregnant participants found a close correlation between PBF and prediabetes risk after adjusting for age, race/ethnicity, and height[29]. To further explore the relationship between PBF and prediabetes, we included 185,586 Chinese individuals in a 5-year follow-up study, revealing a significant positive correlation between PBF and prediabetes risk. This corroborates our initial hypothesis. However, our findings differ significantly from previous studies in several key aspects: (1) Different study populations—while they focused on Americans, our study centered on the Chinese population with a much larger sample size. (2) Various research methodologies and statistical techniques were employed; some studies utilized logistic regression, while our team used Cox proportional hazards models. (3) In contrast to our study,

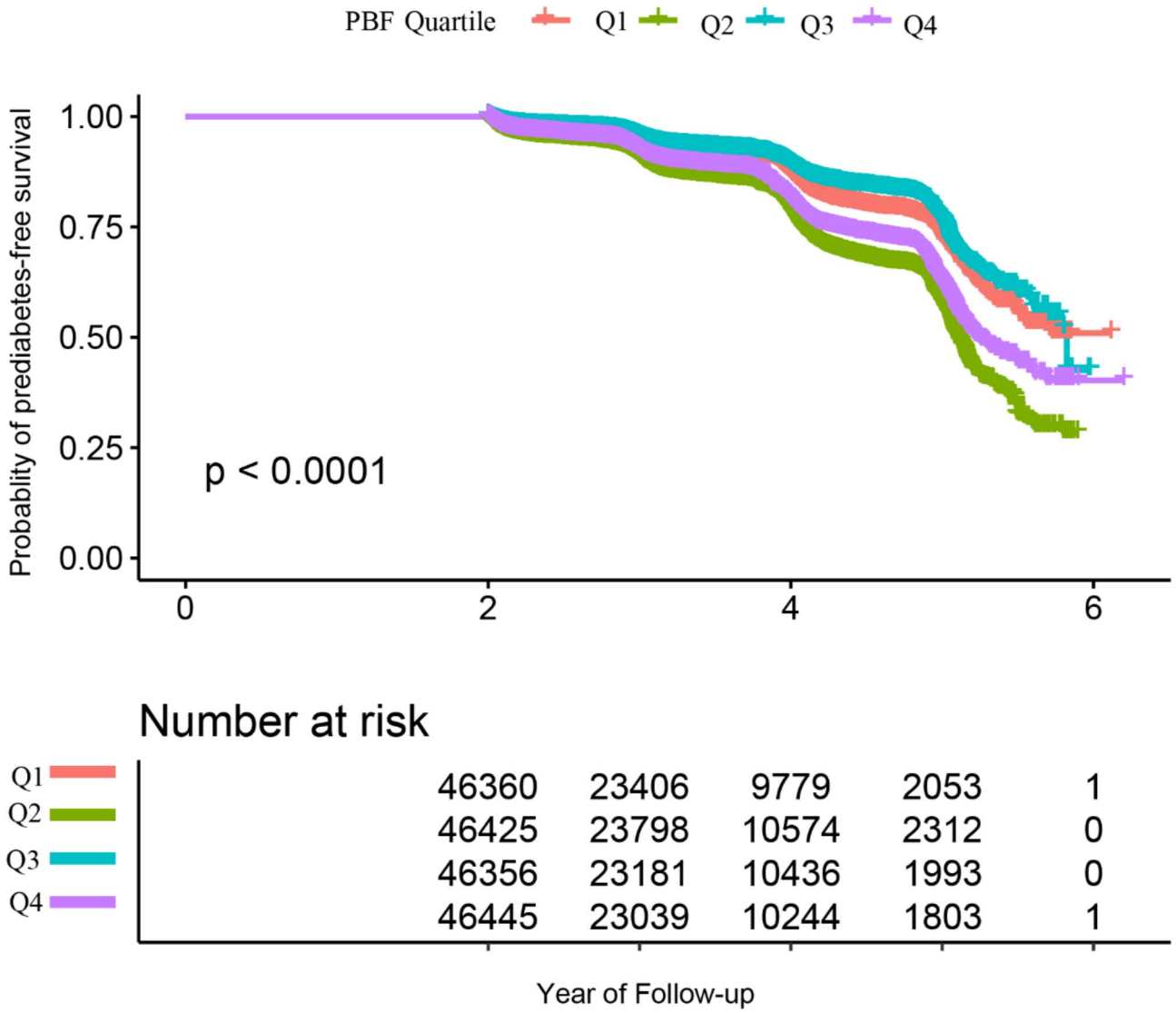

Fig. 4. Kaplan–Meier curves for the probability of prediabetes in Chinese adults. The probability of prediabetes increased progressively with rising PBF, meaning that individual with the highest PBF had the higher probability of prediabetes in Chinese individuals.

