Racial Discrimination and Metabolic Control in Women with Type 2 Diabetes

Julie A. Wagner, PhD; Howard Tennen, PhD; Richard Feinn, PhD; Patrick H. Finan, PhD

Purpose: We investigated whether self-reported racial discrimination was associated with insulin resistance (IR) and glycosylated hemoglobin (A1c) in women with type 2 diabetes in the United States, after controlling for covariates.

Methods: Seventy-seven Black and White women with type 2 diabetes completed the Experiences of Discrimination Scale, which assesses self-reported lifetime frequency of racially motivated discrimination. Participants provided fasting blood samples for assessment of glucose and insulin for determination of IR and A1c. Covariates included age, education, waist circumference, diabetes distress, and stressful life events.

Results: In unadjusted regression analysis discrimination was significantly associated with IR. There was a trend for a race by discrimination interaction, with a weaker effect for Blacks than Whites. Follow up analysis showed that discrimination was significantly associated with IR in both Blacks and Whites, even after adjustment, as was waist circumference. In unadjusted regression analysis, discrimination was significantly associated with A1c. There was a significant race by discrimination interaction. Follow up analysis showed that discrimination was not significantly associated with A1c among Blacks, but was among Whites, even after adjustment, as was diabetes distress and insulin use.

Conclusions: Racial discrimination is associated with insulin resistance in Black and White women with diabetes, and with A1c in White women with diabetes. (*Ethn Dis*. 2013;23[4]: 421–427)

Key Words: Discrimination, Racism, Diabetes, Glycemic Control, Women

From University of Connecticut Health Center (JAW, HT); and Quinnipiac University School of Medicine (RF); and Johns Hopkins University School of Medicine (PHF).

Address correspondence to Julie Wagner, PhD; Division of Behavioral Sciences and Community Health MC3910; University of Connecticut Health Center; 263 Farmington Ave; Farmington, CT 06410; 860.679.4508; 860.679.1342 (fax); juwagner@uchc.edu

Introduction

As the United States becomes ever more diverse, health researchers are increasingly interested in the effects of racial discrimination on health. Self-reported exposure to racial discrimination is linked to a variety of stress related disorders.[1] Exposure to racial discrimination can be stressful, and repeated exposures may confer cumulative stress effects. Although data are not entirely consistent, laboratory[2] and cross-sectional[3] studies have found an association between stress and impaired glucose regulation. Prospective studies of persons with diabetes show that major stressful life events[4] and daily hassles[5] predict worse glycemic control. Thus, exposure to racial discrimination could be a novel risk factor for insulin resistance and/or hyperglycemia. To date, evidence linking self-reported racial discrimination to metabolic control is extremely limited.[6] Our study investigated the relationship between self-reported exposure to racial discrimination and metabolic function in type 2 diabetes.

The effects of racial discrimination on metabolic control may be particularly germane for women, who bear a disproportionate diabetes burden. Furthermore, at a comparable frequency of racist events, women report greater distress about those events than men.[7] Because of the unique legacy of forced migration, slavery, and legally enforced segregation in the United States, early research focused exclusively on Black Americans. More recent literature, including our own,[8,9] has documented that the deleterious effects of unfair treatment may cross racial lines.[10–12] Therefore, the effects of racial discrimination may be best studied in a sample of minority participants and White comparators, for whom exposure to racial discrimination would be expected to vary widely. [11,12]

Insulin resistance (IR) reflects the body's ability to use available insulin and is strongly influenced by adiposity, particularly central adiposity.[13] Glycosylated hemoglobin (HbA1c, or A1c) is a relatively long-term measure of glycemic control, and reflects weeks to months of medical management, particularly the use of exogenous insulin. Thus, IR and A1c reflect related but distinct aspects of metabolic control. We examined associations among self-reported lifetime history of exposure to racial discrimination and IR and A1c in Black and White women with type 2 diabetes. We worked from a model in which exposure to stressful racist events activates biological changes such as elevated stress hormones, and behavioral changes such as appetitive behaviors, that can impair metabolic regulation and increase blood glucose over time. We hypothesized that higher self-reported racial discrimination would be associated with higher IR and higher A1c.

