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Increasing waist circumference is associated with decreased levels of glycated albumin

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ABSTRACT

Background: Glycated albumin (GA) levels are affected by body fat and its distribution. We explored the association of waist circumference (WC) with GA and to assess the extent to which WC influences GA.
Methods: We recruited 1799 subjects (age 26–82 y) from communities. GA was determined using the enzyme method, and glycated hemoglobin A_{1c} (HbA_{1c}) was detected using high-performance liquid chromatography.
Results: Subjects with central obesity had lower GA and GA/HbA_{1c} than those without (both $P < .01$). GA and GA/HbA_{1c} were negatively correlated with central obesity (both $P < .01$), whereas HbA_{1c} was not correlated ($P = .833$). In the euglycemic and hyperglycemic subpopulations, GA and GA/HbA_{1c} showed decreasing trends as WC levels increased (both P for trends < 0.01). WC was a significant negative determinant of GA ($P < .05$). In the hyperglycemic subpopulation, the GA value decreased by approximately 0.15% for each 5 cm increment in WC regardless of the presence of central obesity.
Conclusions: The GA value was reduced by approximately 0.15% for each 5 cm increment in WC, suggesting that more attention should be paid to actual blood glucose underestimated by GA in obese people.

1. Introduction

Obesity is a pathological state in which energy intake and energy expenditure are out of the balance, ultimately leading to an increased risk for several diseases [1]. With the rapid development of social economics and the corresponding changes in diet and physical activity, the increasing prevalence of obesity is becoming a worldwide health concern. In 2014, approximately 641 million people were obese in the world [2]. In addition to excess body fat, obese people tend to have abnormal body fat distribution, and central obesity is especially prevalent. Central obesity is also considered an important health threat, owing to the evidence that subjects with central obesity have a cardiovascular mortality risk approximately two times higher than those without central obesity, even in a normal weight cohort [3]. Central obesity is often accompanied by inflammation and oxidative stress and is closely associated with insulin resistance, which is the driving risk that leads to diabetes [4].

Adequate glycemic control is critical in diabetes care. Glycated albumin (GA), reflecting the average blood glucose in the two to three weeks prior to the measurement, is thought to be a powerful indicator of glycemic control over the span of a month, and its levels are not affected by changes in serum protein levels, anticoagulants, or non-specific reducing substances. GA provides supplementary and valuable information reflecting glycemic control over a shorter period of time in comparison to the current gold standard of glycated hemoglobin A_{1c} (HbA_{1c}) because albumin binds blood glucose faster than hemoglobin does [5]. However, a negative correlation between serum GA levels and body mass index (BMI) was found in a study that included a population with normal blood glucose [6]. Our previous study reported that the value of GA decreased by approximately 0.13% for each 1 kg/m^2 increment in BMI, regardless of the presence of diabetes [7].

Studies related to the influence of central obesity on GA are currently limited. We have previously found that abdominal fat accumulation is significantly and negatively associated with GA [8]. Therefore, GA will underestimate the actual blood glucose of individuals with central obesity; however, no research has focused on the extent to

Abbreviations: 2-h PG, 2-h plasma glucose; ALT, alanine aminotransferase; BMI, body mass index; CRP, C-reactive protein; FINS, fasting insulin; FPG, fasting plasma glucose; GA, glycated albumin; HbA_{1c}, glycated hemoglobin A_{1c}; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment-insulin resistance index; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglyceride; WC, waist circumference.

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which central obesity influences GA. Waist circumference (WC) is an indicator of central obesity and is easy to measure, making it more appropriate for clinical application than visceral fat area assessed by magnetic resonance imaging.

2. Materials and methods

2.1. Subjects

This study enrolled 1799 subjects (age range: 26–82 y) from Shanghai communities from October 2015 to July 2016. All participants underwent an examination comprising a standardized questionnaire, physical examinations and biochemical measurements. The questionnaire included information on current and previous illnesses and medications. Subjects were included if they voluntarily participated in the study and were able to provide the information required for the study. Exclusion criteria included a known history of diabetes and/or hypoglycemic therapy; cardiovascular disease; malignancy; pregnancy; severe anemia or hypoproteinemia; severe liver, kidney or thyroid dysfunction; treatment with steroid hormones or thyroxine; the presence of an infectious condition [C-reactive protein (CRP) > 10 mg/l].

This study was conducted according to the World Medical Association Declaration of Helsinki and was approved by the Ethics Committee of Shanghai Jiao Tong University Affiliated Sixth People's Hospital. All participants provided written informed consent prior to participation.