Exposure	Crude model (HR,95%CI) *P*	Model I(HR,95%CI) *P*	Model II(HR,95%CI) *P*
PBF (per 10%)	1.11 (1.10, 1.13) < 0.0001	1.09 (1.08, 1.11) < 0.0001	1.13 (1.12, 1.15) < 0.0001
(PBF quartiles)			
Q1	Ref	Ref	Ref
Q2	1.75 (1.69, 1.82) < 0.0001	1.54 (1.48, 1.60) < 0.0001	1.25 (1.20, 1.30) < 0.0001
Q3	0.84 (0.81, 0.88) < 0.0001	0.93 (0.89, 0.97) 0.0020	1.00 (0.96, 1.05) 0.9468
Q4	1.45 (1.40, 1.51) < 0.0001	1.40 (1.34, 1.46) < 0.0001	1.40 (1.35, 1.46) < 0.0001
P for trend	< 0.0001	< 0.0001	< 0.0001

Table 3. Relationship between PBF and risk of prediabetes in different models. Crude model: we did not adjust other covariates. Model I: we adjusted SBP, DBP. Model II: we adjusted SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline.

they failed to investigate the non-linear correlation between PBF and prediabetes. (4) They did not account for confounding variables such as SBP, DBP, ALT, TC, TG, BUN, Cr, smoking habits, alcohol consumption, family history of diabetes, and baseline FPG, unlike our study. (5) Their study did not identify a nonlinear cutoff point for the relationship between PBF and prediabetes, which our study did. These studies highlight that

Exposure	Model I (HR,95%CI) *P*	Model II (HR,95%CI) *P*	Model III (HR,95%CI) *P*
PBF (per 10%)	1.12 (1.10, 1.14) < 0.0001	1.13 (1.11, 1.15) < 0.0001	1.14 (1.12, 1.15) < 0.0001
(PBF quartiles)			
Q1	Ref	Ref	Ref
Q2	1.23 (1.18, 1.28) < 0.0001	1.27 (1.22, 1.32) < 0.0001	1.26 (1.21, 1.32) < 0.0001
Q3	0.91 (0.87, 0.96) 0.0002	0.95 (0.90, 1.00) 0.0438	0.97 (0.92, 1.02) 0.2220
Q4	1.37 (1.31, 1.43) < 0.0001	1.39 (1.33, 1.45) < 0.0001	1.40 (1.34, 1.46) < 0.0001
P for trend	< 0.0001	< 0.0001	< 0.0001

Table 4. Relationship between PBF and risk of prediabetes in different sensitivity analyses. Crude model I was a sensitivity analysis performed after excluding participants with age ≥ 65 years old (N = 24,804). we adjusted SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline. Model II was a sensitivity analysis performed after excluding participants with SBP ≥ 140 mmHg (N = 21,403). we adjusted SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline. Model III was a sensitivity analysis performed after excluding participants with DBP ≥ 90 mmHg (N = 17,063). we adjusted SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline.

the relationship between different levels of PBF and prediabetes risk may vary, emphasizing the importance of exploring nonlinear relationships.

To our knowledge, this is the first study to uncover a non-linear relationship between PBF and prediabetes risk, further supporting our hypothesis. Employing a segmented Cox proportional hazards regression model, we were able to clarify the L-shaped relationship between PBF and prediabetes risk, while controlling for potential confounding factors. The analysis revealed that the inflection point for PBF was at 29.5%. Our findings indicated that among individuals with PBF exceeding 29.5%, a 10-unit decrease in PBF was associated with a 52% reduction in the adjusted hazard ratio for prediabetes risk (HR = 1.52, 95% CI: 1.45–1.59). However, if PBF was below 29.5%, the adjusted HR for prediabetes risk does not show a significant change with increasing PBF. These findings have important clinical implications. Firstly, for individuals with PBF above 29.5%, interventions aimed at weight reduction can substantially decrease the risk of prediabetes. Secondly, the critical threshold of 29.5% PBF serves as a crucial indicator for assessing prediabetes risk. Encouraging individuals with elevated PBF to engage in weight reduction strategies such as dietary changes, exercise, and other interventions may help mitigate the risk of prediabetes. Furthermore, our subgroup analysis indicates a significant association between PBF and prediabetes risk in older adults, especially in those aged 80 and above. This finding highlights the necessity of monitoring body fat in this high-risk age group. Moreover, the correlation between PBF and prediabetes risk is stronger in individuals with a BMI ≥ 25 kg/m^2, suggesting that overweight individuals have a higher susceptibility to prediabetes. The increased risk observed in women suggests the need for gender-specific strategies in managing body fat and diabetes risk.

