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"Discrimination is associated with Increased Disease Activity in African American Women with Systemic Lupus Erythematosus: A Study of the Georgians Against Lupus Cohort."

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Thesis Title: *"Discrimination is associated with Increased Disease Activity in African American Women with Systemic Lupus Erythematosus: A Study of the Georgians Against Lupus Cohort."*

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Abstract:

Discrimination in African American Women with Systemic Lupus Erythematosus is Associated with Increased Lupus Disease Activity

Background/Purpose: Systemic lupus erythematosus (SLE) is a multi-system autoimmune disease that disproportionately impacts African American (AA) women in their reproductive years. Furthermore, AA women with SLE experience greater severity of disease and accelerated declines in health, including higher rates of mortality compared to their White counterparts. Recent studies have shown that AA women are more likely to be the victims of discrimination and unfair treatment; and such experiences, as sources of psychosocial stress, can adversely impact the progression of chronic illnesses. The purpose of our study was to examine the cross-sectional association between self-reported discrimination and disease activity among AA women with validated SLE.

Methods: Participants were from the Georgians Organized Against Lupus (GOAL) cohort, a population based sample of validated SLE patients in Atlanta, Georgia. As of 12/2011, 512 individuals returned research surveys, after exclusions, 399 AA females were included in the analysis dataset. Five items were used to assess the frequency of routine experiences of unfair treatment, including instances of being treated with less courtesy and respect, and receiving poorer service. SLE activity was measured using the Systemic Lupus Erythematous Activity Questionnaire (SLAQ). Multivariable linear regression models predicting the SLAQ were specified, controlling for sociodemographic and disease-related characteristics.

Results: In bivariate analysis, the SLAQ was greater in those who reported higher compared to lower levels of discrimination (20.8 ± 9.3 vs. 16.64 ± 8.9, $p<.0001$). BMI, work status, and marital status were also significantly associated with the SLAQ in bivariate analyses, but were not significant in multivariable models. In multivariable analyses, there was a significant positive relationship between UT and SLAQ even after adjustment for demographic, socioeconomic, behavioral, and other health-related covariates. In our final model, UT ($\beta= 0.47$, 95% CI: 0.27, 0.67, $p < 0.001$), as well as ratio of household income to poverty threshold ($\beta= -1.23$, 95% CI: -2.01, -0.44, $p < 0.01$) were significantly associated with the SLAQ.

Discussion: We found a positive association between discrimination and disease activity among AA women with SLE. This finding suggests that discrimination may be a risk factor for greater severity of disease in this population. Our study points to avenues for future research on the mechanisms underlying racial disparities in SLE severity.

Introduction:

Systemic lupus erythematosus (SLE) is a multi-factorial, multisystem autoimmune disease with poorly understood pathophysiology. It is postulated that a combination of genetic susceptibilities, hormonal factors, and environmental influences lead to disease development. (Rahman A 2008) The age of SLE onset is variable, but the diagnosis is generally established in the late twenties and early thirties. (Jimenez et al. 2003). The American College of Rheumatology (ACR) SLE classification criteria are the most common guidelines for SLE diagnosis. ACR recommends that four of the following 11 clinical and immunologic criteria are met which are malar rash, discoid rash, photosensitivity, oral ulcers, arthritis, serositis/pleuritis, renal disease, neurologic disease related to SLE, anemia/thrombocytopenia, positive antibodies (anti-smith, anti-double stranded DNA, and the anti-nuclear DNA). (Hochberg et al. 1997) It has been estimated that there are approximately 14.6 to 78.5 cases of SLE per 100 000 persons in the US. (Michet 1985, Cervera 2010) True population-based estimates of prevalence are difficult to obtain due to misclassification of diagnosis and the heterogeneity of SLE phenotypes across populations.

