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Self-Reported Discrimination, Diabetes Distress, and Continuous Blood Glucose in Women with Type 2 Diabetes

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Abstract

We investigated whether self-reported racial discrimination was associated with continuous glucose levels and variability in individuals with diabetes, and whether diabetes distress mediated these associations. Seventy-four Black and White women with type 2 diabetes completed the Experience of Discrimination scale, a measure of lifetime racial discrimination, and the Problem Areas in Diabetes, a measure of diabetes distress. Participants wore a continuous glucose monitor for 24 h after 8 h of fasting, a standard meal, and a 4-h run in period. Higher discrimination predicted higher continuous mean glucose and higher standard deviation of glucose. For both mean and standard deviation of glucose, a race × discrimination interaction indicated a stronger relationship between discrimination and glucose for Whites than for Blacks. Diabetes distress mediated the discrimination–mean glucose relationship. Whites who report discrimination may be uniquely sensitive to distress. These preliminary findings suggest that racial discrimination adversely affects glucose control in women with diabetes, and does so indirectly through diabetes distress. Diabetes distress may be an important therapeutic target to reduce the ill effects of racial discrimination in persons with diabetes.

Keywords

Diabetes; Glucose; Women; Discrimination; Racism; Diabetes distress

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Introduction

A growing literature documents associations between exposure to racial discrimination and stress-related cardio metabolic conditions [1] such as central adiposity [2], cardiovascular function [3], and inflammation [4]. Racial discrimination may act as an environmental stressor that elicits mental, behavioral, and/or biological stress reactions. In the setting of diabetes, such stress reactions can affect glucose [5]. Yet, evidence linking discrimination to glucose is limited [6], and the putative mechanisms have not been elucidated. This study investigated the relationship between racial discrimination and glucose in persons with type 2 diabetes.

Exposure to discrimination is associated with distress [7], low levels of psychological resources [8], and health risk behaviors [9]. In these ways, racial distress may influence diabetes distress (DD) among persons with diabetes. DD is characterized by frustration, fatigue, low motivation for self-care, and suboptimal diabetes planning, problem solving, and adherence [10]. DD is associated with worse glucose control in cross-sectional and time-concordant longitudinal studies [10]. Thus, DD could be one mechanism through which discrimination impairs glucose.

In the United States, racial discrimination is most commonly reported by people of color, and African Americans in particular [11]. Data suggest that Whites also report lifetime racial discrimination, albeit at much lower rates [11]. When Whites do report racial discrimination, it appears to be equally deleterious for their health across a range of outcomes. For example, Hausmann and colleagues [12] examined self-reported discrimination in health care among White, African American, and Latino patients in the 2004 Behavioral Risk Factor Surveillance System Survey (BRFSS). Perceived discrimination was associated with worse health status for the overall sample and stratified analyses revealed that this relationship held for Whites, specifically. Fujishiro [13] examined reports of racial privilege in the workplace in the 2004 BRFSS. Reports of being treated worse than other races in the workplace were associated with poor health for all racial groups, including Whites. Our own work has shown that self-reported racial discrimination is related to worse cardiovascular function, even in a primarily White sample [14]. Thus, racial discrimination may be best studied in a multi-ethnic sample for whom exposure to racist events would be assumed to vary widely.

Studies examining glucose control have traditionally relied on glycosylated hemoglobin (A1c) or intermittent self-monitoring of blood glucose with finger pricks. While well accepted and very useful, these measures have several limitations. First, A1c provides information on central tendency of glucose, but provides no information regarding glucose variability. Although controversial, data suggest that glucose variability plays a role in the development of long-term vascular complications of diabetes independent of mean glucose [15]. Second, self-monitoring of blood glucose yields data only when the patient performs a finger prick. Therefore, even perfect compliance with a strict regimen will only yield, for example, 4 glucose levels per day, and compliance with such regimens is difficult to achieve.