Participants and Methods

Seventy-seven participants with type 2 diabetes were recruited by advertisements.

We hypothesized that higher self-reported racial discrimination would be associated with higher insulin resistance and higher A1c.

Inclusion criteria were: being born and raised in the US; having two parents of African descent and identifying as Black or African American, or two parents of European descent and identifying as White, Caucasian, or European American. Women were excluded if they self-identified as Hispanic, or if they had: known or suspected cardiovascular disease; acute medical or psychiatric problems, or; drug or alcohol use disorder. Participants abstained from tobacco, exercise, caffeine, diabetes medications, food and beverages (except water) for 8 hours before a morning laboratory session. After providing informed consent, participants provided a fasting blood sample, completed questionnaires, and were compensated $50. Procedures were approved by the University of Connecticut Health Center Institutional Review Board.

Table 1. Participant characteristics, % or M (SD)

	Black $n = 39$	White $n = 38$	*P*	Total $N = 77$
Age	52.54 (10.01)	59.21 (12.40)	<.05	55.83 (11.67)
% education >12 yrs	64.10	73.70	.36	68.80
Waist, cm	114.08 (15.00)	103.05 (14.74)	<.01	108.57 (15.77)
Diabetes duration, yrs	11.26 (9.17)	6.11 (5.98)	<.01	8.71 (8.14)
% insulin using	38.50	23.70	.22	31.20
A1c NGSP	7.33 (1.55)	6.44 (1.36)	<.05	6.89 (1.53)
HOMA-IR	8.39 (12.95)	4.33 (3.92)	.07	6.22 (9.23)
EOD	13.63 (9.38)	1.58 (3.25)	<.01	7.52 (9.21)
PAID	58.43 (25.19)	46.69 (21.10)	<.05	52.57 (23.77)
SRRS	4.43 (3.65)	3.63 (3.37)	.33	4.03 (3.51)

NGSP, National Glycohemoglobin Standardization Program; HOMA-IR, homeostatic model assessment of insulin resistance; EOD, Experiences of Discrimination Scale; PAID, Problem Areas In Diabetes Scale; SRRS, Social Rating Readjustment Scale.

Measures

Independent Variable – Racial Discrimination

Self-reported frequency of lifetime racial discrimination was measured with the 9-item Experiences of Discrimination Scale (EOD).[14] Items address 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 (possible range 0–45). Test/re-test reliability has been established with Black, Latino, and White respondents. Factor analysis shows a single underlying construct.[14]

Dependent Variables

A1c was measured in the University of Connecticut Health Center clinical laboratory using high pressure liquid chromatography with the Synchron^{R} system by Beckman Coulter^{TM}, Inc.

Log transformed homeostatic model assessment of IR (logHOMA-IR) was calculated from fasting glucose and insulin values[15] according to the standard formula: log (fasting glucose × fasting insulin)/22.5. Fasting glucose was analyzed at the University of Connecticut Health Center clinical laboratory using the LXI R system by Beckman Coulter^{TM}. Fasting insulin was measured at the University of Connecticut Health Center clinical research center core laboratory with a kit from Siemens Medical Solutions Diagnostics.

Covariates

Self-reported demographic covariates included race, age, and education >12 years dummy coded as yes/no.

Clinical covariates included waist circumference and non-racial stressors and distress, waist circumference was measured by the study nurse by taking measurements at the midpoint between the upper iliac crest and the lower costal margin in the midaxillary line. Insulin use was per self-report and dummy coded as yes/no. Because insulin use is a clinical result of, rather than cause of, IR, we did not include it in analyses of IR. However, because of its strong effects on glycemic control, we did include it in analyses of A1c. Diabetes duration was highly correlated with age, so was not included.

