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Lower handgrip strength levels probably precede triglyceride glucose index and associated with diabetes in men not in women

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ABSTRACT

Aims/Introduction: To explore the relationship between handgrip strength per weight (HGS/W), triglyceride glucose index (TyG) and diabetes, and whether lower HGS levels precede TyG in the Chinese elderly population.

Materials and Methods: Two linear regression models were used to explore the association of whether baseline HGS/W predicted follow-up variation of TyG or baseline TyG predicted follow-up variation of HGS/W. The logistic regression model was used to examine the relationship between baseline HGS/W and future diabetes.

Results: A total of 4,561 participants in the China Health and Retirement Longitudinal Study were enrolled, of which 47.0% were men, and the mean age was 58.7 years (standard deviation 8.68 years). A lower baseline HGS/W significantly correlated with a higher level of follow-up TyG ($\beta = -0.173$, $P = 0.002$). The baseline level of HGS/W was significantly negatively associated with the incidence risk of diabetes (rate ratio 0.375, $P = 0.004$). However, in sex stratification, the statistical association between HGS/W and TyG and diabetes was only in men.

Conclusions: Our results showed that HGS/W was inversely associated with TyG and diabetes, and lower HGS/W levels preceded TyG levels in the elderly population. However, the effect was inconsistent between men and women, and the possible mechanism would require further clarification.

INTRODUCTION

Diabetes, as one of the top causes of world death, is a chronic disease because of the resistance of insulin or the lack of enough insulin[1]. Over time, diabetes might develop into systemic diseases[2]. The number of patient with diabetes is growing globally[3]. China ranked among the top five globally in morbidity and mortality of diabetes in 2017[4], and these numbers are increasing[5]. However, more than half of diabetes (or prediabetes) patients are undiagnosed[6]. In addition, under the coronavirus disease 2019 pandemic background, it was reported that diabetes patients have a higher chance of developing severe coronavirus symptoms and have a poor prognosis[7]. However, because of coronavirus disease 2019, hospital resources are redistributed, and citizens must reduce social interactions, restricting medical testing and treatment for diabetes or prediabetes patients. Hence, it will reduce the future diabetes burden if there is a simple method for predicting diabetes, and patients or potential patients can self-test at home and report to their family doctor online.

The handgrip strength (HGS) test is a non-invasive, simple upper limb muscle function test[8]. HGS is becoming a predictor of heart disease, vascular disease, peripheral artery disease, nerve damage and malnutrition[9]. Furthermore, some studies have explored the relationship between HGS and diabetes in people of different ages and races[10,11]. Nevertheless, the relationship is still unknown among the Chinese elderly. Therefore, the present study aimed to examine the correlation between HGS and diabetes in Chinese older people.

Muscle movement depends on glucose metabolism[12]. However, insulin resistance (IR) reduces the muscle's ability to process glucose and weakens muscle strength[13,14]. In addition, IR is one of the central mechanisms of diabetes progression[15]. Most diabetes patients, especially obese patients, are resistant to

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insulin. Hence, IR testing is used in early diabetes screening[16]. Therefore another goal of our research was to explore the temporal relationship between HGS and IR. The triglyceride glucose index (TyG) can replace standard methods of IR measurement, such as the euglycemic-hyperinsulinemic clamp test and the homeostasis model assessment of insulin resistance index[17,18]. Furthermore, the more accessible and affordable price gives TyG a greater advantage in large-scale screening. Hence, the TyG index was used to identify IR in the present study.

MATERIALS AND METHODS

Study population

The China Health and Retirement Longitudinal Study (CHARLS) is carried out by the National School of Development of Peking University and is a nationally representative survey. The national baseline survey was carried out from June 2011 to March 2012. A multistage, random cluster sampling process was carried out in 450 villages/urban areas, and 10,287 households aged ≥45 years as a representative sample were finally selected. Furthermore, baseline questionnaires were used in these households every 2 years. In addition, their blood samples were also collected in 2011 and 2015. Additional information about the CHARLS can be found on the website: http://charls.pku.edu.cn/en.

The present study was a post-hoc analysis. Figure 1 describes the details of the sampling process. A total of 17,708 individuals were at baseline. We excluded individuals aged <45 years (n = 1,099) or those who had diabetes and cancer (n = 2,366) at baseline. Then, individuals were excluded if 2011 data were missing for ethnicity, education level and sex (n = 1,413), or for height, weight and HGS (2,989), or for systolic blood pressure (SBP), diastolic blood pressure (DBP) or pulse (n = 131), or in high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides (TG) or fasting plasma glucose (FPG; n = 2,815). Thus, 6,895 participants attended the follow up. Furthermore, individuals were excluded because of missing data in height, weight, HGS (n = 237), TG or FPG (n = 2079) in 2015. Finally, 4,561 participants (2,144 men and 2,417 women) were included in the present study.