In the last decade, the advent of continuous glucose monitoring (CGM) has provided a new, complimentary approach to characterizing glucose control [16]. The most important benefits relate to the automatic and nearly continuous (i.e., every 5 min) measurement of glucose. This method improves reliability through frequent measurements, decreases reliance on patient compliance, and yields indices of glycemic variability not afforded by A1c or intermittent self-monitoring.

Continuous glucose monitoring systems collect and store glucose data in an ongoing fashion for several days at a time. All of the currently available CGM devices are minimally invasive, and consist of a sensor probe that passes through the skin into the subcutaneous tissue, usually in the abdomen. The sensor detects glucose in the interstitial fluid and transmits the glucose data to a receiver. CGM systems can be configured in a “real-time” mode with the receiver showing continuously updated glucose measurements with direction and rate-of-change information directly to the patient, or a “blind” mode that records the continuous glucose data but does not display the glucose measurements to the patient. In both modes, the data stored in the receiver can be downloaded for review and analysis at a later time. Several CGM systems are approved by the Food and Drug Administration for clinical use, and CGM is generally safe because the sensors are inert [17]. CGM is being used in behavioral research to evaluate the effects of behavioral interventions on diabetes control, as a behavior modification and teaching tool in diabetes self-management interventions, and to investigate basic bio-behavioral processes [16]. We employed CGM in this study for the latter purpose.

This study investigated the relationship between self-reported exposure to racial discrimination and continuous glucose levels and variability among Blacks and Whites, and tested whether DD mediated this relationship. We hypothesized that greater exposure to racial discrimination would be associated with higher levels of DD that would, in turn, predict higher mean glucose and higher glucose variability. This study investigated women, who experience greater racial stress [18] and DD [19] than do men, and employed a multi-racial sample in order to maximize variability in exposure to racial discrimination.

Methods

Recruitment

Inclusion criteria for Black women were: having two parents of African descent, being born and raised in the US, and identifying as Black or African American. For Whites: two parents of European descent, born and raised in the US, and identifying as White, Caucasian, or European American. Women were excluded if they self-identified as Hispanic, or if they had: known or suspected (e.g., angina) coronary artery disease, acute medical or psychiatric problems; drug or alcohol use disorder; or lack of reliable transportation to the study site. All were naïve to continuous glucose monitoring. Participants were recruited from newspaper and radio advertisements, and state employee paycheck inserts.

Procedures

Participants fasted for 8 h before a morning laboratory session at the University of Connecticut clinical research center where a research nurse collected data. After providing informed consent, participants were instrumented with a continuous glucose monitor, to which they were blinded, and that was calibrated by a research nurse. Participants consumed a standardized breakfast, which consisted of a prepared nutritional drink and then completed psychosocial questionnaires.

Participants were instructed to engage in typical activities over the next 24 h, maintaining routine physical activity and eating patterns. During that time, participants completed paper and pencil logs regarding their degree of adherence to dosing, timing, and adjustment of diabetes medications on a 5-point scale (0 = “not at all” to 4 = “definitely”). On the same 5-point scale they also rated the degree to which they adhered to healthy food choices and portions. They also provided a written qualitative log of physical activity which was then coded by study staff as sedentary (e.g., watching TV), light (e.g., light housekeeping), or moderate (e.g., walking). They then returned several days later to return the equipment and receive $125 compensation.

These data were collected as part of a study during which participants wore the CGM for several days and experimental manipulation (mental stressor exposure) was performed on some days. Data from the first complete 24-h period *without* experimental manipulation are reported here. Procedures were approved by the UConn Health Center Institutional Review Board.

Measures

The predictor, racial discrimination, was assessed with the 9-item Experiences of Discrimination scale (EOD; [20]). Items concern the frequency (0 = “never” to 5 = “four or more times”) of ever having experienced discrimination because of “race, ethnicity, or color” in specified situations such as “at school” and “getting service in a store or restaurant”, with a total score based on the sum of items. The EOD was designed to assess racial discrimination across race and ethnicity. In a sample of Black, White, and Hispanic subjects, psychometric analyses confirmed one underlying factor, adequate test–retest reliability, and that scores were not associated with social desirability [20]. In this study, α = .86 among Blacks, and α = .79 among Whites.

