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Racial Differences in Glycemic Markers: A Cross-sectional Analysis of Community-Based Data

Elizabeth Selvin, PhD, MPH,
Welch Center for Prevention, Epidemiology and Clinical Research and the Johns Hopkins Bloomberg School of Public Health, 2024 E. Monument Street, Suite 2-600, Baltimore MD 21287

Michael W. Steffes, MD, PhD,
University of Minnesota, MMC 609, Office: Room B203 Mayo, 420 Delaware Street, S.E., Minneapolis, MN 55455

Christie M. Ballantyne, MD,
Department of Medicine, Baylor College of Medicine, 6565 Fannin Street, Room F756, Mail Station A601, Houston, TX, 77030

Ron C. Hoogeveen, PhD,
Department of Medicine, Baylor College of Medicine, 6565 Fannin St. MS F-701, Room F756, Mail Station F701, Houston, TX, 77030

Josef Coresh, MD, PhD, and
Welch Center for Prevention, Epidemiology and Clinical Research and the Johns Hopkins Bloomberg School of Public Health, 2024 E. Monument Street, Suite 2-600, Baltimore MD 21287

Frederick L. Brancati, MD, MHS
Welch Center for Prevention, Epidemiology and Clinical Research and the Department of Medicine, The Johns Hopkins University, 2024 East Monument St, Suite 2-600, Baltimore, MD 21205

Abstract

Background—Although black and white differences in hemoglobin A_{1c} (HbA_{1c}) values are well established, recent studies suggest that the difference might not reflect differences in glycemia.

Objective—To investigate racial disparities in glycemic markers, including those that reflect biological processes independent of hemoglobin glycation and erythrocyte turnover.

Design—Cross-sectional.

Setting—Community-based.

Participants—1376 nondiabetic and 343 diabetic adults in a substudy of the Atherosclerosis Risk in Communities Study.

Measurements—Hemoglobin A_{1c}, fasting glucose, glycated albumin, fructosamine, and 1,5-anhydroglucitol levels.

Results—In persons with and without diabetes, black persons had significantly higher values of HbA_{1c}, glycated albumin, and fructosamine levels compared with white persons before and after adjustment for covariates and fasting glucose concentration. Serum 1,5-anhydroglucitol, which is reduced in the setting of hyperglycemia-induced glycosuria, was lower in black persons compared with white persons, although this difference was statistically significant only in nondiabetic adults.

Disclosure: The authors have no relevant financial relationships to disclose.

Limitation—The design was cross-sectional, a limited number of participants with a history of diabetes were included, and the study did not include integrated measures of circulating nonfasting glycemia

Conclusion—Black and white differences in glycated albumin, fructosamine, and 1,5-anhydroglucitol parallel black and white differences in HbA_{1c} values. Racial differences in hemoglobin glycation and erythrocyte turnover cannot explain racial disparities in these serum markers. The possibility that black persons have systematically higher levels of nonfasting glycemia deserves further study.

In a major change to clinical guidelines, glycated hemoglobin A_{1c} (HbA_{1c}) has recently been recommended for use as a diagnostic test for diabetes in the United States (1). However, there is on-going debate about the interpretation of HbA_{1c} values among black persons and the possible need for race-based HbA_{1c} cut points (2–14). Black persons are well known to have higher HbA_{1c} values than their white counterparts in both the presence and absence of diabetes (2, 4, 15–21) and even in the setting of a low fasting glucose measurement (13, 17). It is unclear whether this disparity stems from racial differences in pre- or postprandial glycemia (6, 13, 22), the tendency of hemoglobin to undergo glycation (5), erythrocyte turnover, or erythrocyte permeability to glucose. Serum glycemic markers, such as fructosamine, glycated albumin, and 1,5-anhydroglucitol offer ways to evaluate racial disparities in glycemia that are biologically independent of erythrocyte turnover and hemoglobin glycation.