The incidence rates of SLE vary considerably across population groups. The disease affects females approximately 9 times more often than it affects males (Rahman A 2008) Along with the striking gender differences, there are also marked racial disparities. Incidence of SLE in African American women of childbearing age is about 3 times greater than those for white women. (Peschken CA, et al 2000) Population based studies have shown that not only do African Americans have higher prevalence and incidence rates of lupus, but African Americans also carry excess SLE-related morbidity compared to non-African Americans. In particular, African-Americans compared to whites are more likely to have lupus nephritis (Cooper GS, 2002, McCarty DJ 1995), and tend to sustain earlier SLE-induced renal damage. All of these factors lead to higher SLE-specific mortality rates in African Americans compared

to Caucasians. (Ward 1990, Uribe 2003.) Based on these studies, race appears to be a significant risk factor for both the development of SLE and disease prognosis.

As race in the United States is a primarily social rather than a biologic construct, it is likely that racial disparities in SLE frequency and severity are related, at least in part, to differences in social conditions. (Feldman et al. 2003, Chae D. 2010) In the US, African American women are more likely to face social and environmental factors that could increase SLE severity. For example, geographic clusters of high SLE mortality occur in areas with high rates of poverty, and greater numbers of racial minorities, indicating that neighborhood factors are likely implicated in SLE outcomes. (Walsh 2006) At the neighborhood level, financial stressors and deprivation appear to be risk factors for SLE severity and accelerated disease progression. (Jolly 2010, Lotstein 1998, Kasitanon 2006, Alarcon 2001) Social and economic deprivation appears to exert its effects on health at both aggregate (e.g., neighborhood) and individual levels. One particular factor, the experience of interpersonal discrimination, has been associated with higher rates of chronic diseases such as hypertension, obesity and insulin resistance. (Hunte 2009, Butler 2002). Research also shows that African American women are exposed to higher levels of discrimination than Caucasian females. (Schulz 2000, Krieger 1994, Dailey 2010, Lewis 2011) Since African American women experience higher levels of discrimination, it is necessary to examine the pathways through which African American women biologically embody these experiences. One postulated pathway is that the body interprets discrimination as chronic stress. (Kreiger 1990, Williams 2008)

Psychological stress is the result of a process through which environmental demand challenges, strains, and exceeds the adaptive capacity of the individual resulting in changes that put that individual at risk for adverse health events. (Mwendwa 2011). Stress has been linked to many disease processes including, increased rates of cancer deaths, hypertension, and myocardial infarction (Khansari 2009,

Russ 2011). It is postulated that dysfunction of the hypothalamic pituitary axis, the dulling of the anti-inflammatory effects of cortisol and increased allostatic load coalesce into an increased baseline level of inflammation and increased biological vulnerability to disease in individuals exposed to chronic stress. (Geronimus, 2010) Recently, researchers have linked discrimination to the biological markers of the stress (Cohen et al. 2006, Kalinowski et al. 2004.) These biological markers include increased levels of inflammatory cytokines such as tumor necrosis factor alpha (TNF-a), and other neuroendocrine markers of high stress (Harrell et al. 2003,Geronimus 2010). Empiric research suggests that African Americans are more likely to suffer the physiologic effects of chronic exposure to this stressor. (Barnes et al. 2004; Taylor and Turner 2002: Geronimus 2010.) The biologic factors associated with chronic stress also play a role in the pathophysiology of auto immune diseases, especially SLE. For instance, Tumor Necrosis Factor-alpha, Interleukin-1 and other cytokines are released in response to chronic stress, and are found to be elevated in SLE patients (Jimenez, 2003; Aringer and Smolen 2004).

Taken together, the evidence shows that discrimination leads to increased chronic stress, which can lead to increased biological mediators of chronic disease, including SLE. In our study, we examine the association between discrimination and SLE disease activity in the Georgians Against Lupus Cohort (GOAL). We propose to measure discrimination using the Williams perceived discrimination scale, a validated five-question survey (Hunte et. al, 2009). The outcome in our study is measured by another validated instrument called the Systemic Lupus Activity Questionnaire (SLAQ) used to assess patient self-reported disease activity. (Karlson et. al., 2003) We hypothesize that individuals who experience higher levels of interpersonal discrimination will have higher disease activity scores.