The potential mediator, DD, was measured with the Problem Areas in Diabetes scale (PAID; [21]). This measure taps diabetes distress associated with 20 common diabetes problems such as frustration with failure to meet treatment goals and preoccupation with food. In this study, among Blacks, α = .94, and among Whites, α = .95.

The outcome, CG, was measured with the MiniMed CGM Gold (Medtronic Diabetes, Northridge, California). After a 4 h run-in period, the following 24 h of glucose recording was used for analysis. MiniMed CGM Gold obtains 288 glucose readings per day. Residing inside a permeable membrane, a subcutaneous electrode sends interstitial glucose measurements to a monitor every 10 s. The MiniMed CGM Gold system uses a blind mode in which the minute to minute glucose levels are stored but not displayed, so there is no

reactivity. Twice per day, the system beeps to remind the participant to calibrate the system using a standard glucometer which was provided to the participant. Studies support the system's reliability [22– 24].

MiniMed CGM Gold software reports mean and standard deviation of glucose for each 24 h period of recording. It also provides the percent time above, within, and below target range, which was set a priori at 70–140 mg/dl.

Potential confounders were tested for consideration as model covariates: age, educational attainment, body mass index, diabetes duration, and insulin use, as well as psychological variables associated with reporting discrimination, namely, depressive symptoms and hostility. Depressive symptoms were measured with the Center for Epidemiological Studies Depression scale [25]. For our study, $\alpha = .90$ among Blacks and $\alpha = .86$ among Whites. Depressive symptoms negatively influence recall of past events [26]. Trait hostility was measured with the hostility subscale of the Buss and Perry Aggression Questionnaire [27]. In this sample, $\alpha = .85$ among Blacks, and $\alpha = .86$ among Whites. High scores are related to hostile submissiveness to mistreatment [28] and anger in response to provocation [29].

To avoid the risk of overfitting models in this relatively small sample, we chose covariates separately for mean CG and standard deviation CG. If the covariate reduced the regression coefficient of discrimination or if the covariate was itself a predictor of glucose at $p < .10$, it was retained. If a covariate did not reduce the coefficient of discrimination and it was not a predictor of CG, then it was removed.

For mean CG, educational attainment and insulin use met these criteria and were included in the final model. For standard deviation CG, age and insulin use met criteria for inclusion as covariates and were retained in an adjusted model.

Data Analysis

The aim of the study was to determine if there is a relationship between perceived racism and continuous mean blood glucose. We powered the study to be able to detect a medium size correlation ($r = 0.3$) with power of 0.8 using a two-sided alpha level of 0.05. This resulted in an estimated sample size of 80 participants.

Multiple linear regressions were used to estimate the relationship between racial discrimination and mean CG with SPSS v19.0. If the adjusted linear regression showed a significant relationship between discrimination and continuous glucose, then a test of indirect effects with accelerated, bias corrected, bootstrapped (5,000 cases) estimation [30] was conducted to examine whether DD mediated the discrimination effect on glucose.

Results

Sample

See Table 1 for descriptive statistics. Most participants (91.9 %) had a high school education or greater, and 42.9 % were married. Compared to Whites, Blacks reported more racial discrimination (M = 13.6, SD = 9.4 vs. M = 1.6, SD = 3.3), were younger (M = 51.8, SD =

9.8 vs. M = 59.4, SD = 12.6), had higher BMI (M = 38.9, SD = 10.2 vs. M = 33.4, SD = 6.8), and greater DD (M = 58.4, SD = 25.2 vs. M = 46.9, SD = 21.4), all $p < .05$.

The majority of participants (55.8 %) were taking oral hypoglycemic agents only and 31.1 % used insulin as part of their regimen. On average, participants spent 46 % time above, 52 % time within, and 2 % time below the glucose target range. Blacks had marginally higher mean CG (155.4 vs. 139.0), $p = .07$. There were no significant racial differences for standard deviation CG, $p = .7$.