Because individuals who report discrimination may also be individuals who experience stressors and distress more generally, we accounted for non-racial stressors and distress. We controlled for stressful life events that are not necessarily related to discrimination with the Social Readjustment Rating Scale by Holmes and Rahe.[16] Stressful life events in the past year that were endorsed on the checklist, such as death of a spouse or serious illness/injury, were summed for a total score. We controlled for diabetes distress with the Problem Areas in Diabetes (PAID) scale[17] which assesses 20 common diabetes problems such as not having clear and concrete goals for your diabetes care. For this study, alpha=.94 for Blacks and .95 for Whites.

Data Analysis

Multiple linear regressions were used to estimate the relation of race, discrimination, and their interaction, with IR and A1c. Where evidence of a race by discrimination interaction was detected, follow up unadjusted and adjusted analyses were conducted for Blacks and Whites separately. Analyses were conducted in SPSS v17.0.

RESULTS

Table 1 provides descriptive statistics by race, and Table 2 provides correlations. Participants were on average 56 years old, with diagnosed

Table 2. Correlations among discrimination, insulin resistance, and A1c by race

	% or M (SD)	EOD score	HOMA-IR	A1c
EOD score				
Black	13.63 (9.38)	-		
White	1.58 (3.25)	-		
Total	7.52 (9.21)	-		
HOMA-IR				
Black	8.39 (12.95)	.35[a]	-	
White	4.33 (3.92)	.42[b]	-	
Total	6.22 (9.23)	.39[b]	-	
A1c				
Black	7.33 (1.55)	.14	.22	-
White	6.44 (1.36)	.44[b]	.51[b]	-
Total	6.89 (1.53)	.31[b]	.37[b]	-

EOD, experiences of discrimination scale; logHOMA-IR=log transformed homeostatic model assessment of insulin resistance.
[a] $P<.05$.
[b] $P<.01$.

diabetes for 8 years, in good glycemic control (mean A1c = 6.9), and with approximately 1/3 using insulin. Compared to Whites, Blacks were younger, had longer diabetes duration, higher A1c, greater waist circumference, and reported more discrimination.

Insulin Resistance

In unadjusted analysis for the total sample, discrimination was significantly associated with IR [ß = .40, $t(74)$ = 3.65, $P<.001$]. With main effects for discrimination and race in the model, there was a trend for a race by discrimination interaction predicting IR, $P<.10$, indicating a marginally weaker relationship between discrimination and IR for Blacks than for Whites. (Table table 3a)

Follow up analyses examined this association in Blacks and Whites separately. In unadjusted analysis among Blacks, discrimination was significantly associated with IR [ß = .42, $t(35)$ = 2.64, $P<.05$]. With age, education, waist circumference, diabetes distress, and stressful life events added to the model, discrimination remained a significant independent predictor of IR among Blacks, $P<.05$. (Table 3b)

In unadjusted analysis among Whites, discrimination was significantly associated with IR [ß = .42, $t(37)$ = 2.80, $P<.01$]. With age, education, waist circumference, diabetes distress, and stressful life events added to the model, discrimination remained a significant independent predictor of IR among Whites, $P<.05$. (Table 3c)

Hemoglobin A1c

In unadjusted analyses for the total sample, discrimination was significantly associated with A1c [ß = .31, $t(72)$ = 2.98, $P<.01$]. With main effects for discrimination and race in the model, a race by discrimination interaction was significantly associated with A1c, $P<.05$, indicating a weaker relationship between discrimination and A1c for Blacks than for Whites. (Table 4a)

Follow up analyses examined this association in Blacks and Whites separately. In unadjusted analysis among Blacks, discrimination was not associated with A1c, $P=.26$. Therefore, no adjusted analyses were performed among Blacks. (Table 4b)

In unadjusted analysis among Whites, discrimination was significantly associated with A1c [ß = .44, $t(37)$ = 2.90, $P<.01$]. With age, education, waist circumference, diabetes distress, stressful life events, and insulin use added to the model, discrimination remained a significant independent predictor of IR among Whites, $P<.05$. (Table 4c)

DISCUSSION

The main findings of our study are that higher self-reported lifetime exposure to discrimination is associated with higher insulin resistance among Black and White women with diabetes and higher A1c among White women with diabetes. These findings add to the sparse literature to date indicating that discrimination is associated with metabolic risk.[18,19] The effect sizes we observed in multivariate models (partial eta^2 .13–. 22) were small, but are commensurate with those observed for other psychosocial influences on metabolic control. For example, a meta-analysis of the relationship between depression and insulin resistance[20] reported a pooled effect of .19, and a meta-analysis of the relationship between depression and A1c[21] reported a standard effect of .17.