Peking University's ethical review committee (IRB 00001052-11015) approved the study protocol on 20 January 2011. The procedures followed the ethical standards of the responsible committee of Peking University and the Chinese Center for Disease Control and Prevention, and written informed consent was obtained from all participants or their proxies.

Exposures and covariates

Demographics (age, sex, education and ethnicity), lifestyle factors (drinking and smoking status) and diseases (diabetes and cancer) were obtained through the structured home interview by CHARLS trained health staff. In addition, health behaviors information was collected from a self-reported health questionnaire: (i) frequency of alcohol consumption (never, once a month and more than once a month); and (ii) smoking status in the past year (never, former and current smoker).

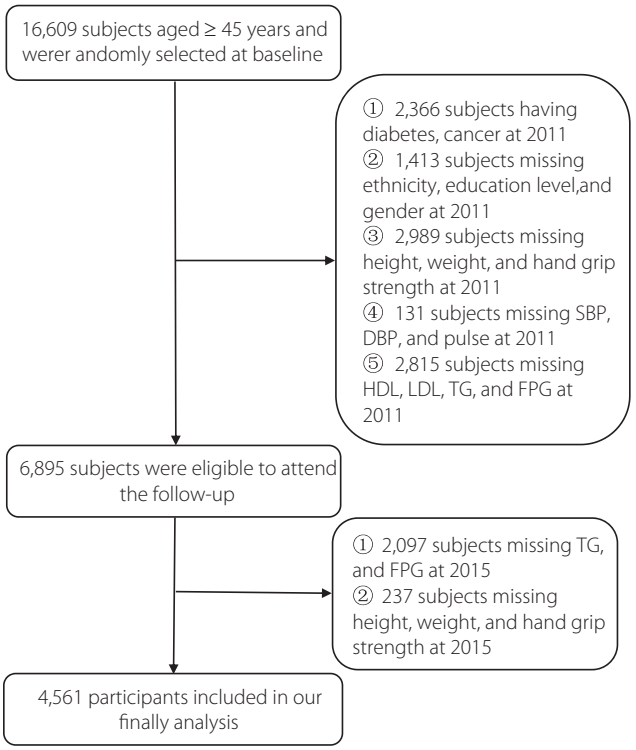

Figure 1 | Flow chart for selection of study participants. DBP, diastolic blood pressure; FPG, fasting plasma glucose; LDL, low-density lipoprotein; SBP, systolic blood pressure; TG, triglycerides.

Furthermore, CHARLS describes the blood pressure indexes (cholesterol indexes, FPG, biochemical blood indexes and others) collection and measurement methods on their website (http://charls.pku.edu.cn/en). We used an auto-analyzer (Olympus AU640 Auto-Analyzer; Olympus Corp., Kobe, Japan) to measure TG, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol, and Omron™ HEM-7200 Monitor (Omron Co., Ltd., Dalian, China) to measure SBP, DBP and pulse in the sitting position in a 5-min rest interval.

We used a mechanical handgrip meter (WL-1000 Mechanical Handgrip Meter; Nantong, China) to measure HGS. The participant held a handgrip meter suitable for their hands' size with their elbows at a 90° angle on the body sides. Then, the participant used the maximum strength to hold the handgrip meter for a few seconds and then release it. There were four measurements: first and third with the dominant hand, and second and fourth with the other hand. After each measurement, the research interviewer recorded the result and handed the meter to the participant. Participants whose hands had surgery, or had swelling, inflammation, severe pain or injury in the past 6 months were excluded. Participants whose hand had the symptoms as aforementioned only measured the other

hand. In addition, the HGS per weight (HGS/W) was calculated by HGS/W = HGS (kg) / weight (kg). We used the TyG index to replace the standard methods of IR measurement. TyG index was calculated through the following formula: TyG = ln [fasting TG (mmol/L) × FPG (mmol/L) × 0.5 × 159.37].

According to the American Diabetes Association criteria, in the present study, diabetes patients are defined as: self-reported diabetes diagnosed by a doctor during an individual's interview or the person whose FPG was ≥126 mg/dL (7.0 mmol/mol), or glycated hemoglobin ≥6.5% (48 mmol/mol) from blood test reports[19].