Hemoglobin A_{1c} results from the glycation of hemoglobin in erythrocytes and represents long-term (2- to 3-month) glycemia. In contrast, fructosamine and glycated albumin reflect the modification of serum proteins (mainly albumin) by glucose and are markers of 2- to 4-week endogenous glucose exposure. 1,5-Anhydroglucitol is a marker of glycemia-induced glycosuria because reabsorption of filtered 1,5-anhydroglucitol in the proximal tubule is competitively inhibited by glucose (25, 26). Lower serum 1,5-anhydroglucitol reflects high circulating glucose and the occurrence of glycosuria over the previous 1 to 2 weeks (26–31).

We compared nontraditional serum glycemic markers (glycated albumin, fructosamine, and 1,5-anhydroglucitol) with standard markers (HbA_{1c} and fasting glucose) in participants in the Atherosclerosis Risk in Communities (ARIC) Study to determine if the documented higher values of HbA_{1c} in black persons were also observed for serum measures of glycemia. We hypothesized that HbA_{1c}, fructosamine, and glycated albumin values would be higher in black persons, and 1,5-anhydroglucitol would be lower in black persons, as compared with white persons before and after adjustment for fasting glucose concentration.

Methods

Study Population

We conducted a cross-sectional study of participants from the ARIC study who participated in the ARIC Carotid Magnetic Resonance Imaging (CARMRI) substudy. The ARIC study is an ongoing, community-based prospective cohort study of 15 792 black and white adults originally enrolled from 1987 to 1989 from 4 U.S. communities (Forsyth County, North Carolina; Jackson, Mississippi; Minneapolis, Minnesota; and Washington County, Maryland) (32–34). Just more than 2000 participants from the original cohort, now aged 60 to 84 years, were recruited into the CARMRI substudy from 2004 to 2005 using a stratified sampling plan (35). Black participants were enrolled only at the Forsyth County and Jackson field centers. In addition to the magnetic resonance imaging examination, trained technicians did a comprehensive clinical examination, obtained blood specimens, and conducted an interview to obtain information on health status and risk factors. Our final sample was

limited to 1719 participants (343 with a history of diabetes and 1376 without) after excluding those who fasted less than 8 hours (n = 20) or who were missing variables of interest (n = 327). Of the 424 black participants included in this study sample, most (n = 396) were recruited from the Jackson field center.

Institutional review boards at each clinical site approved the study protocol, and written informed consent was obtained from all participants

Measurement of Glycemic Markers

Hemoglobin A_{1c} was measured from whole blood samples as part of the original CARMRI protocol using the Tina-quant II method (Roche Diagnostics) implemented on a Roche Hitachi 911 Analyzer. This method is standardized to the Diabetes Control and Complications Trial assay. In 2009, we measured glycated albumin (Asahi Kasei Lucica GA-L, Tokyo, Japan), fructosamine (Roche Diagnostics), and 1,5-anhdroglucitol (GlycoMark, Winston-Salem, North Carolina) from stored serum specimens using a Roche Modular P800 system. The inter-assay coefficients of variation were 2.7% for glycated albumin, 3.7% for fructosamine, and 4.8% for 1,5-anhydroglucitol.

Other Variables of Interest

Other measurement protocols in ARIC CARMRI were identical to those implemented in the original ARIC study (35). Blood samples were assayed for total and high-density lipoprotein cholesterol, glucose, and high-sensitivity C-reactive protein levels using conventional techniques. Body mass index was computed from measured height and weight. Information on cigarette smoking and alcohol consumption was elicited during the interview. Resting systolic blood pressure (average of 2 readings) was measured using a random-zero sphygmomanometer. Participants were asked to bring current medications to the visit, and information on cholesterol-and blood pressure–lowering medications was also obtained during the interview. Diabetes history was determined by use of glucose-lowering medications or a self-reported physician diagnosis of diabetes. Previous history of coronary heart disease included a reported history of coronary heart disease, an adjudicated coronary heart disease event during follow-up to the CARMRI visit, or both (34).