2.2. Biochemical measurements

The following biochemical indices were measured in morning venous blood samples after a 10-h overnight fast: fasting blood glucose (FPG), HbA_{1c}, GA, fasting insulin (FINS), blood lipids [serum total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C)], CRP, alanine aminotransferase (ALT), and creatinine. For subjects with no prior diagnosis of diabetes mellitus, a 2-h plasma glucose (2-h PG) blood sample was obtained after a 75-g oral glucose tolerance test. All biochemical indicators were measured based on standard methods as previously described [9]. The homeostasis model assessment-insulin resistance (HOMA-IR) index was calculated using the formula HOMA-IR = FPG (mmol/l) × FINS (mU/l)/22.5.

Serum albumin levels were measured via bromocresol green method (Shanghai Kehua Bio-Engineering Co., Ltd.). GA was measured using an enzyme method (Lucica GA-L, Asahi Kasei Pharma) on a 7600 analyzer (Hitachi), and the intra-assay and interassay CVs were < 3.30% and < 4.73%, respectively. HbA_{1c} was detected by high-performance liquid chromatography (Variant II hemoglobin analyzer, Bio-Rad), for which the intra-assay and inter-assay CVs were < 2.58% and < 3.39%, respectively.

2.3. Body fat parameters and covariates

The standard methods of anthropometric assessments, including blood pressure, height and weight, have been described elsewhere [9]. BMI was calculated as weight (kg) / height^2 (m^2). WC was measured by a tape around the horizontal plane between the inferior costal margin and the iliac crest on the mid-axillary line with the subject in a standing position. Central obesity was defined as WC ≥ 90 cm in men and WC ≥ 85 cm in women [10]. According to the 1999 World Health Organization criteria, impaired glucose regulation was diagnosed when 6.1 mmol/l ≤ FPG < 7.0 mmol/l and/or 7.8 mmol/l ≤ 2-h PG < 11.1 mmol/l; diabetes was diagnosed when FPG ≥ 7.0 mmol/l and/or 2-h PG ≥ 11.1 mmol/l [11]. Both impaired glucose regulation and diabetes were classified as hyperglycemia.

2.4. Statistical analyses

The SPSS for Windows software (ver. 20.0) was used for all statistical analyses. The normality of the data distribution was determined by the one-sample Kolmogorov-Smirnov test. Continuous variables are expressed as the means ± SD or medians with interquartile ranges. Categorical variables are expressed as numbers with percentages. Comparisons between the 2 groups were performed with Student's *t*-test for data with a normal distribution, the Mann-Whitney *U* test for data with a skewed distribution, and the χ^2 test for categorical variables. The subjects were divided into 8 groups according to WC with 5 cm increments. A one-way ANOVA was used for trend analyses of between-group variance of GA, HbA_{1c}, and GA/HbA_{1c} levels. Logistic regression was performed to examine the relationship among GA, HbA_{1c}, and GA/HbA_{1c} levels and central obesity. The independent correlations among WC and GA, HbA_{1c}, and GA/HbA_{1c} levels were investigated using multivariate linear regression analyses. All reported *P* values were 2-tailed, and $P < .05$ was considered statistically significant.

3. Results

3.1. Characteristics of the study subjects

A total of 1799 subjects comprising 885 euglycemic subjects and 914 hyperglycemic subjects aged 60 y (55–65 y) were enrolled in this study. Table 1 lists the general clinical characteristics of the subjects. Overall, the levels of GA, HbA_{1c}, and GA/HbA_{1c} were 13.8 ± 1.6%, 5.7 ± 0.5%, and 2.4 ± 0.3, respectively. The median WC was 84.00 (78.00–90.00) cm, and 657 (36.5%) subjects among the total population exhibited central obesity. Compared with the subjects without central obesity, the proportion of subjects with diabetes and impaired glucose regulation was significantly higher in those with central obesity (19.3% vs 9.1%, $P < .01$; 41.2% vs 36.1%, $P < .01$, respectively). In the euglycemic or hyperglycemic groups, the GA and GA/HbA_{1c} levels were lower in subjects with central obesity than in those without (13.0 ± 1.1% vs 13.6 ± 1.1%, $P < .01$; 13.9 ± 1.7% vs 14.4 ± 1.9%, $P < .01$, respectively). In the euglycemic group, serum albumin levels were also lower in subjects with central obesity ($P < .01$). In the hyperglycemic group, compared with subjects without central obesity, subjects with central obesity had higher levels of HbA_{1c} (5.9 ± 0.5% vs 5.8 ± 0.6%, $P < .01$); the 2-h PG levels were elevated in subjects with central obesity as well ($P < .05$). In addition, subjects with central obesity tended to have a higher BMI, WC, systolic blood pressure, diastolic blood pressure, FPG, FINS, HOMA-IR, TG, LDL-C, CRP, ALT and creatinine levels regardless of the presence of hyperglycemia, whereas HDL-C was significantly lower in subjects with central obesity ($P < .05$).