Body mass index (BMI) was calculated as the weight divided by the square of height (kg/m^2), and those who had BMI ≥28 were defined as obese.

Statistical analysis

Percentiles, mean (standard deviation) and median (interquartile range) describe the central and discrete trends for categorical variables, normally distributed continuous variables and non-normally distributed continuous variables, respectively. Simultaneously, *t*-test, Mann–Whitney *U*-test or Pearson's χ^2-tests were used to compare the statistical significance between men and women.

Three regression models were built to examine associations between baseline HGS/W, future TyG and diabetes (x = HGS/W in 2011 and y = diabetes cases and y = TyG variation in 2015, respectively), and between baseline TyG, future HGS/W and diabetes (x = TyG in 2011 and y = diabetes cases and y = HGS/W variation in 2015, respectively). Baseline HGS/W and TyG were, respectively, adjusted in model 1. In addition, model 2 was adjusted in baseline HGS/W, baseline TyG, sex, ethnicity and age. Based on model 2, model 3 added the level of education in 2011, current smoking, alcohol drinking, SBP, DBP, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol variables. Then, the three models were constructed in groups of men, women, obese participants and non-obese participants separately. In addition, the variance expansion factor was examined for the multicollinearity among independent variables. The variance expansion factor >10 means variables significant in multicollinearity. Statistical significance was accepted when $P \leq 0.05$. All statistical analyses were carried out by IBM SPSS version 25.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Of the 4,561 participants (mean age 58.7 years, standard deviation 8.68 years), 2,144 were men, and 2,417 were women. Population characteristics of baseline and follow up are shown in Table 1. baseline SBP, DBP, HR and FPG, and future diabetes showed no significant sex differences.

Table 2 showed the prospective association of baseline HGS/W with follow-up TyG and diabetes. From the result of model 3, we found that the level of HGS/W was inversely associated with the level of follow-up TyG (β = –0.173, $P = 0.002$). At the same time, we found that a higher level of HGS/W was related to a lower incidence risk of diabetes (rate ratio [RR] 0.375, $P = 0.004$).

Table 3 showed the prospective correlation between baseline TyG, follow-up HGS/W and diabetes. We found that the level of TyG was positively correlated with the incidence risk of follow-up diabetes (RR 1.712, $P < 0.001$). However, the level of baseline TyG was not significantly related to the level of follow-up HGS/W (β = –0.007, $P = 0.057$).

Table 4 showed the results of subgroup analysis by sex. For men, the level of HGS/W was inversely associated with the level of follow-up TyG (β = –0.204, $P = 0.010$), and a higher level of HGS/W was related to a lower incidence risk of diabetes (RR 0.250, $P = 0.004$). However, the level of baseline HGS/W variation was not related with the level of follow-up TyG variation (β = –0.150, $P = 0.055$) and the incidence risk of diabetes (RR 0.577, $P = 0.270$) for women.

Table 5 showed the results of subgroup analysis by obese or non-obese. For non-obese participants, the level of HGS/W was inversely associated with the level of follow-up TyG (β = –0.192, $P = 0.001$), and a higher level of HGS/W was related to a lower incidence risk of diabetes (RR 0.416, $P = 0.021$). However, the level of baseline HGS/W variation was positively related to the level of follow-up TyG variation (β = 0.615, $P = 0.009$), and the level of HGS/W was not related to incidence risk of diabetes (RR 7.463, $P = 0.084$) for obese participants.

DISCUSSION

Until now, the conclusions of HGS and diabetes have been conflicting. Furthermore, little research data concentrate on Chinese data. Therefore, we used the CHARLS data to explore the relationship between GS, IR and diabetes. The results of the present study were fivefold: (i) confirming that the baseline HGS/W was negatively related to future diabetes; (ii) finding HGS/W was correlated with TyG; (iii) verifying baseline HGS/W levels preceded follow-up TyG levels; (iv) finding the effect of HGS/W on TyG and diabetes in men, not in women; and (v) finding the effect of HGS/W on TyG and diabetes in non-obese participants, not in obese participants.

After controlling for confounding factors, we found that HGS/W was inversely associated with future diabetes among Chinese older people. Previous studies support this finding. Mainous *et al.* showed that people in the USA aged >45 years with diabetes had a significant relationship with lower combined HGS[20]. In addition, in a Korea nationwide survey, HGS was negatively related to type 2 diabetes[11]. A 40–69 years age group multi-ethnic UK study confirmed that a high HGS was associated with a low diabetes prevalence, and this correlation had ethnic differences[10,21]. Hence, the present research complements supplementing the relationship between HGS and future diabetes in the Chinese older population.