Statistical Analysis

Characteristics of the study population were calculated both overall and by black or white race. We compared mean values of each glycemic marker by race separately in persons with and without a history of diagnosed diabetes. We used multivariable linear regression models to assess the independent association of race with each glycemic marker in original units and expressed in SD units (standardized regression) after adjustment for confounding factors and fasting glucose concentration. All analyses were weighted by the inverse of the sample fractions in the study sampling strata using methods for the analysis of complex sample survey design data (35). All statistical analyses were performed by using Stata/SE, version 11.0 (StataCorp, College Station, Texas).

Role of the Funding Source

The funding source had no role in the study design, conduct, or interpretation of results.

Results

Table 1 shows the characteristics of the study population. Black participants were less likely to be men, had a higher body mass index, higher total cholesterol concentration, and higher C-reactive protein concentration as compared with white persons. Compared with white persons, black persons were more likely to have diabetes, to have less than a high school

education, or to be taking blood pressure–lowering medication. In contrast, black persons were less likely to be current drinkers, have a history of coronary heart disease, and be taking cholesterol-lowering medications. Differences in each glycemic marker by race and diabetes status are shown in Table 2. In this unadjusted comparison, black persons had significantly higher values of HbA_{1c}, glycated albumin, and fructosamine, as compared with white persons with and without diabetes. 1,5-anhydroglucitol, which is inversely related to glycosuria, was lower in black persons compared with white persons, although this difference was only of borderline statistical significance.

The Figure shows the adjusted standardized differences (SD units) in glycemic markers. Even after adjustment for all covariates and fasting glucose concentration, black persons had higher values of HbA_{1c}, glycated albumin, and fructosamine as compared with white persons. The standardized difference between blacks and whites for serum fructosamine and serum glycated albumin was larger than that for fasting glucose and similar to the difference observed for HbA_{1c} (that is, about 0.5 SDs higher in black persons compared with white persons). After adjustment, racial differences in fasting glucose and 1,5-anhydroglucitol were only statistically significant among persons without a history of diabetes. Tables 3 and 4 show the crude and adjusted mean differences by race for each glycemic marker (black minus white) in original and SD units. In sensitivity analyses, we compared weighted and unweighted results to evaluate the effect of the complex sample survey design. Results were similar in unweighted analyses that did not account for the complex sampling design (data not shown). Because geographic differences cannot be easily separated from race differences as most black participants were recruited from the Jackson field center, we repeated our analyses using data only from the Forsyth County field center (which recruited both black persons and white persons). Results from this subanalysis (data not shown) were very similar.

Discussion

Our results confirm that HbA_{1c} is higher in black persons compared with white persons, even in analyses stratified by diabetes status and after adjustment for known confounding factors and for fasting glucose concentration. We also demonstrated that markers of serum protein glycation---glycated albumin and fructosamine---are higher in black persons compared with white persons with differences similar in magnitude to those observed for HbA_{1c}. Similarly, 1,5-anhydroglucitol, which is lowered by postprandial glycemic excursions, was lower in black persons compared with white persons, but these results were weaker. Our results are consistent with other analyses from the ARIC study that demonstrate equal performance of HbA_{1c} in black and white persons for the prediction of vascular events, mortality, and microvascular disease (36, 37).

As in previous studies (2, 13), fasting glucose concentrations were similar in black compared with white persons. It is important to note that the study with the most accurate representation of true glycemic exposure over time, the A1C-Derived Average Glucose study, demonstrated that a single fasting glucose is a poor measure of average glycemia, whereas average blood glucose—assessed using continuous blood glucose monitoring—correlates very highly with HbA_{1c} ($r = 0.89$) (22). This implies that racial differences in HbA_{1c} might be driven by racial differences in nonfasting glycemia.