Overall, diary data revealed good medication adherence, poor nutrition, and predominantly sedentary behavior on the day of CGM. All participants reported adequate same-day medication dosing (M = 3.8, SD = 0.53, range = 2–4). Ratings were lower for making healthy food choices (M = 2.6, SD = 1.1, range = 0–4). For physical activity, 2 % reported walking, 32 % light activity, and 67 %, reported only sedentary activities. There were no racial differences on diary data.

Predicting Mean CG

In an unadjusted model, there was a main effect for discrimination on mean CG; higher discrimination predicted higher mean CG, beta = 0.24, $F(1,73) = 4.48$, $p < .05$. Education and insulin met criteria for inclusion as covari-ates, $p < .10$, and were therefore retained in an adjusted model. With these covariates included, the discrimination effect on mean CG remained significant, beta = 0.25, $p < .05$.

There was a marginally significant discrimination × race interaction on mean CG, $p = .05$, indicating a stronger relationship between discrimination and mean CG for Whites than Blacks. In subgroup analysis, discrimination did not predict mean CG glucose among Blacks, beta = 0.08, $p = .65$, but did among Whites, beta = 0.40, $p < .05$, even after controlling for education and insulin, beta = 0.36, $p < .05$.

Mediation results indicated a significant indirect effect of discrimination on mean CG through DD (indirect = .09, 95 % CI = .03–.23, Fig. 1). The mediation effect was not significantly moderated by race; however the parameter from DD to mean CG was approximately twice as large for Whites as for Blacks.

Predicting Standard Deviation CG

In an unadjusted model, there was a main effect for discrimination on standard deviation CG. Higher discrimination predicted higher standard deviation CG, beta = 0.29, $F(1, 73) = 6.70$, $p < .05$. Age and insulin use met criteria for inclusion as covariates, $p < .10$, and were therefore retained in an adjusted model. With these covariates included, the discrimination effect on standard deviation CG became nonsignificant, beta = .16, $p = .14$. Because the main effect became nonsignificant with covariate adjustment, we did not test for mediation by DD.

There was a discrimination × race interaction, $F(3,71) = 6.30$, $p < .05$, indicating a stronger relationship between discrimination and standard deviation CG for Whites than for Blacks. In subgroup analysis, there was a trend for discrimination to predict standard deviation CG

glucose among Blacks, beta = 0.31, $p = .06$ which remained a trend after controlling for age and insulin, beta = 0.24, p = .09. The effect for discrimination was significant among Whites, beta = 0.59, $p < .05$, and remained significant after controlling for age and insulin, beta = 0.39, $p < .01$.

Discussion

The main finding of this study is that self-reported racial discrimination was associated with continuous glucose in women with type 2 diabetes. Specifically, higher discrimination was associated with higher mean CG in White women, and with higher CG variability in both Black and White women with type 2 diabetes. To our knowledge, this is the first study linking racial discrimination to glycemic control among individuals with diabetes. Results from unadjusted analyses suggest that individuals who report more discrimination have higher glucose, and they also have more labile, i.e., harder to control, glucose. We also found, predictably, that insulin was strongly associated with CG because it is typically prescribed for those patients whose hyperglycemia can no longer be controlled by oral agents. Thus, in our analyses, the strong effect for insulin overshadowed the effect of discrimination on glucose variability.

Our second finding is that, in general, some of the effects of discrimination on glucose crossed racial lines. Whereas early research on the health effects of discrimination focused exclusively on Blacks, more recent literature has documented that the effects of unfair treatment are evident in other racial and ethnic groups [3, 12–14]. In the US, Blacks virtually always report higher levels of discrimination than other racial and ethnic groups do, but the *relationship* between discrimination and outcomes is not exclusive to Blacks.