Table 1 | Baseline characteristics of study participants by sex and abdominal obesity

Characteristic	Men (n = 2,145)	Women (n = 2,416)	P	Total
Baseline (2011)				
Age (years)	59.9 (8.67)	57.8 (8.59)	<0.001	58.7 (8.68)
Ethnicity, n (%)				
Han	2010 (93.7)	2225 (92.1)	0.039	4237 (92.9)
Others	135 (6.3)	191 (7.9)		326 (7.1)
Education, n (%)				
Illiteracy	664 (31.0)	1435 (59.4)	<0.001	2110 (46.0)
Primary school	632 (29.4)	464 (19.2)		1097 (24.0)
Middle school	571 (26.6)	363 (14.9)		934 (20.5)
High school	184 (8.6)	128 (5.4)		312 (6.8)
Tertiary high school or above	94 (4.4)	26 (1.1)		120 (2.7)
Current smoking, n (%)	1619 (75.5)	183 (7.6)	<0.001	1802 (39.5)
Drinking ≥1 times/month, n (%)	971 (45.3)	184 (7.6)	<0.001	1155 (25.3)
SBP (mmHg)	129.1 (20.07)	129.2 (21.48)	0.945	129.1 (20.82)
DBP (mmHg)	75.7 (12.50)	75.2 (11.74)	0.189	75.4 (12.11)
Pulse (b.p.m.)	71.7 (10.76)	72.1 (9.60)	0.221	71.9 (10.16)
HDL-C (mg/dL)	49.10 (39.82–59.92)	51.42 (42.91–61.08)	<0.001	50.26 (41.37–60.70)
LDL-C (mg/dL)	110.18 (90.85–132.60)	118.30 (97.04–141.89)	<0.001	114.43 (93.94–136.86)
Weight (kg)	61.4 (11.05)	56.3 (10.68)	<0.001	58.7 (11.15)
BMI (kg/m^2)	22.8 (3.49)	24.0 (3.91)	<0.001	23.4 (3.76)
Waist circumference (cm)	83.4 (11.77)	84.1 (12.8)	0.056	83.8 (12.33)
FPG (mmol/L)	5.56 (0.671)	5.57 (0.609)	0.459	5.56 (0.639)
TG (mmol/L)	1.31 (0.852)	1.44 (0.843)	<0.001	1.38 (0.850)
TyG	6.21 (0.561)	6.32 (0.539)	<0.001	6.27 (0.553)
HGS/W	0.60 (0.144)	0.44 (0.128)	<0.001	0.52 (0.155)
Follow up (2015)				
Weight (kg)	61.9 (11.90)	56.8 (11.68)	<0.001	59.2 (2.05)
BMI (kg/m^2)	23.1 (3.79)	24.3 (4.29)	<0.001	23.7 (4.11)
WC (cm)	83.8 (13.74)	85.2 (13.67)	<0.001	84.6 (13.72)
HGS/W	0.54 (0.145)	0.40 (0.117)	<0.001	0.47 (0.148)
TyG	6.27 (0.588)	6.42 (0.564)	<0.001	6.35 (0.580)
DM	276 (12.9)	324 (13.4)	0.521	606 (13.2)

Total n = 4,561. BMI, body mass index; DBP, diastolic blood pressure; DM, diabetes; FPG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; HGS/W, handgrip strength per bodyweight; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides; TyG, triglyceride glucose index; WC, waist circumference. TyG = ln [fasting TG (mmol/L) × FPG (mmol/L) × 0.5 × 159.37]. HGS/W = HGS (kg) / weight (kg).

Table 2 | Prospective associations of baseline handgrip strength per bodyweight with follow up triglyceride glucose index and diabetes

	Follow-up TyG		Incident DM	
	β (95% CI)	P	RR (95% CI)	P
Model 1				
HGS/W	−0.279 (−0.372 to −0.185)	<0.001	0.333 (0.188–0.590)	<0.001
Model 2				
HGS/W	−0.246 (−0.354 to −0.138)	<0.001	0.317 (0.164–0.613)	0.001
Model 3				
HGS/W	−0.173 (−0.281 to −0.065)	0.002	0.375 (0.191–0.736)	0.004

Model 1: adjusted for baseline triglyceride glucose index (TyG). Model 2: adjusted for factors in model 1 and baseline age, ethnicity and sex. Model 3: adjusted for all variables in model 2 plus baseline education, current smoking, alcohol drinking, systolic blood pressure, diastolic blood pressure, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. CI, confidence interval; DM, diabetes; HGS/W, handgrip strength per bodyweight; RR, rate ratio.