Several limitations of this study deserve mention. First, the number of persons with a history of diagnosed diabetes ($n = 343$) was relatively few. Second, we had measurements of each glycemic marker at only 1 point in time in the CARMRI Study, a subsample of the ARIC population. Third, the cross-sectional design precluded us from examining the long-term clinical implications of the observed racial differences in glycemic makers. Furthermore,

because all black participants were recruited at 2 study sites in the ARIC CARMRI study (Jackson, Mississippi and Forsyth County, North Carolina) we could not definitively separate the effects of race from those of region, although it is worth noting that the racial differences in HbA_{1c} and fasting glucose observed here are of similar magnitude and direction to those observed in the National Health and Nutrition Examination Survey and other cohorts. Finally, we only had direct measurements of glucose on fasting samples; we lacked direct data on glycemia at other time points.

Strengths of this study were the rigorous measurement of diabetes risk factors, the biracial community-based sample, the comparative analyses of different glycemic markers, and excellent laboratory performance demonstrated for each of the serum glycemic markers.

Glycated albumin, fructosamine, and 1,5-anhydroglucitol are unaffected by hematologic factors. Thus, the main implication of our study is that racial disparities in HbA_{1c} values are not explained by racial differences in hemoglobin glycatability, erythrocyte turnover, or erythrocyte permeability. One interpretation of our findings is that higher HbA_{1c} reflects higher concentrations of nonfasting glycemia in black persons compared with their white counterparts. Previous studies have documented that black persons consume diets of higher glycemic index and glycemic load compared with white persons (40, 41)—such diets could produce higher concentrations of postprandial glycemia. However, without direct measurements of nonfasting glycemia, we were unable to confirm this interpretation. Future research should directly compare nonfasting glycemia in black persons and white persons and investigate dietary and nondietary factors as possible mediators.

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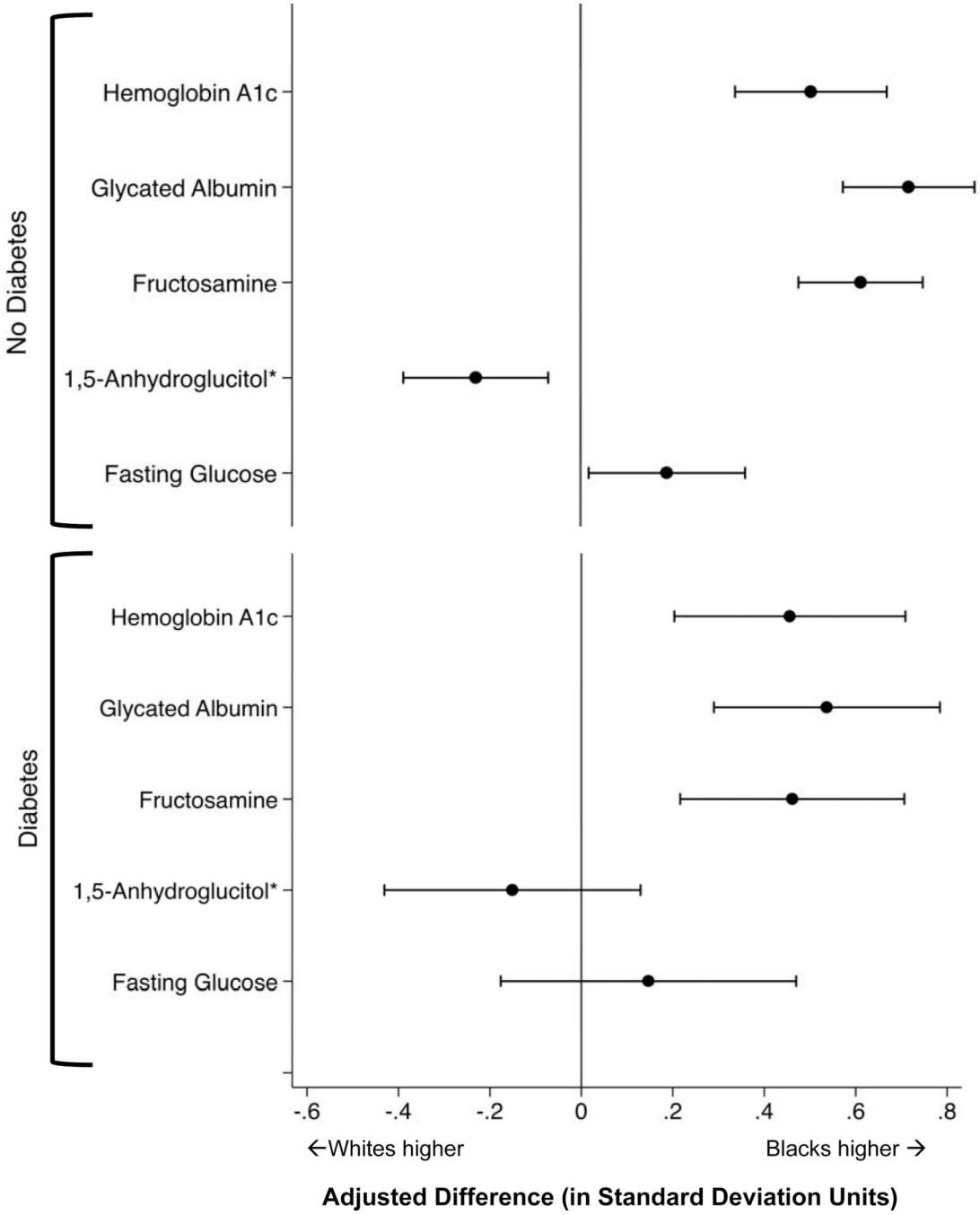