3.2. Correlation of GA, HbA_{1c}, and GA/HbA_{1c} with central obesity

To explore the relationships among GA, HbA_{1c}, GA/HbA_{1c} levels and central obesity, three different models were created and are shown in Fig. 1. In Model 1, GA and GA/HbA_{1c} levels were both inversely correlated with central obesity after adjusting for age and gender (OR = 0.841, $P < .01$; OR = 0.136, $P < .01$, respectively). The correlation remained unchanged after further adjustment for blood pressure, lipids, etc. in Model 2 ($P < .01$). Furthermore, the negative correlations of GA and GA/HbA_{1c} levels with central obesity remained significant even when HOMA-IR and 2-h PG were adjusted in Model 3 (OR = 0.795, $P < .01$; OR = 0.269, $P < .01$, respectively). In Model 1 and Model 2, HbA_{1c} was positively correlated with central obesity ($P < .05$), but the correlation disappeared after further adjustment of HOMA-IR and 2-h PG ($P = .833$).

Table 1
Clinical characteristics of the study subjects.

Variables	Euglycemia		Hyperglycemia	
	Central obesity (−)	Central obesity (+)	Central obesity (−)	Central obesity (+)
N, (Men/Women)	626 (209/417)	259 (118/141)	516 (207/309)	398 (171/227)
Age (y)	58 (54–63)	59 (54–64)	62 (57–66)	61 (56–65)*
BMI (kg/m^2)	22.39 ± 2.26	26.96 ± 2.43**	22.74 ± 2.11	27.18 ± 2.56**
WC (cm)	79.00 (73.88–82.13)	92.00 (89.00–96.00)**	80.00 (76.00–84.00)	93.00 (89.50–97.00)**
SBP (mmHg)	125 (113–136)	133 (123–146)**	130 (119–142)	137 (125–149)**
DBP (mmHg)	75 (68–82)	79 (72–87)**	77 (71–83)	81 (74–89)**
FPG (mmol/l)	5.42 (5.19–5.72)	5.57 (5.32–5.78)**	6.11 (5.66–6.49)	6.25 (5.80–6.76)**
2-h PG (mmol/l)	6.12 (5.14–6.97)	6.13 (5.18–6.93)	8.82 (7.89–10.10)	9.06 (8.08–11.06)*
HbA_{1c} (%)	5.6 ± 0.4	5.6 ± 0.4	5.8 ± 0.6	5.9 ± 0.5**
GA (%)	13.6 ± 1.1	13.0 ± 1.1**	14.4 ± 1.9	13.9 ± 1.7**
GA/HbA_{1c}	2.4 ± 0.2	2.3 ± 0.2**	2.5 ± 0.3	2.3 ± 0.2**
FINS (mu/l)	7.19 (5.26–9.38)	10.49 (8.05–14.41)**	8.77 (6.28–12.14)	13.15 (9.79–17.88)**
HOMA-IR	1.74 (1.27–2.35)	2.58 (1.94–3.59)**	2.39 (1.71–3.38)	3.69 (2.60–5.23)**
TC (mmol/l)	5.30 (4.69–5.92)	5.33 (4.68–5.97)	5.37 (4.77–6.13)	5.43 (4.87–6.19)
TG (mmol/l)	1.15 (0.87–1.68)	1.52 (1.12–2.25)**	1.42 (1.00–2.12)	1.73 (1.31–2.41)**
HDL-C (mmol/l)	1.49 (1.25–1.74)	1.28 (1.11–1.54)**	1.43 (1.20–1.69)	1.29 (1.10–1.46)**
LDL-C (mmol/l)	3.19 ± 0.81	3.33 ± 0.79*	3.31 ± 0.84	3.44 ± 0.83*
CRP (mg/l)	0.66 (0.34–1.24)	1.07 (0.58–1.98)**	0.86 (0.39–1.58)	1.32 (0.78–2.66)**
Albumin (g/l)	49.49 ± 2.39	48.97 ± 2.38**	49.92 ± 2.28	49.77 ± 2.47
ALT (U/l)	17.00 (13.00–22.00)	20.00 (16.00–28.00)**	18.00 (14.00–23.00)	22.00 (17.00–30.00)**
Creatinine (μmol/l)	61.50 (53.00–72.00)	64.00 (55.00–77.00)*	62.00 (54.00–73.00)	65.00 (56.00–75.00)*