The first main finding of our study is that higher self-reported racial discrimination was associated with higher insulin resistance (IR) in both Black and White women. Biologically plausible mechanisms between discrimination and IR can be postulated. Exposure to

The main findings of our study are that higher self-reported lifetime exposure to discrimination is associated with higher insulin resistance among Black and White women with diabetes and higher A1c among White women with diabetes.

Table 3a. Discrimination by race interaction predicts insulin resistance in unadjusted analysis in the total sample

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			5.89	3,69	.00	.20
Parameter Estimates						
	B	SE	Beta	*t*	*P*	Partial eta^2
Predictors						
Race	.01	.06	−.03	.17	.87	.00
Discrimination	−.02	.02	−.36	−.68	.50	.01
Race × Discrimination	.02	.01	.80	1.69	.09	.04

Table 3b. Discrimination predicts insulin resistance in adjusted analysis among Black participants

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			2.77	6,28	.03	.37
Parameter Estimates						
	B	SE	Beta	*t*	*P*	Partial eta^2
Predictor						
Discrimination	.02	.01	.45	2.78	.01	.22
Covariates						
Age	.01	.01	.14	.80	.43	.02
Education	−.03	.04	−.13	−.77	.45	.02
Waist circumference	.01	.00	.40	2.51	.02	.18
Diabetes distress	−.00	.00	−.16	−.96	.35	.03
Stressful life events	−.01	.02	−.12	−.70	.49	.02

Table 3c. Discrimination predicts insulin resistance in adjusted analysis among White participants

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			4.53	6.31	.00	.47
Parameter Estimates						
	B	SE	Beta	*t*	*p*	Partial eta^2
Predictor						
Discrimination	.04	.02	.34	2.13	.04	.13
Covariates						
Age	.00	.01	−.01	−.07	.94	.00
Education	−.02	.04	−.10	−.62	.54	.01
Waist circumference	.01	.00	.47	3.29	.00	.26
Diabetes distress	.00	.00	.14	.94	.35	.03
Stressful life events	−.00	.02	−.02	−.15	.88	.00

a racist event may activate biological and behavioral stress responses that impair metabolic regulation. Repeated or prolonged stress induces persistent elevation of counterregulatory hormones including cortisol, glucagon, adrenalin, and growth hormones[22] which increase glucose directly, and some of which can direct fat deposits viscerally.[22] Stress-related increases in central adiposity can lead to or exacerbate IR. Consistent with this hypothesis, in our sample both discrimination and waist circumference were significantly associated with IR. Thus, exposure to a stressful racist event may be an environmental insult that promotes the release of stress hormones, thereby increasing waist circumference and IR. Our cross-sectional data do not permit a temporally sound test of this potential mediation. Future studies should longitudinally investigate stress hormones and adiposity as potential mechanisms linking discrimination and IR.

Apart from biological mechanisms, behavioral mechanisms may also play a role. Racist exposures have been shown to increase risk taking[23] and appetitive behaviors[24,25] and to decrease self-control[26] which could worsen IR through medication nonadherence, poor nutritional choices, sedentary behavior, and sleep disruption.[27] Changes in health behavior following exposure to acute and chronic racial discrimination should be investigated in future studies.

A second finding of our study is that higher self-reported discrimination was associated with higher A1c, but only among White women.