Moreover, the effect of discrimination on mean CG was observed only among Whites. Although counter-intuitive, similar findings have been previously reported for health outcomes other than glucose. For example, Ayalon et al. [31] observed a stronger relationship between discrimination and mental health for Whites than Blacks. Similarly, Barnes et al. [32] found that participants in the Chicago Health and Aging Project who reported more perceived discrimination had a higher relative risk of death (hazard ratio = 1.05) and that this association was stronger among Whites than Blacks. Some have interpreted this pattern of relationships as meaning that Blacks may be more “used to” discrimination than Whites, and therefore find it less stressful. We do not concur with this interpretation. Alternatively, we suggest that Whites who report discrimination may be unique individuals who are sensitive to noxious stimuli, vulnerable to distress, and susceptible to its effects on diabetes self-care. The meaning of reports of racial discrimination by Whites, and the experiences of social distancing that are viewed as discriminatory by them, require further investigation.

Our third main finding is that DD mediated the association between discrimination on the one hand, and mean glucose on the other hand. Recent data suggest that the diabetes regimen per se may generate more distress than the emotional burden of living with diabetes [33]. DD is related to glycemic control inasmuch as it reflects compromised mastery, self-efficacy, problem solving, adherence, self-control, and motivation [10]. Discrimination is

negatively associated with many of these factors [8, 11, 34, 35]. Our diary data suggest that unhealthy food choices and medication adjustments may be particular self-care behaviors important for CG in this adult, sedentary, type 2 population. We and others have reported associations between discrimination and appetitive behaviors, including unhealthy food choices [36], overeating [37], and increased risk taking [35]. The finding that the relationship between DD and mean CG was robust across race may be particularly important to Black women who reported higher DD than did White women.

The observed relationship between higher DD and suboptimal glycemic control is consistent with other reports [10]. Importantly, DD is modifiable. In the REDEEM trial, Fisher and colleagues [33] tested computer assisted interventions to reduce DD. The interventions significantly reduced DD, and reductions in DD were accompanied by significant improvements in healthy eating, physical activity, and medication adherence, although not in A1c. Both the REDEEM [33] and DiaMIND [38] trials found that participants with the highest DD at baseline benefited most from behavioral intervention. Interventions that address both DD and self-management may prove useful in reducing both DD and A1c [39].

We were surprised that depression, hostility, and body mass index did not affect the relationship between racial discrimination and CG. Depressive symptoms and hostility may not have emerged because recruits were excluded if they had psychiatric or substance use comorbidities. Moreover, previous studies have found DD to be more strongly linked to glucose than is depression [10]. BMI may not have emerged because our sample of women with type 2 was almost uniformly overweight, limiting variability in BMI.

Our main findings are consistent with a growing body of literature suggesting that unfair treatment is associated with worse health outcomes [9]. Discrimination includes unfair treatment, social distancing and aggressive behaviors. Infrequent overt events (e.g., aggression, harassment), or frequent subtle events (e.g., rejection, exclusion) may both have deleterious effects [40]. Racial discrimination is one example of social inequity and unfairness that can influence the social gradient of cardiometabolic health [41].

Limitations

While novel and provocative, these findings should be interpreted with caution. First, the study employed a relatively small sample, although the sample size was on par with most behavioral studies that employ CGM [16]. Second, given the ambulatory design of this study, we did not measure counter-regulatory hormones (hormones that oppose glucose) through which exposure to stressful stimuli could directly increase glucose levels. Third, the cross sectional design is a limitation, although the time-frames of the assessments (lifetime discrimination, current diabetes distress, followed by real time glucose) strengthens causal inference. Williams [9] notes that cross-sectional designs do not address directionality between discrimination and distress, i.e., the possibility that distress or mental health symptoms can lead an individual to detect and/or report more discrimination. It is therefore noteworthy that the few prospective studies [e.g., 42] have found a positive association between perceived discrimination and subsequent changes in mental health symptoms. However, these prospective studies have investigated minority samples. One might