Methods:

Participants:

We analyzed data collected between August and November of 2011 from the annual survey of the GOAL cohort, which is an ongoing population based study of patients with validated SLE in Atlanta, Georgia the participants for this study were recruited among cases reported to the ongoing Georgia Lupus Registry (GLR). The GLR receives information from a variety of sources including twenty hospitals, thirty-five individual, practicing rheumatologists, ten nephrologists, twenty dermatologists, and 10 commercial laboratories in the Atlanta metro area. The purpose of the GOAL cohort is to assess the physical, psychosocial, and socio-economic burden of SLE using validated instruments.

As of December 2011, there were 512 individuals participating in the GOAL cohort. Eighty six percent of these (442) were women. The survey included 429 African Americans (83 percent), 39 non-Hispanic whites (7.6 percent) 26 Hispanics (5 percent) with the remaining 4.3 percent composed of Asians, Hawaiian Pacific islanders, and persons whose race was not specified. Due to the insufficient numbers of males and non-African Americans, the current analysis is restricted to the 399 African American females participating in the GOAL cohort.

Discrimination (Main Exposure):

We assessed discrimination using a five-item version of the Williams Discrimination scale. This is a validated five item questionnaire that aims to measure routine, everyday experiences of interpersonal discrimination (Williams 1997). Respondents reported how often they (1) were treated with less courtesy, (2) received poorer service, (3) thought others viewed them as not smart, (4) observed others acting as if they were afraid of them, (5) felt threatened by or harassed by others in the last three months. The possible range of the runs from scale (1-6) where 1 and 6 points represented the highest and the lowest frequency, respectively was reverse coded and summed across the five questions (range

6-30). The Cronbach's alpha for this questionnaire is 0.88. (Williams 1997) After answering each question, respondents were asked to attribute the main reason (age, gender, race, ancestory, height, wieght or sexual orientation) why these experiences happened, and if they did not experience any events of discrimination, the the final attribution question was skipped. Since many of the respodents marked more than one reason, we did not use reasons for disccimination in any of the analyses. On exploratory analysis, the the discrimination scores were approximately normally distributed and were highly correlated (r=.56, p<.001) with the outcome.

Lupus Disease Activity (Main Outcome)

The main outcome in this study was the SLAQ score calculated based on questionnaire responses. The SLAQ is a 24-item questionnaire that collects data on 24 specific measures of disease activity such as weight loss, fatigue and fevers with responses for each item expressed on a four-point scale (from 0-no problems to 3-severe problems). The possible score values range from a minimum of zero to a maximum of seventy two. This questionnaire was validated using the physician administered Systemic Lupus Activity Measure (SLAM) with the laboratory based items on the SLAM scale removed. Analysis of correlation between SLAM and SLAQ scores produced a Pearson's coefficient of .63 (P<.0001). The SLAQ's positive predictive values for disease activity ranged from 60-90%. (Karlson et. al., 2003) The disease activity outcome was expressed as a continuous measure , since all assumptions of normality were met, and because disease activity is clinically viewed as a continuum (Rahman A 2008).

Covariates:

The body mass index (BMI) was calculated from the patient-reported height and weight during the survey initiation. BMI was included in unadjusted analysis and in the multivariable models as a continuous variable. The age of each participant was calculated using reported date of birth subtracted

from the date of the questionnaire. With respect to smoking status each participant was categorized as current smoker versus former or never smoker. Employment was defined as unemployed, part-time, or full time. Insurance status was coded as private, public, or uninsured. Education was divided into four levels: less than 12 years (no high school diploma), 12 years (finished high school), 12-15 (some college), and at least 16 years (college and beyond.) Marital status was coded as never married, previously married, or currently married. Disease duration was a continuous variable derived from the date of questionnaire minus the patient reported date of SLE diagnosis . Poverty index is a variable that compares each participant's annual household income to the poverty threshold for Georgia in 2011. The reported annual household income was divided by the income that defines the poverty threshold with a value of 1.0 representing income that is exactly at the poverty level. Poverty index was converted to a dichotomous variable coded as≤1.0 versus >1.0.