The association between discrimination and A1c was attenuated, but remained significant, when insulin use was added to the model. Typically, over the natural history of type 2 diabetes, insulin is prescribed when lifestyle and oral agents fail to control A1c. Thus, although insulin lowers glucose, it may also be a marker for disease progression and worsened glycemic control. Diabetes distress was also significantly associated

Table 4a. Discrimination by race interaction predicts A1c in unadjusted analysis in the total sample

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			4.50	3,69	.01	.17
Parameter Estimates						
	B	SE	Beta	*t*	*P*	Partial eta^2
Predictors						
Race	−.39	.24	−.26	−.16	.11	.02
Discrimination	−.14	.09	−.90	−1.67	.09	.03
Race × Discrimination	.08	.04	1.06	2.22	.03	.06

Table 4b. Discrimination does not predict A1c in unadjusted analysis among Black participants[a]

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			2.81	1,34	.26	.04
Parameter Estimates						
	B	SE	Beta	*t*	*P*	Partial eta^2
Predictor						
Discrimination	.03	.03	.20	1.16	.26	.04

[a] Because the unadjusted analysis was not significant, no adjusted analyses were performed.

Table 4c. Discrimination predicts A1c in adjusted analysis among White participants

Omnibus Model						
			F	*df*	*P*	Partial eta^2
Full Model			10.72	7,30	000	.71
Parameter Estimates						
	B	SE	Beta	*t*	*P*	Partial eta^2
Predictor						
Discrimination	.11	.05	.25	2.12	.04	.13
Covariates						
Age	−.00	.01	−.02	−.13	.90	.00
Education	.01	.11	.01	.10	.90	.00
Waist circumference	−.01	.01	−.13	−1.14	.26	.04
Diabetes distress	.02	.01	.34	2.89	.01	.22
Stressful life events	−.06	.05	−.14	−1.20	.24	.05
Insulin use	2.07	.37	.65	5.58	.00	.51

with A1c in these White women. In addition to those mechanisms described above for IR, we speculate that experiencing discrimination and living with diabetes both consume psychological resources that, when depleted, impair self-care and, consequently, A1c.

Our finding for the effects of discrimination on A1c among Whites but not Blacks is counter-intuitive, yet has empirical precedent. For example, in the Chicago Health and Aging Project, 4154 older adults were followed for 4.5 years.[28] Participants reporting more perceived discrimination had a higher relative risk of death (hazard ratio=1.05) and this association was stronger among Whites than among Blacks (Whites HR=1.12; Blacks HR=1.03).

In studies of racial discrimination, Whites nearly always report some discrimination, albeit lower than Blacks. Whites may belong to other groups that experience discrimination, such as Arab, Jewish, Polish and Hispanic individuals. Whites may also report racial discrimination as the demographics become more diverse in their workplace, in their community and in the larger society. In this way, Whites may be, or may perceive themselves to be, in the minority and/or disadvantaged by their relative social position. For example, living in areas with a high percentage of Blacks is associated with lower reports of discrimination among African-Americans but higher reports among Whites.[29]

Notwithstanding national changes in demographics, studies that show a stronger effect of discrimination on health for Whites than for minorities still raise questions about the characteristics of Whites who report racial discrimination. They may be individuals who are also vulnerable to other psychosocial problems and distress more generally. We controlled for some psychosocial confounders in this regard, and the effect of discrimination persisted. The psychological attributes associated with reports of discrimination among

Whites (and indeed among Blacks) remain a legitimate research question, and until this is elucidated, firm conclusions cannot be drawn about associations between self-reported discrimination and metabolic function.

Our findings that discrimination affected IR in both Blacks and Whites, but affected A1c only in Whites require further investigation. These findings could suggest that, compared to Whites, Blacks may exhibit fewer behavioral reactions to discrimination that directly effect glucose, such as high carbohydrate nutritional choices. They may also reflect racial differences in the relationships among insulin resistance, insulin secretion, and A1c.[30]

Limitations and Future Directions

Our cross-sectional study that did not date the occurrences of discriminatory events does not address the complex and potentially bidirectional relationship between discrimination and diabetes control, nor the temporal ordering of psychological factors. For example, discrimination may elicit stress responses that impair metabolic control, or conversely, poor metabolic control may worsen mood and thereby increase negative attributions about interpersonal stimuli. Our study employed a sample from a single geographic location, with relatively well-controlled diabetes, which limits its generalizability. The sample was small, approaching the maximum acceptable ratio of variables to observations. Because the sample was not randomly recruited, they may not generalize to the larger population of Black and White women with diabetes. Moreover, findings may not apply to men, or to other racial and ethnic groups.