This study exhibits several strengths. Firstly, it uncovered the first-ever non-linear relationship between PBF and the likelihood of prediabetes. Moreover, the ample sample size provides a robust basis for screening and managing prediabetes prevalence in healthy populations, thereby reducing potential biases. Sensitivity analyses and the use of a segmented Cox proportional hazards regression model further validated our findings, pinpointing the critical PBF thresholds that influence prediabetes risk. These results underscore the importance of monitoring PBF and initiating early interventions for diabetes prevention. Additionally, subgroup analyses highlighted varying impacts of PBF on prediabetes risk across different groups, affirming the reliability of our results. Nonetheless, we reveal a stronger association between PBF and prediabetes risk among females. This may be related to mechanisms of active fat metabolism in female adipocytes, which can impact insulin resistance and metabolic health, potentially heightening prediabetes risk in women with higher PBF[30–32].

However, our study has several limitations. Firstly, it only involved individuals of Chinese descent, indicating the need for further exploration of the relationship between PBF and prediabetes risk across diverse genetic backgrounds. Secondly, as with all observational research, there may still be unmeasured or uncontrolled confounding factors, even after adjusting for recognized potential confounders. Thirdly, the average follow-up duration of participants was relatively short; extending this period in future studies could help reduce potential biases or chance findings from short-term observations. Fourthly, this report constitutes a secondary analysis of existing databases. Although adjustments were made for many confounding factors, variables not included in the databases were not accounted for. Fifthly, the current diagnostic criteria for prediabetes have limitations, primarily relying on impaired fasting glucose without considering oral glucose tolerance tests, glycated hemoglobin, and multiple fasting glucose measurements. This single-index approach may result in the underdiagnosis of prediabetes. Therefore, future studies assessing prediabetes incidence should consider measuring additional variables to improve diagnostic accuracy and comprehensiveness. Sixthly, the retrospective nature of this study establishes correlations between body fat percentage and prediabetes risk without establishing a definitive cause-and-effect relationship. Seventh, we recognize that the accuracy of the BFP calculated by this formula needs validation against gold standard methods, such as DEXA. To enhance the reliability of our study, we suggest that future research conduct comparative analyses between this formula and gold standard measurements to assess its accuracy. Eighth, in our analysis, we found that the association between PBF and prediabetes risk was more pronounced in the 60–70 age group and the ≥ 80 age group. In the sensitivity analysis, excluding individuals