Figure.

Adjusted standardized difference in glycemic markers (95%CI), by race (black or white) in persons with and without diabetes. Adjusted for age, sex, systolic blood pressure, hypertension medication use, body mass index, low-density and high-density lipoprotein cholesterol levels, cholesterol-lowering medication use, cigarette smoking, prevalent coronary heart disease, education level, log-transformed C-reactive protein, family history of diabetes, and fasting glucose level (except for the model of fasting glucose). * Serum 1,5-anhydroglucitol is inversely related to glycemia-induced glycosuria.

Table 1

Participant Characteristics Overall and by Race

Characteristic	Overall (*n* = 1719)	White Persons (*n* = 1295)	Black Persons (*n* = 424)
Mean age (±SE), *y*	70.3 ± 0.2	70.6 ± 0.2	69.0 ± 0.3
Men (±SE), %	43.2 ± 1.5	45.7 ± 1.7	33.2 ± 2.7
Mean body mass index (±SE), kg/m^2	29.0 ± 0.2	28.5 ± 0.2	31.1 ± 0.4
Mean total cholesterol level (±SE)			
mmol/L	5.00 ± 0.03	4.95 ± 0.36	5.20 ± 0.60
mg/dL	193.1 ± 1.2	191.1 ± 1.4	200.6 ± 2.3
Mean HDL-cholesterol level (±SE), mg/dL			
mmol/L	1.30 ± 0.01	1.29 ± 0.01	1.32 ± 0.01
mg/dL	50.0 ± 0.5	49.8 ± 0.5	51.0 ± 0.7
Mean systolic blood pressure (±SE), *mm Hg*	126.2 ± 0.6	125.6 ± 0.6	128.6 ± 1.2
Cholesterol-lowering medication use (±SE), %	44.2 ± 1.5	46.2 ± 1.7	36.2 ± 2.7
Blood pressure medication use (±SE), %	64.4 ± 1.5	61.2 ± 1.7	76.9 ± 2.5
Median C-reactive protein (IQR), *mg/L*	2.0 (1.0–4.4)	1.8 (0.9–3.9)	3.0 (1.3–6.8)
Smoking status (±SE), %			
Current	7.8 ± 0.8	7.1 ± 0.9	10.7 ± 1.8
Former	41.6 ± 1.5	44.1 ± 1.7	31.7 ± 2.6
Never	50.5 ± 1.5	48.8 ± 1.7	57.6 ± 2.8
Drinking status (±SE), %			
Current	51.1 ± 1.4	57.3 ± 1.6	26.5 ± 2.6
Former	28.9 ± 1.3	25.5 ± 1.5	42.5 ± 2.9
Never	20.0 ± 1.1	17.2 ± 1.3	31.0 ± 2.7
Education level (±SE), %			
< high school	14.8 ± 0.9	11.3 ± 1.0	28.4 ± 2.6
High school or equivalent	43.8 ± 1.5	47.3 ± 1.8	30.4 ± 2.7
≥ college	41.3 ± 1.5	41.4 ± 1.7	41.2 ± 2.9
Coronary heart disease (±SE), %	9.7 ± 0.8	10.7 ± 1.0	5.4 ± 1.3
Diabetes (±SE), %	17.3 ± 1.1	14.5 ± 1.2	28.5 ± 2.6
Family history of diabetes (±SE), %	23.5 ± 1.3	22.4 ± 1.4	27.8 ± 2.2