We were surprised by the lack of association between racism and theoretically related variables such as education. We suspect that these effects may not have emerged because our sample was small, and participants were excluded if they had comorbidities that are more prevalent in samples with low education, such as substance abuse.

Strengths of our study include careful measurement of objective biological outcomes in a vulnerable group of diabetic women. Our study is the first to demonstrate associations between exposure to racial discrimination and poor diabetes control. Future studies should examine these associations longitudinally in order to elucidate temporal ordering. They should also employ larger, population based samples and include additional groups that experience discrimination, most notably Latinos.

CONCLUSIONS

In conclusion, in a sample of women with type 2 diabetes, self-reported racial discrimination was associated with higher insulin resistance among Blacks and Whites, and higher A1c among Whites. Future longitudinal studies should investigate behavioral and physiological reactivity to racial stress in large samples of high risk groups.

ACKNOWLEDGMENTS

Supported by grants from NIDDK 5R21DK 074468 and the University of Connecticut Health Center General Clinical Research Center (M01RR006192).

REFERENCES

1. Pascoe AE, Richman LS. Perceived discrimination and health: a meta-analytic review. *Psychol Bull*. 2009;135:531–554.
2. Esposito-Del Puente A, Lillioja S, Bogardus C, et al. Glycemic response to stress is altered in euglycemic Pima Indians. *Int J Obes Relat Metab Disord*. 1994;18(11):766–770.
3. Pyykkönen AJ, Räikkönen K, Tuomi T, Eriksson JG, Groop L, Isomaa B. Stressful life events and the metabolic syndrome: the prevalence, prediction and prevention of diabetes (PPP)-Botnia Study. *Diabetes Care*. 2010;33:378–384.
4. Helgeson VS, Escobar O, Siminerio L, Becker D. Relation of stressful life events to metabolic control among adolescents with diabetes: 5-year longitudinal study. *Health Psychol*. 2010;29:153–159.
5. Aikens JE, Mayes R. Elevated glycosylated albumin in NIDDM is a function of recent everyday environmental stress. *Diabetes Care*. 1997;20:1111–1113.
6. De Vogli R, Brunner E, Marmot MG. Unfairness and the social gradient of metabolic syndrome in the Whitehall II Study. *J Psychosom Res*. 2007;63:413–419.
7. Wagner J, Abbott G. Depression and depression care in diabetes: relationship to perceived discrimination in African Americans. *Diabetes Care*. 2007;30:364–366.
8. Wagner JA, Tennen H, Finan PH, Ghuman N, Burg MM. Self-reported racial discrimination and endothelial reactivity to acute stress in women. *Stress Health*. 2013;29(3):214–221.
9. Wagner J, Lampert R, Tennen H, Feinn R. Exposure to discrimination and heart rate variability reactivity to acute stress among women with diabetes. Under review.
10. Williams DR, John DA, Oyserman D, Sonnega J, Mohammed SA, Jackson JS. Research on discrimination and health: an exploratory study of unresolved conceptual and measurement issues. *Am J Public Health*. 2012;102(5):975–978.
11. Grandner MA, Hale L, Jackson N, Patel NP, Gooneratne NS, Troxel WM. Perceived racial discrimination as an independent predictor of sleep disturbance and daytime fatigue. *Behav Sleep Med*. 2012;10(4):235–249.
12. Hausmann LR, Jeong K, Bost JE, Ibrahim SA. Perceived discrimination in health care and health status in a racially diverse sample. *Med Care*. 2008;46,:905–914.
13. Miyazaki Y, Glass L, Triplitt C, Wajcberg E, Mandarino LJ, DeFronzo RA. Abdominal fat distribution and peripheral and hepatic insulin resistance in type 2 diabetes. *Am J Physio Endocrin Metab*. 2002;283:1135–43.
14. Krieger N, Smith K, Naishadham D, Hartman C, Barbeau EM. Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health. *Soc Sci Med*. 2005;61: 1576–1596.
15. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985;28:412–419.
16. Holmes TH, Rahe RH. The Social Readjustment Rating Scale. *J Psychosom Res*. 1967; 11(2):213–218.
17. Polonsky WH, Anderson BJ, Lohrer PA, et al. Assessment of diabetes-related distress. *Diabetes Care*. 1995;18:754–760.
18. Cunningham TJ, Seeman TE, Kawachi I, et al. Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation