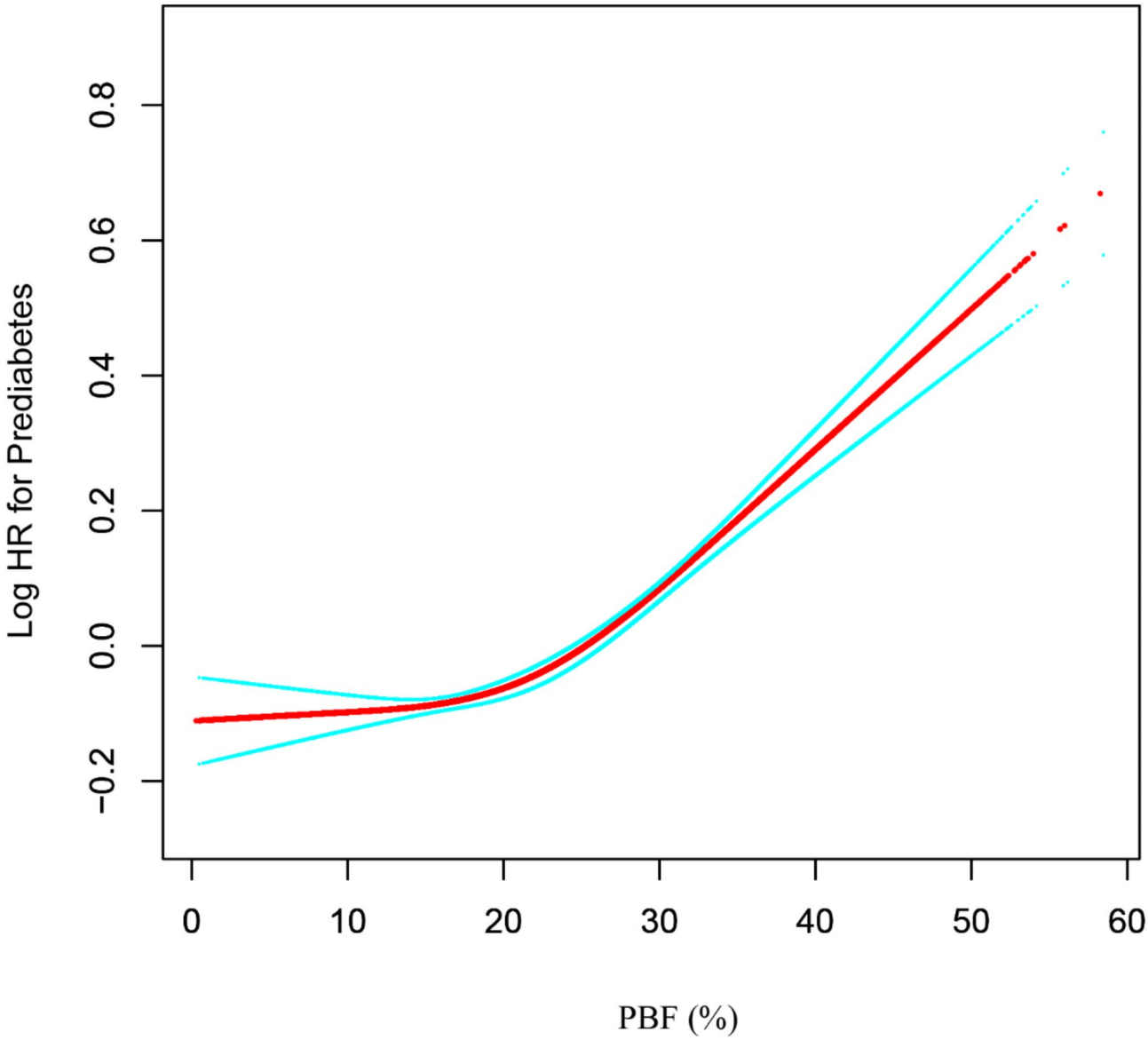

Fig. 5. The non-linear relationship between PBF and risk of diabetes. The relationship between PBF and risk of diabetes was non-linear after adjusting for SBP, DBP, ALT, TC, TG, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline.

Outcome: prediabetes	HR, 95%CI	*P* -value
Standard Cox regression PBF (per 10%)	1.13 (1.12, 1.15) < 0.0001	<0.0001
Two-piecewise Cox regression		
Inflection points of PBF (per 10%)	29.5	
<29.5	1.01 (0.99, 1.03)	0.4128
≥29.5	1.52 (1.45, 1.59)	< 0.0001
P for log-likelihood ratio test	< 0.001	

Table 5. The result of the two-piecewise linear regression model. We adjusted for SBP, DBP, ALT, TC, TG, HDL-c, LDL-c, BUN, Cr, smoking status, Drinking status, Family history of diabetes and FPG at baseline.

aged ≥ 60 years could lead to an underestimation or misinterpretation of the overall results. Lastly, body fat percentage and other parameters were only measured at baseline in our study, without considering changes over time. Future efforts should focus on collecting more comprehensive information, including data on changes in body fat percentage over time.