Central obesity (+) versus Central obesity (−).
Abbreviation: BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; 2-h PG, 2-h plasma glucose; HbA_{1c}, glycated hemoglobin A_{1c}; GA, glycated albumin; FINS, fasting insulin; HOMA-IR, homeostasis model assessment-insulin resistance index; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; CRP, C-reactive protein; ALT, alanine aminotransferase.
* $P < .05$.
** $P < .01$.

3.3. The effect of WC on the levels of GA, HbA_{1c}, and GA/HbA_{1c}

Subjects were divided into groups according to the values of WC < 70 cm, 70– < 75 cm, 75– < 80 cm, 80– < 85 cm, 85– < 90 cm, 90– < 95 cm, 95– < 100 cm and ≥100 cm. The cut-off points were also applied to the euglycemic and hyperglycemic subpopulations. As shown in Fig. 2, GA showed a downward trend with increasing levels of WC in the total population (P for trend < 0.01). In subjects with euglycemia or hyperglycemia, the decreasing trends of GA were also found to accompany increasing levels of WC (both P for trends < 0.01); however, the changing trends of HbA_{1c} failed to reach statistical significance (both P for trends > 0.05), whereas GA/HbA_{1c} displayed a downward trend (both P for trends < 0.01).

A multiple linear regression analysis was performed in which GA, HbA_{1c} and GA/HbA_{1c} were designated as dependent variables. Table 2 shows that WC is a significant negative determinant of GA and GA/HbA_{1c} after controlling for potential confounding variables, including age, gender, blood pressure, TG, HDL-C, LDL-C, CRP, HOMA-IR, 2-h PG and BMI (both $P < .05$). In addition, there was no correlation between WC and HbA_{1c} ($P = .138$). In subjects with hyperglycemia, WC remained independently correlated with GA ($P < .05$) but not with HbA_{1c} or GA/HbA_{1c} ($P = .398$; $P = .112$).