in the CARDIA cohort of 4 US communities. *Soc Sci Med*. 2012;75(5):922–931.
19. Hickson DA, Lewis TT, Liu J, et al. The associations of multiple dimensions of discrimination and abdominal fat in African American adults: the Jackson Heart Study. *Ann Behav Med*. 2012;43(1):4–14.
20. Kan C, Silva N, Golden SH, et al. A systematic review and meta-analysis of the association between depression and insulin resistance. *Diabetes Care*. 2013 Feb;36(2): 480–489.
21. Lustman PJ, Anderson RJ, Freedland KE, de Groot M, Carney RM, Clouse RE. Depression and poor glycemic control: a meta-analytic review of the literature. *Diabetes Care*. 2000;23(7):934–942.
22. Surwit RS, Schneider MS, Feinglos MN. Stress and diabetes mellitus. *Diabetes Care*. 1992;15:1413–22.
23. Jamieson JP, Koslov K, Nock MK, Mendes WB. Experiencing discrimination increases risk taking. *Psychol Sci*. 2013;24(2):131–139.
24. Wagner JA, Osborne CY, Mendenhall EA, Budris LM, Belay S, Tennen HA. Beliefs about racism and health among African American women with diabetes: a qualitative study. *J Nat Med Assoc*. 2011;103:224–232.
25. Johnson P, Risica PM, Gans KM, Kirtania U, Kumanyika SK. Association of perceived racial discrimination with eating behaviors and obesity among participants of the SisterTalk study. *J Natl Black Nurses Assoc*. 2012;23(1): 34–40.
26. Gibbons FX, O'Hara RE, Stock ML, Gerrard M, Weng CY, Wills TA. The erosive effects of racism: reduced self-control mediates the relation between perceived racial discrimination and substance use in African American adolescents. *J Pers Soc Psychol*. 2012;102(5): 1089–1104.
27. Donga E, van Dijk M, van Dijk G, et al. A single night of partial sleep deprivation induces insulin resistance in multiple metabolic pathways in healthy subjects. *J Clin Endocrinol Metab*. 2010;95:2963–2968.
28. Barnes LL, de Leon CF, Lewis TT, Bienias JL, Wilson RS, Evans DA. Perceived discrimination and mortality in a population-based study of older adults. *Am J Public Health*. 2008; 98(7):1241–1247.
29. Borrell LN, Kiefe CI, Diez-Roux AV, Williams DR, Gordon-Larsen P. Racial discrimination, racial/ethnic segregation, and health behaviors in the CARDIA study. *Ethn Health*. 2013;18(3):227–243.
30. Bacha F, Gungor N, Lee S, Arslanian SA. Type 2 diabetes in youth: are there racial differences in β-cell responsiveness relative to insulin sensitivity? *Pediatr Diabetes*. 2012;13(3):259–265.

AUTHOR CONTRIBUTIONS

Design and concept of study: Wagner, Tennen
Data analysis and interpretation: Wagner, Tennen, Feinn, Finan
Manuscript draft: Wagner, Tennen, Feinn, Finan
Statistical expertise: Wagner, Feinn, Finan
Administrative: Wagner, Tennen