Statistical Analysis:

All continuous variables were summarized using mean, median, standard deviation as well as maximum and minimum values. The distributions of all categorical variables were assessed using frequencies and percentages. Missing values were recorded for each covariate. Study participants were categorized as experiencing high or low level of discrimination using a cutoff of greater than 13 and less than or equal to 13 on the totaled discrimination survey score. Parametric and non-parametric tests were used to compare distributions of continuous variables between the two levels of discrimination. Chi-square tests were employed for categorical data. The crude analyses involving continuous variables were conducted using correlation and partial correlation coefficients as well as visual examination of scatter plots

Multivariable analyses evaluating the relation between level of discrimination and disease activity were carried out using linear regression models with results expressed as regression coefficients and 95 %

confidence intervals. The modeling strategy followed the hierarchally well formulated approach (Kleinbaum, 2010). Interaction was assessed for all two-way interaction terms involving the main exposure variable. If the p-value of the two-way product terms was less than .05, then interaction was considered significant. Confounding was assessed using the change-in-estimate approach. If the regression coefficient was more than ten percent different in the presence of a particular covariate, then the variable was considered a confounder and remained in the model. Collinearity was assessed for all lower order terms using variance inflation factors, conditional indices, variance decomposition proportions. Influential observation analysis was conducted using jack-knife residuals as well as Cook's distance. On multivariable analysis, approximately 14.7% of observations were missing. These were handled using multiple imputation. All analyses were performed using SAS version 9.3 (SAS institute, Cary NC)

Results:

Cohort Characteristics and Bivariate Analysis:

Table 1 shows the characteristics of the entire study population by level of self-reported discrimination. Of the 399 women participating in this study, 28.6% (n=144) reported experiencing high levels of discrimination. The mean SLAQ scores (standard deviations [SD]) were 16.64 (8.87) for the low discrimination group and 20.68 (9.30) for the high discrimination group ($p<0.001$).

Women reporting higher levels of discrimination were more likely to have higher mean BMIs (BMI=31.13, SD=7.94) compared to women with lower levels of exposure (mean BMI=29.32, SD=7.67.)

Of the total cohort, 55.42 percent (n=220) were unemployed. Individuals in the high discrimination exposure group were more likely to be unemployed compared to their low exposure counterparts (66.7% vs. 52.1%, $p< .05$) The participants did not differ significantly with respect to age, insurance status, education attainment or disease duration.

Discrimination and increased Disease Activity (Multivariable Analysis)

Table 2 presents the results of the multivariable linear regression analyses examining the association between discrimination and SLE disease activity as measured by the SLAQ scores. Model 1 adjusts only for baseline characteristics and smoking status. Model 2 additionally adjusts for social and economic characteristics. Model 3 includes all covariates used in the previous two models and also controls for disease characteristics. Across all three models, there was a positive relation between discrimination and disease activity. In Model 1, the regression coefficient reflecting the association between discrimination and SLAQ score was .418 (95% CI: .23, .61). The corresponding regression coefficients (95% CIs) for Models 2 and 3 were .46 (.25, .66) and .47 (.27, .67), respectively.

There was positive relation (β=3.63, 95% CI: .87, 6.39) between smoking status and disease activity in Model 1, but this result was no longer statistically significant as more covariates were added to the analysis. Age and disease activity were inversely related in Model 1 (β =-.07 95% CI: -.14, -.006, p=.031) but the association was no longer evident in Models 2 and 3. In Model 2, women that were unemployed had significantly higher disease activity scores (β=3.04 95% CI: .04, 6.03) when compared to fully employed study participants but this association was not statistically significant after disease duration was added to the model (Model 3). There was an inverse association between poverty and disease activity with fully adjusted regression coefficient of -1.23 (95% CI: -2.10, -.44: p=.0025) in Model 3.