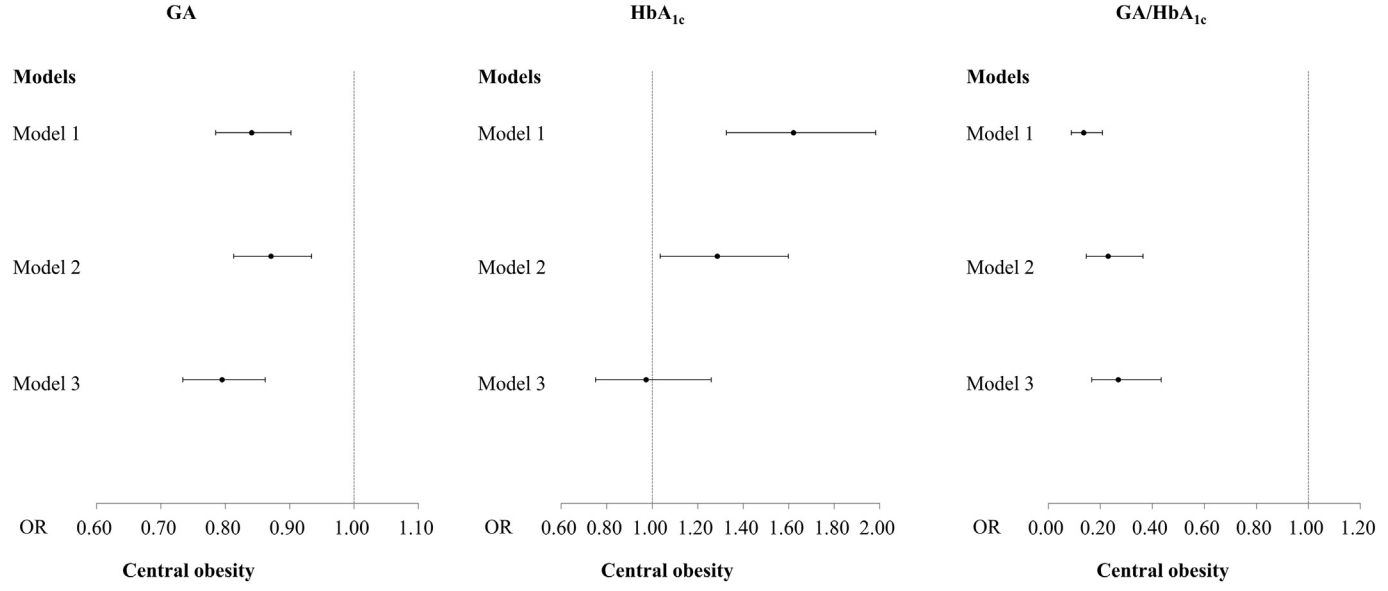

Fig. 1. The association of GA, HbA_{1c}, and GA/HbA_{1c} levels with risk of central obesity in different models.

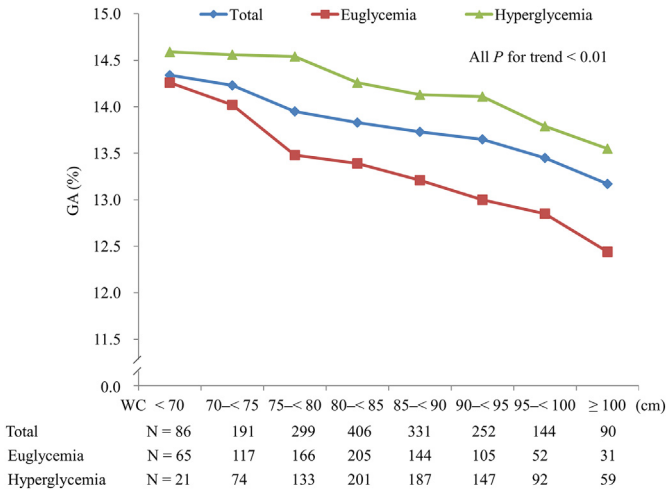

Fig. 2. The distribution of GA levels according to 5 cm increments in waist circumference in all subjects, euglycemic subjects and hyperglycemic subjects. GA presented a downward trend with increasing waist circumference levels (all *P* for trends < 0.01).

In the total population, the regression equation of WC and GA was GA (%) = −0.028 × WC (cm) + 16.199. That is, each 5 cm increment in WC corresponded to a 0.14% decrease in the GA value. In the subpopulation with hyperglycemia, the regression equation for WC and GA was GA (%) = −0.030 × WC (cm) + 16.788. For each 5 cm increment in WC, the GA value was reduced by approximately 0.15%; moreover, further analysis showed that the regression equation in subjects without central obesity was GA (%) = −0.030 × WC (cm) + 16.803, and it was GA (%) = −0.030 × WC (cm) + 16.732 in subjects with central obesity. Therefore, regardless of the presence of central obesity, the GA value decreased by approximately 0.15% for each 5 cm increment in WC.

4. Discussion

We demonstrated that GA displayed a downward trend with increasing level of WC, regardless of the presence of hyperglycemia, in a community-based cohort, and the value of GA decreased by 0.14% for each 5 cm increment in WC. The correlation was also notable in subjects with hyperglycemia, whether they had central obesity or not, and the GA value decreased by approximately 0.15% for each 5 cm increment in WC.

GA, an indicator reflecting the mean glucose concentration over the previous 2 to 3 weeks, has an absolute advantage in evaluating short-term blood glucose control. Accumulating evidence has revealed that the combination of GA with FPG and HbA_{1c} is sufficient to improve the efficiency of screening and diagnosing diabetes and GA is closely correlated with chronic complications and the pathophysiological progression of diabetes [12–17]. In addition, it is more convenient to use in clinical practice owing to the reasonable evidence that nonfasting measurements of GA levels could be employed for diabetes screening as well [18]. However, the levels of GA can vary based on certain clinical factors, including body fat. A study from Japan demonstrated that BMI was an independent negative risk factor for GA in 212 nondiabetic individuals [19]. Another study also reported a significantly inverse correlation between BMI and GA in 84 young euglycemic adults in New Zealand [6]. Our previous study revealed that in 2562 outpatients who received an oral glucose tolerance test at the hospital in Shanghai, the levels of GA were independently and negatively correlated with BMI regardless of diabetes status. For each 1 kg/m^2 increase in BMI, the value of GA was reduced by approximately 0.13% [7]. Obesity refers to not only excessive body fat but also abnormal body fat distribution. Central obesity has a closer association with related metabolic diseases than an isolated increase in body fat. In terms of central obesity, we previously found that visceral fat area assessed using magnetic resonance imaging was also independently associated with GA in 2563 subjects with normal blood glucose [8].