HDL = high-density lipoprotein; IQR = interquartile range.

Table 2

Mean (95% CI) of Glycemic Markers in Persons With and Without Diabetes, Overall and by Race

Variable	HbA_{1c}, %	Glycated Albumin, %	Fructosamine, *umol/L*	1,5-AG, *ug/mL*	Fasting Glucose *mg/dL*
No diabetes (*n* = 1295)	5.70 (5.66–5.74) [SD, 0.62]	13.85 (13.72–13.99) [SD, 2.06]	233.3 (231.6–235.1) [SD, 26.1]	17.40 (16.93–17.86) [SD, 6.51]	106.5 (105.1–108.0)
Diabetes (*n* = 424)	6.31 (6.16–6.46) [SD, 1.40]	16.14 (15.56–16.72) [SD, 5.25]	254.7 (248.9–260.6) [SD, 53.8]	15.60 (14.79–16.41) [SD, 7.62]	118.0 (113.8–122.1)
No diabetes					
White persons (*n* = 1081)	5.57 (5.53–5.61) [SD, 0.47]	13.40 (13.29–13.51) [SD, 1.40]	228.4 (226.9–230.0) [SD, 20.0]	18.02 (17.54–18.51) [SD, 6.15]	102.0 (100.8–103.2)
Black persons (*n* = 295)	5.93 (5.83–6.03) [SD, 0.86]	14.56 (14.30–14.82) [SD, 2.14]	238.6 (235.3–241.9) [SD, 27.2]	17.10 (16.25–17.93) [SD, 6.63]	106.9 (104.4–109.5)
P value	<0.0001	<0.0001	<0.0001	0.0612	0.0005
Diabetes					
White persons (*n* = 214)	6.45 (6.31–6.59) [SD, 0.86]	16.51 (16.01–17.00) [SD, 3.28]	262.1 (255.9–268.3) [SD, 39.7]	13.70 (12.46–14.94) [SD, 7.36]	133.4 (127.5–139.3)
Black persons (*n* = 129)	7.25 (6.86–7.64) [SD, 1.90]	20.10 (18.50–21.68) [SD, 7.84]	295.1 (279.9–310.4) [SD, 76.0]	11.85 (10.21–13.50) [SD, 8.35]	145.6 (134.7–156.5)
P value	0.0001	<0.0001	0.0001	0.0786	0.0549

Abbreviations: 1,5-AG = 1,5-anhydroglucitol; hemoglobin A_{1C} = HbA_{1C}; SD = estimated population standard deviation.