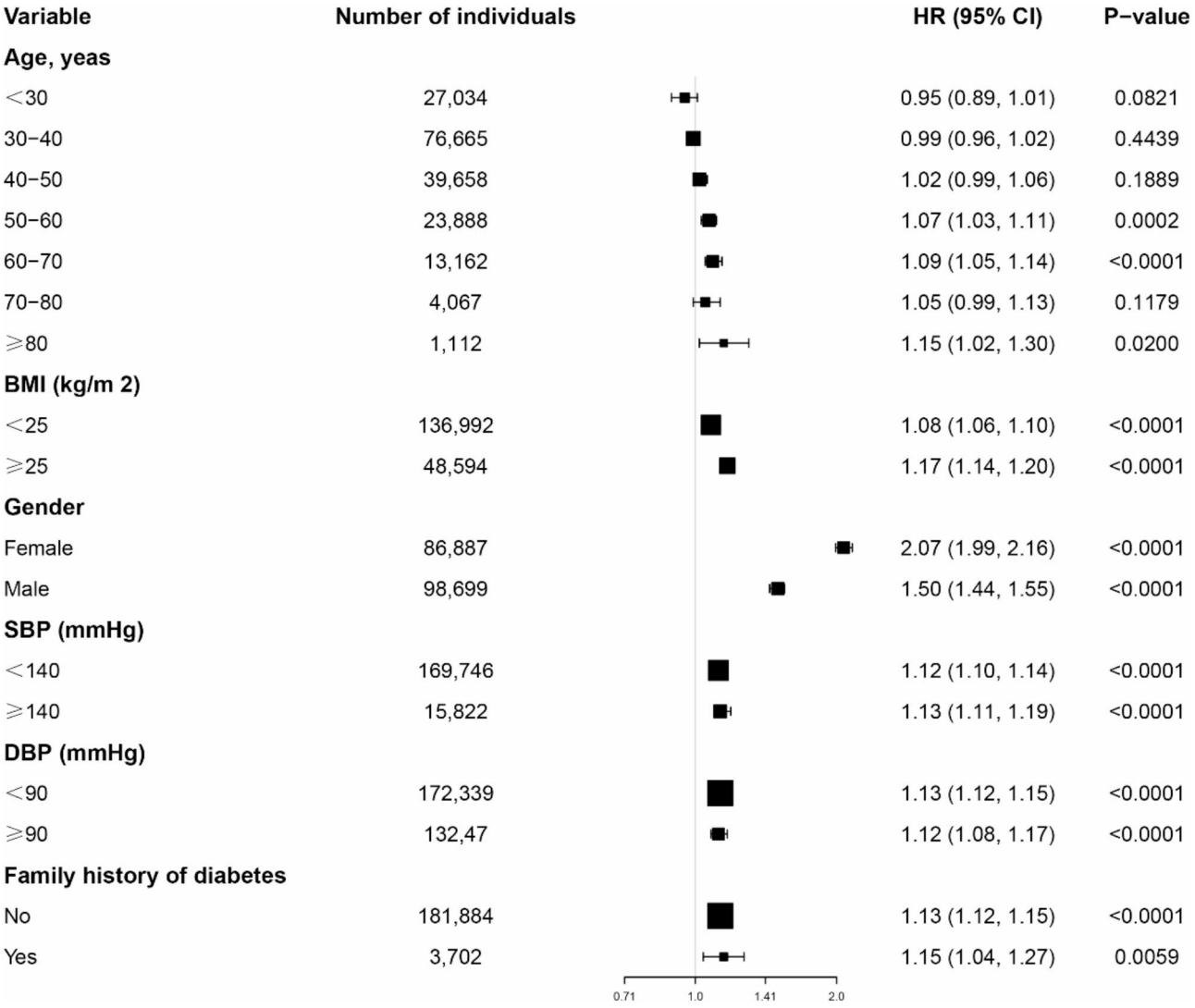

Fig. 6. Stratified associations between PBF and risk of prediabetes by age, sex, BMI, SBP, DBP, and family history of diabetes. In each case, the model is not adjusted for the stratification variable when the stratification variable was a categorical variable.

Conclusion

For the first time, we observed a positive and non-linear association between PBF and prediabetes development. Additionally, we examined the impact of PBF around its inflection point on prediabetes. When PBF exceeds 29.5%, each 10-unit increase is associated with a 52% higher risk of prediabetes in adults. However, below 29.5%, the increase in prediabetes risk is not significant with rising PBF. The critical threshold of 29.5% PBF serves as a key indicator for assessing prediabetes risk. Weight reduction interventions can significantly lower the risk of prediabetes for individuals with PBF above 29.5%.

Data availability

The DATADRYAD database (http://www.datadryad.org) provided all the initial data. The dataset was sourced from a publicly accessible study published in 2018 titled "Association of body mass index and age with diabetes onset in Chinese adults: a population-based cohort study," which can be found at https://doi.org/10.1136/bmjopen-2018-021768http://dx.doi.org/. For those interested, the dataset can be retrieved from the following link: https://doi.org/10.5061/dryad.ft8750vhttps://doi.org/10.5061/dryad.ft8750v.

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Author contributions

Xin Li and Haomiao Yu contributed to the study design and drafting of the manuscript. Xin Li, Ke Yu and Haomiao Yu were responsible for statistical analysis, research, and interpreting the data, ensuring data integrity and analysis accuracy. Zhenhuan Huang and Zhe Deng contributed to the discussion, and reviewed the manuscript. All authors have read and approved the final manuscript.

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Declarations

Competing interests
The authors declare no competing interests.

Ethical approval
The studies involving human participants were reviewed and approved by The Rich Healthcare Group Review Board. The ethics committee waived the need for written informed consent.

Additional information
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