speculate that the directionality may differ by minority/majority status, i.e., that Whites with mental health symptoms detect discrimination, and Blacks who experience discrimination go on to develop mental health symptoms. Prospective studies are required to test this hypothesis. Fourth, discrimination was per self-report. While perceiving discrimination is quite common at some point over the lifespan, there is considerable variability in the frequency of perceiving discrimination. Although one individual may perceive discrimination in a situation that another individual might not, we suggest the effects of stress exposure are mediated through an individual's appraisal of and coping with the stressor [43]. Thus, the objective nature of the stressor is less important than the subjective experience of it. Finally, we did not match the race of the participant to the race of the staff person collecting the data, none of whom were Black.

These limitations are generally outweighed by the strengths of the study including a well characterized sample, measurement of continuous glucose, investigation of both glucose mean and glucose variability, investigation of Blacks along with a White comparator group, and careful attention to potential confounds. Future research should attempt to replicate the linkages between discrimination, DD, and glucose in a larger sample. Prospective studies that employ men and explore various types of unfair treatment are indicated.

New Contribution to the Literature

To our knowledge, this is the first study linking racial discrimination to glucose control and variability. If these preliminary findings are supported, they may be clinically important. Diabetes distress may be an important therapeutic target to reduce the ill effects of racial discrimination in persons with diabetes.

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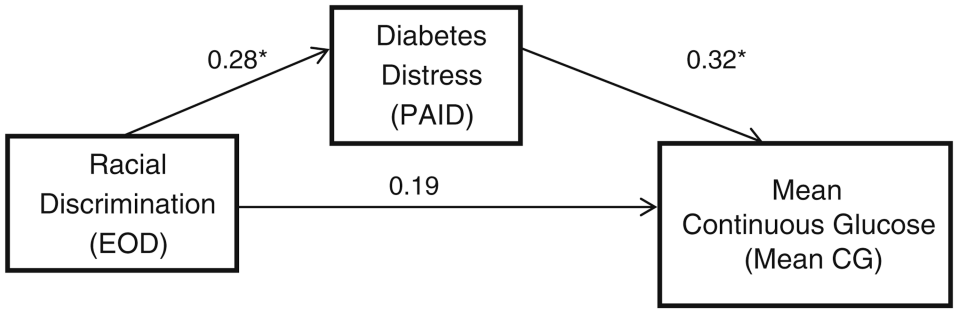

Fig. 1.

Standardized path diagram: diabetes distress mediates the relationship between racial discrimination and mean of continuous glucose. * $p < 0.05$; EOD experiences of discrimination scale; *PAID* problem areas in diabetes scale; *CG* continuous glucose

Table 1

Sample characteristics

	Black N = 37 %/M (SD)	White N = 37 %/M (SD)	*p*	Total N = 74 %/M (SD)
Age	52.54 (10.01)	59.21 (12.40)	.01	55.83 (11.67)
% Education > 12 years	13.5	2.7	.09	8.1
Diabetes duration (years)	11.46 (9.37)	5.97 (6.01)	<.01	8.72 (8.29)
CESD	11.25 (9.46)	12.73 (9.64)	.51	12.00 (9.51)
PAID	58.43 (25.19)	46.76 (21.38)	<.05	52.59 (23.93)
Hostility	17.81 (6.53)	18.08 (7.33)	.87	18.00 (6.89)
BMI	38.78 (10.22)	33.38 (6.80)	<.05	36.12 (9.06)
EOD	13.62 (9.38)	1.55 (3.29)	<.01	7.59 (9.25)
Mean 24 h–CG	155.36 (41.46)	139.01 (35.80)	.07	147.18 (39.34)
SD 24 h–CG	33.55 (24.01)	31.74 (19.21)	.71	32.64 (21.62)
% Time above target	51.87 (31.18)	39.71 (30.00)	.10	45.80 (31.99)
% Time within target	46.21 (32.91)	58.50 (29.81)	.09	52.36 (32.00)
% Below target	1.78 (6.75)	1.53 (5.67)	.90	1.65 (6.12)