Table 3 | Prospective associations of baseline triglyceride glucose index with follow-up handgrip strength per bodyweight and diabetes

	Follow-up HGS/W		Incident DM	
	β (95% CI)	*P*	RR (95% CI)	*P*
Model 1				
TyG	−0.013 (−0.019 to −0.006)	<0.001	1.781 (0.527–2.076)	<0.001
Model 2				
TyG	−0.013 (−0.019 to −0.006)	<0.001	1.835 (1.572–2.142)	<0.001
Model 3				
TyG	−0.007 (−0.014 to −0.000)	0.057	1.712 (1.427–2.053)	<0.001

Model 1: adjusted for baseline triglyceride glucose index (TyG). Model 2: adjusted for factors in model 1 and baseline age, ethnicity and sex. Model 3: adjusted for all variables in model 2 plus baseline education, current smoking, alcohol drinking, systolic blood pressure, diastolic blood pressure, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. DM, diabetes; HGS/W, handgrip strength per bodyweight; RR, rate ratio.

Table 4 | Prospective associations of baseline handgrip strength per bodyweight with follow-up triglyceride glucose index and diabetes according to sex

	Follow-up TyG		Incident DM	
	β (95% CI)	*P*	RR (95% CI)	*P*
Men				
Model 1				
HGS/W	−0.155 (−0.305 to −0.005)	0.043	0.196 (0.081–0.478)	<0.001
Model 2				
HGS/W	−0.262 (−0.418 to −0.107)	0.001	0.255 (0.101–0.646)	0.004
Model 3				
HGS/W	−0.204 (−0.360 to −0.048)	0.010	0.250 (0.097–0.64)	0.004
Women				
Model 1				
HGS/W	−0.224 (−0.375 to −0.072)	0.004	0.314 (0.122–0.808)	0.016
Model 2				
HGS/W	−0.242 (−0.395 to −0.089)	0.002	0.391 (0.150–1.018)	0.054
Model 3				
HGS/W	−0.150 (−0.303 to 0.003)	0.055	0.577 (0.218–1.531)	0.270

Model 1: adjusted for baseline triglyceride glucose index (TyG). Model 2: adjusted for factors in model 1 and baseline age, ethnicity. Model 3: adjusted for all variables in model 2 plus baseline education, current smoking, alcohol drinking, systolic blood pressure, diastolic blood pressure, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. DM, diabetes; HGS/W, handgrip strength per bodyweight; RR, rate ratio.

In addition, some studies showed that there was no relationship between incident diabetes and HGS[22,23]. The diabetes patients in both articles were identified by self-report. Therefore, we believe the null findings could be caused by underestimating the number of diabetes patients. Another extensive multicentric study showed that HGS was not associated with the incidence of type 2 diabetes mellitus[24]. A possible explanation for this study is the relatively short follow-up time and young age. As evidence, one study showed baseline HGS had a significant inverse relationship with the incidence of type 2 diabetes mellitus during a 10-year follow-up period[25].

Although the potential mechanisms responsible for the association of HGS with diabetes have not been fully understood, the following findings could explain the relationship between HGS and the incidence of diabetes. First, defective glucose sensing at the β-cell and IR are two crucial pathophysiological factors for abnormal glucose metabolism[26]. Second, the levels of IR and the protein content of glucose transporter-4 can be increased by muscle strength training[27]. IR participates in the relationship between diabetes and grip strength, which can be explained by the inactivation of the insulin receptor substrate-1 (IRS-1). Pro-inflammatory cytokines promote the phosphorylation of an IRS-1 by relevant protein kinases, such as c-JUN N-terminal kinase and kappa-B kinase β[28]. Then phosphorylated IRS-1 inhibits insulin sensitivity[28]. In addition, can directly reduce IRS-1 activity[29]. Skeletal muscle is the principal place where insulin regulates glucose absorption[30]. Thus, abnormal glucose and energy metabolisms cause the change of HGS. The present results have also shown that the baseline HGS/W level associated with the future IR level, and the IR level positively

Table 5 | Prospective associations of baseline handgrip strength per bodyweight with follow-up triglyceride glucose index and diabetes according to obesity at 2011