Discussion:

Our study provides additional evidence to support the hypothesis that social stressors, such as poverty and discrimination, are associated with negative health outcomes in African American women (Alarcon, 2001, Alarcon 1998, Liang 1991, Clark 1999, Clark 2001, Williams 2008). Consistent with other studies, our analysis showed that these social stressors were associated with more severe disease among SLE patients in particular (Walsh 2006, Jolly 2010, Lotstein 1998, Kasitanon 2006). To our knowledge, this investigation was the first to examine the association between discrimination and disease severity among African American women with SLE. . After controlling for likely confounders we observed a statistically significant half-point increase in the SLAQ score per each one-point increase in the discrimination score among African American women. The higher the SLAQ score, the more likely a patient is to need to transition to biologic therapy and to be seen in the clinic more quickly (Romero-Diaz 2011). Rheumatologists us the SLAQ score as a way to follow patients overtime and determine the need for therapeutic alterations.

In our study population, we found a clear positive association between individuals disease activity and interpersonal discrimination. Along with the finding that discrimination is associated with SLE severity our results indicate that there is an inverse relation between household income, adjusted for the number of people residing the house, and lupus activity in AA women. This finding is also consistent with the literature regarding SES and SLE (Estel 2010, Walsh 2006, Barr 2003).

The main limitation of our study, as with all cross-sectional studies, is the inability to determine the temporal order of exposure to discrimination and SLE disease activity. For this reason it is not possible to use this study result for causal inference. Future research must reproduce the reported association using other metrics of disease activity as well as using longitudinal study designs. Another limitation to

this study relates to the measurement of discrimination. After the study participants filled out the section on discrimination, they were asked to supply the principle reasons why they thought these events were directed at them. Many people provided more than one reason, which makes it difficult to separate the individual effects of discrimination based on race, age, gender, sexual orientation, or physical attribute. Thus additional analyses assessing the perceived reasons for discrimination are needed.

As with all survey-based measures there is a general question about the accuracy of self-reported measures of discrimination and disease activity. It has also been shown that there is variation among and between racial groups as to what is perceived as discrimination. (Paradies, 2006) It is important to point out, however, that discrimination was found be more likely underreported, rather than overreported, in at least in one previous study (Williams, 2003) If the underestimation is non-differential or if people with higer disease activity underestimate their discrimination, then our results would actually underestimate the association between disease activity and discrimination. In addition, it is worth noting that self reported SLE severity based on SLAQ was validated against physician assessment, which is accepted to be a good proxy for biologic disease activity. In previous literature SLAQ scores have demonstrated a mean positive predictive value of 75% for detecting clinically significant changes in disease activity. (Romero-Diaz, 2011)

The results of this study support the hypothesis that interpersonal discrimination, a psychosocial stressor, is related to increased disease activity in AA women with SLE. Additionally this study provides support to the mounting body of evidence that suggests poverty is a risk factor for SLE activity. It will be necessary for future studies to explore this association between SLE disease activity and discrimination in a few different ways. First, since empiric studies have shown that discrimination is not a static process

(Hunte, 2011: Paradies, 2006) it will be necessary to focus on the longitudinal measurement of discrimination and to use the life-course model to investigate if these events have a cumulative or threshold effect. Secondly, future research must disentangle the effects of discrimination due to different causes and examine if there is interaction involving various causes of discrimination. Third, additional studies need to examine patient-reported as well as biologic outcomes in relation to discrimination. The results of this and other studies warrant the adoption of a more complex ecosocial, multi-disciplinary research agenda that examines the physiological pathways of social stressors involving perceived discrimination and SLE.

References:

Aringer, M. and J. S. Smolen (2004). "Tumour necrosis factor and other proinflammatory cytokines in systemic lupus erythematosus: a rationale for therapeutic intervention." Lupus **13**(5): 344-347.

Bricou, O., O. Taieb, et al. (2006). "Stress and coping strategies in systemic lupus erythematosus: a review." Neuroimmunomodulation **13**(5-6): 283-293.