Table 3

Crude and Adjusted Differences in Glycemic Markers, by Race (Black and White)

Variable	HbA_{1c}, %	Glycated Albumin, %	Fructosamine, *umol/L*	1,5-AG, *ug/mL*	Fasting Glucose, *mg/dL*	
No diabetes					*mmol/L*	*mg/dL*
Crude, $\bar{x}_{diff}$ (±SE)	0.36 ± 0.06*	1.16 ± 0.14*	10.16 ± 1.84*	−0.93 ± 0.50		4.95 ± 1.42†
Model 1, β (±SE)‡	0.33 ± 0.05*	1.31 ± 0.14*	15.17 ± 1.69*,§	−1.65 ± 0.52†		3.19 ± 1.47//
Model 2, β (±SE)¶	0.29 ± 0.05*	1.17 ± 0.12*	13.49 ± 1.53*,§	−1.47 ± 0.52†		–
Diabetes						
Crude, $\bar{x}_{diff}$ (±SE)	0.80 ± 0.21*	3.59 ± 0.85*	33.03 ± 8.39*	−1.84 ± 1.05		12.17 ± 6.34
Model 1, β (±SE)‡	0.65 ± 0.19†	3.08 ± 0.84*	28.27 ± 9.10†,§	−1.56 ± 1.15		6.11 ± 6.86
Model 2, β (±SE)¶	0.56 ± 0.16*	2.71 ± 0.64*	24.01 ± 6.50*,§	−1.09 ± 1.04	–	

1,5-AG = 1,5-anhydroglucitol; HbA_{1C} = hemoglobin A_{1C}.

*$P < 0.001$

†$P < 0.01$

‡Model 1 is adjusted for age, sex, systolic blood pressure, hypertension medications, body mass index, low and high-density lipoprotein cholesterol levels, cholesterol-lowering medications, cigarette smoking, prevalent coronary heart disease, education level, log-transformed C-reactive protein, and family history of diabetes.

§Additionally adjusted for serum albumin.

//$P < 0.05$

¶Model 2 includes variables in Model 1 and fasting glucose concentration.

Table 4

Crude and Adjusted Differences in Standardized Glycemic Markers by Race (Black – White)

	HbA1c (per 1 SD)	Glycated albumin (per 1 SD)	Fructosamine (per 1 SD)	1,5-AG (per 1 SD)	Fasting glucose (per 1 SD)
No Diabetes					
Crude, $\bar{x}_{diff}$ (SE)	0.63 (0.10)***	0.71 (0.09)***	0.46 (0.08)***	−0.15 (0.08)	0.29 (0.08)**
Model 1, β (SE)	0.58 (0.09)***	0.81 (0.09)***	0.69 (0.08)†***	−0.26 (0.08)**	0.19 (0.09)*
Model 2, β (SE)	0.51 (0.08)***	0.72 (0.07)***	0.62 (0.07)†***	−0.23 (0.08)**	-
Diabetes					
Crude, $\bar{x}_{diff}$ (SE)	0.65 (0.17)***	0.71 (0.17)***	0.63 (0.16)***	−0.25 (0.14)	0.29 (0.15)
Model 1, β (SE)	0.52 (0.15)***	0.61 (0.17)***	0.54 (0.17)†**	−0.22 (0.16)	0.15 (0.16)
Model 2, β (SE)	0.46 (0.13)***	0.54 (0.13)***	0.46 (0.13)†***	−0.15 (0.14)	-

Model 1: Adjusted for age, sex, systolic blood pressure, hypertension medications, body mass index, LDL- and HDL-cholesterol, cholesterol-lowering medications, cigarette smoking, prevalent coronary heart disease, education level, log-transformed C-reactive protein, and family history of diabetes.

Model 2: Variables in Model 1 + fasting glucose.

*P<0.05

**P<0.01

***P<0.001

†Additionally adjusted for serum albumin.