	Follow-up TyG		Incident DM	
	β (95%CI)	*P*	RR (95%CI)	*P*
Obese (480)				
Model 1				
HGS/W	0.324 (–0.067 to 0.715)	0.104	4.527 (0.709–28.913)	0.110
Model 2				
HGS/W	0.599 (0.140 to 1.057)	0.011	7.486 (0.805–69.650)	0.077
Model 3				
HGS/W	0.615 (0.154 to 1.075)	0.009	7.463 (0.764–72.914)	0.084
non-obese (4081)				
Model 1				
HGS/W	–0.263 (–0.362 to –0.164)	<0.001	0.371 (0.197–0.699)	0.002
Model 2				
HGS/W	–0.245 (–0.361 to 0.130)	<0.001	0.385 (0.185–0.801)	0.011
Model 3				
HGS/W	–0.192 (–0.307 to 0.077)	0.001	0.416 (0.197–0.878)	0.021

Model 1: adjusted for baseline triglyceride glucose index (TyG). Model 2: adjusted for factors in model 1 and baseline age, ethnicity and sex. Model 3: adjusted for all variables in model 2 plus baseline education, current smoking, alcohol drinking, systolic blood pressure, diastolic blood pressure, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. DM, diabetes; HGS/W, handgrip strength per bodyweight; RR, rate ratio.

correlated with the diabetes incidence risk. Hence, the IR mechanism might explain the negative correlations between HGS and diabetes.

Furthermore, the temporal correlation results only showed that baseline HGS/W was related to future IR. The possible cause is that IR is an impaired response in the whole body, primarily in the liver, adipose tissue and muscle[31]. Hence, early IR will not necessarily appear in muscles. However, if decreased HGS levels occur early, IR will occur in the future. Hence, the present result verifies that baseline HGS/W level preceded the future IR level. Therefore, HGS can be a predictor of future IR and diabetes in older people[20].

Considering the sex dimorphism of HGS, we stratified sex. We only found that low GS levels were associated with IR and diabetes in the older male group. Similarly, a USA study of older adults found a negative correlation between GS and FPG in men[32]. Furthermore, white and black men had a stronger association between GS and diabetes than white and black women[21]. The same result was also found in a Korean group aged 65–80 years[33]. Another study showed that HGS was inversely related to type 2 diabetes incidence risk among 1,632 men[34]. That study reported that low HGS could cause a 27% attributable population fraction in men[34]. In addition, they found that the factor for incidence risk of type 2 diabetes is reduced muscle strength in men[34]. The mechanism might be due to the chromosomes and sociocultural differences; men have relatively more substantial muscle mass[35], higher inflammatory levels[36] and lower insulin sensitivity[37] than women.

In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors in developing insulin resistance[38]. Therefore, we carried out subgroup analysis according to obese or non-obese in our research regarding the inconsistent results between non-obese and obese participants. We believe this inconsistent research result might be due to the small number (480) of obese participants. In addition, one study that was carried out in the Korean population aged between 30 and 79 years showed similar findings that muscle strength was independently associated with diabetes in the non-obese population, but not in the obese population[39]. The World Health Organization uses BMI calculated by height and weight as a measure of nutritional status. Therefore, BMI does not reflect the ratio of muscle to fat[40]. This might be another reason why there is no direct statistical correlation between the HGS/W level of obese people and the risk of diabetes.

Two assumptions were crucial during the present study. As our research was a strictly observational cohort study, the association between GS/W and diabetes might not be causal. Therefore, to minimize the potential bias of reverse causality, we excluded some individuals with a history of diabetes at baseline. Second, we can infer the temporal relationship between grip strength (GS/W) and incident diabetes as the prospective cohort design of CHARLS.

At the same time, the present study also had some limitations. First, the crucial confounding factors, such as dietary patterns and physical activity, were not considered in the present study. Regular physical exercise could prevent age-related muscle strength loss[41] and increase insulin sensitivity[42]. On this basis, we are more confident that HGS could be a valuable predictor of the risk of diabetes. Another limitation is that we did

not distinguish type 1 and type 2 diabetes in the present study. Type 1 diabetes mainly appears during adolescence, rarely in older age. Hence, the present study is dominated by type 2 diabetes.

HGS/W can be used as a simple, easy to measure and inexpensive indicator to predict IR and diabetes by combining all related research. HGS/W is more comfortable to detect and monito than biochemical indicators. In addition, HGS/W might help primary care physicians stratify possible patients requiring further blood test screening. Furthermore, we require further studies to clarify the pathophysiological mechanisms underlying the relationship.

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DISCLOSURE

The authors declare no conflict of interest.

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