Jimenez, S., R. Cervera, et al. (2003). "The epidemiology of systemic lupus erythematosus." Clin Rev Allergy Immunol **25**(1): 3-12.

Kozora, E., M. C. Ellison, et al. (2005). "Major life stress, coping styles, and social support in relation to psychological distress in patients with systemic lupus erythematosus." Lupus **14**(5): 363-372.

Rahman A, I. D. (2008). "Systemic lupus erythematosus. ." New England Journal of Medicine **358**((9)): 929–939.

McCarty DJ, Manzi S, Medsger TA Jr, Ramsey-Goldman R, LaPorte RE, Kwoh CK. Incidence of systemic lupus erythematosus: Race and gender differences. Arthritis Rheum 1995; 38: 1260–1270

Cooper GS, Parks CG, Treadwell EL et al. Differences by race, sex andage in the clinical and immunologic features of recently diagnosedsystemic lupus erythematosus in the southeastern United States. Lupus 2002; 11: 161–167.

Ward MM, Studenski S. Clinical manifestations of systemic lupus erythematosus. Identification of racial and socioeconomic influences. Arch Intern Med 1990;150(4):849–53.

Uribe AG, Alarcon GS. Ethnic disparities inpatients with systemic lupus erythematosus. Curr RheumatolRep 2003;5(5):364–9.

Feldman, Marcus W., Richard C. Lewontin, and Mary-Claire King (2003). Race: A Genetic Melting-Pot. Nature, 424:374.

Chae, D. Framing Race and Health, psycho-social biological approach: 2010.

Krieger, Nancy (2008). Does Racism Harm Health? Did Child Abuse Exist Before1962? On Explicit Questions, Critical Science, and Current Controversies: An Ecosocial Perspective. *American Journal of Public Health*, 98(9 Suppl):S20-S25.

LaVeist, Thomas A. (2000). On the Study of Race, Racism, and Health: A Shift From Description to Explanation. *International Journal of Health Services*, 30(1):217-219.

Jones, Camara P. (2000). Levels of Racism: A Theoretical Framework and a Gardener's Tale. *American Journal of Public Health*, 90(8):1212-1215.

Williams DR, Lavizzo-Mourey R, Warren RC. The concept of race and health status in America. Public Health Rep. 1994 Jan-Feb;109(1):26-41.

Hunte HE, Williams DR. The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. Am J Public Health. 2009; 99(7):1285–1292.

Butler C, Tull ES, et al. Internalized racism, body fat distribution, and abnormal fasting glucose among African-Caribbean women in Dominica, West Indies. J Natl Med Assoc. 2002;94(3):143–148.

Mwendwa DT, Gholson G, Sims RC, Levy SA, Ali M, Harrell CJ, Callender CO, Campbell AL Jr. Coping with perceived racism: a significant factor in the development of obesity in African American women? J Natl Med Assoc. 2011 Jul;103(7):602-8.

Khansari, N., Shakiba, Y., & Mahmoudi, M. (2009). Chronic inflammation and oxidative stress as a major cause of age-related diseases and cancer. Recent Patents on Inflammation & Allergy Drug Discovery, 3(1), 73–8

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. New England Journal of Medicine, 338(3), 171–179

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. Annals of the New York Academy of Sciences, 896, 30–47

Russ TC, Stamatakis E, Hamer M, Starr JM, Kivimäki M, Batty GD. Association between psychological distress and mortality: individual participant pooled analysis of 10 prospective cohort studies. BMJ. 2012 Jul 31;345:e4933. doi: 10.1136/bmj.e4933.

Arline T. Geronimus & Margaret T. Hicken & Jay A. Pearson & Sarah J. Seashols & Kelly L. Brown & Tracey Dawson CruzHum Do US Black Women Experience Stress-Related Accelerated Biological Aging? A Novel Theory and First Population-Based Test of Black-White Differences in Telomere Length. Nat (2010) 21:19–38

Harrell, Jules P., Sadiki Hall, and James Taliaferro (2003). Physiologic Responses to Racism and Discrimination: An Assessment of the Evidence. *American Journal of Public Health*, 93(2):243-248.