WC is widely used in assessing central obesity due to its simple measurement, and it can identify the risks for metabolic and cardiovascular diseases as effectively as the current gold standard of measuring visceral fat areas [9,20,21]. However, the extent to which central obesity indicators influence GA levels has not yet been detected. The strength of this study was that we identified the inverse correlation between WC, the simple indicator of central obesity, and GA; moreover, the correlation was assessed within the strata of hyperglycemia and central obesity. WC was correlated with GA levels independent of metabolic factors such as blood glucose, blood lipids and blood pressure; furthermore, a decreased GA value was found to accompany increasing levels of WC in the total population as well as the subpopulation with hyperglycemia, demonstrating significant implications for the clinical application of GA in blood glucose monitoring.

GA/HbA_{1c} is reported to be closely associated with chronic complications of diabetes [22–25]. In 613 patients with type 2 diabetes, retinopathy was found to be significantly associated with GA/HbA_{1c} [26]. Therefore, the relationship between GA/HbA_{1c} and body fat parameters has recently received increased attention. We previously reported a significant decrease in GA/HbA_{1c} with increasing BMI levels in 1223 diabetic patients and 1339 nondiabetic individuals; thus, GA/HbA_{1c} was shown to be independently and negatively influenced by

Table 2
Associations among GA, HbA_{1c}, GA/HbA_{1c}, and WC in total and hyperglycemic subjects.

Models	GA			HbA_{1c}			GA/HbA_{1c}		
	standardized β	t	*P*	standardized β	t	*P*	standardized β	t	*P*
Total									
Model 1	−0.174	−7.173	< 0.001	0.140	5.749	< 0.001	−0.309	−12.949	< 0.001
Model 2	−0.213	−8.217	< 0.001	0.010	0.398	NS	−0.242	−8.609	< 0.001
Model 3	−0.147	−3.450	0.001	−0.063	−1.484	NS	−0.110	−2.392	0.017
Hyperglycemia									
Model 1	−0.144	−4.226	< 0.001	0.136	3.987	< 0.001	−0.295	−8.877	< 0.001
Model 2	−0.184	−5.163	< 0.001	0.034	0.985	NS	−0.250	−6.492	< 0.001
Model 3	−0.126	−2.068	0.039	−0.050	−0.846	NS	−0.105	−1.593	NS

Model 1: adjusted for age and gender.
Model 2: adjusted for age, gender, SBP, DBP, TG, HDL-C, LDL-C, CRP, HOMA-IR and 2-h PG.
Model 3: adjusted for age, gender, SBP, DBP, TG, HDL-C, LDL-C, CRP, HOMA-IR, 2-h PG and BMI.
Abbreviation: GA, glycated albumin; HbA_{1c}, glycated hemoglobin A_{1c}; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; CRP, C-reactive protein; HOMA-IR, homeostasis model assessment-insulin resistance index; 2-h PG, 2-h plasma glucose; BMI, body mass index.

BMI [7]. In the present study, WC was negatively correlated with GA/HbA_{1c} regardless of blood glucose status.

The detailed mechanisms behind the inverse relationship between central obesity and GA are not well understood. Recent studies have suggested that inflammation may play an important role. Obesity is a chronic low-grade inflammatory disease state. Inflammation reduces the rate of albumin synthesis and increases its rate of catabolism, which ultimately leads to an increasing conversion of serum albumin in obese people [19]. In addition, GA plays a role in stimulating monocytic cells to secrete increased amounts of pro-inflammatory cytokines. Recent studies have revealed that GA is directly involved in the signaling pathway and the stimulation of these pro-inflammatory biomarkers, which leads to increased levels of inflammation [27]. The specific mechanisms involved in the relationship between WC and GA need future research to be clarified.

5. Limitations

We acknowledge that our study has several limitations. First, this study only recruited subjects from communities in Shanghai, where the results might have been affected by population and regional variables. Second, the cross-sectional study design did not allow for the clarification of a causal relationship between WC and GA, which must be further confirmed in large-scale prospective studies and mechanistic studies.

6. Conclusion

In conclusion, subjects with central obesity had lower GA levels than those without central obesity. The negative correlation between WC and GA was independent of metabolic factors, including blood glucose, blood lipids and blood pressure. In subjects with hyperglycemia, the value of GA decreased by approximately 0.15% for each 5 cm increment in WC regardless of central obesity.

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