Abdou, Calvin J. Hobel, Laura Glynn, and Curt Sandman (2008). Stress and Blood Pressure During Pregnancy: Racial Differences and Associations with Birthweight. *Psychosomatic Medicine*, 70(1):57-64.

Taylor, J., & Turner, R. J. (2002). Perceived discrimination, social stress, and depression in the transition to adulthood: Racial contrasts. Social Psychology Quarterly, 65(3), 213–225.

Barnes, L. L., Mendes De Leon, C. F., Wilson, R. S., Bienias, J. L., Bennett, D. A., & Evans, D. A. (2004). Racial differences in perceived discrimination in a community population of older blacks and whites. Journal of Aging and Health, 16(3), 315–337.

Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., & Seeman, T. (2006). Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults(CARDIA) Study. Psychosomatic Medicine, 68(1), 41–50.

Kalinowski, L., Dobrucki, I. T., & Malinski, T. (2004). Race-specific differences in endothelial function: Predisposition of African Americans to vascular diseases. Circulation, 109(21), 2511–2517.

Karlson EW, Daltroy LH, Rivest C, Ramsey-Goldman R, Wright EA, Partridge AJ, Liang MH, Fortin PR. Validation of a Systemic Lupus Activity Questionnaire (SLAQ) for population studies. Lupus. 2003;12(4):280-6.

MH Liang and PR Fortin.Validation of a systemic lupus activity questionnaire (SLAQ) for population studies. Lupus (2003) 12, 280-286

Walsh SJ, Gilchrist A. Geographical clustering of mortality from systemic lupus erythematosus in the United States: contributions of poverty, Hispanic ethnicity and solar radiation. Lupus. 2006;15(10):662-70.

Barr RG, Seliger S, Appel GB, Zuniga R, D'Agati V, Salmon J, Radhakrishnan J. Prognosis in proliferative lupus nephritis: the role of socio-economic status and race/ethnicity. Nephrol Dial Transplant. 2003;18(10):2039-46.

Jolly M, Mikolaitis RA, Shakoor N, Fogg LF, Block JA. Education, zip code-based annualized household income, and health outcomes in patients with systemic lupus erythematosus. J Rheumatol. 2010;37(6):1150-7.

Lotstein DS, Ward MM, Bush TM, Lambert RE, van Vollenhoven R, Neuwelt CM. Socioeconomic status and health in women with systemic lupus erythematosus. J Rheumatol. 1998;25(9):1720-9.

Kasitanon N, Magder LS, Petri M. Predictors of survival in systemic lupus erythematosus. Medicine (Baltimore). 2006;85(3):147-56.

Alarcon GS, McGwin G Jr, Bastian HM, Boseman J, Lisse J, Fessler BJ, Friedman AW, Reveille JD. Systemic lupus erythematosus in three ethnic groups. VII [correction of VIII]. Predictors of early mortality in the LUMINA cohort. LUMINA Study Group. Arthritis Rheum. 2001;45(2):191-202.

Liang MH, Partridge AJ, Baltroy LH, Straaton KV, Galper SR, Holman HR. Strategies for reducing excess morbidity and mortality in blacks with systemic lupus erythematosus. Arthritis Rheum. 1991;34(9):1187-96.

Alarcon GS, Friedman AW, Straaton KV, Moulds JM, Lisse J, Bastian HM, McGwin G Jr, Bartolucci AA, Roseman JM, Reveille JD. Systemic lupus erythematosus in three ethnic groups: III. A comparison of characteristics early in the natural history of the LUMINA cohort. LUpus in MInority populations: NAture vs. Nurture. Lupus.1999;8(3):197-209.

Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. J Behav Med. 2009;32(1):20-47.

Krieger N. Racial and gender discrimination: risk factors for high blood pressure? Soc Sci Med. 1990;30(12):1273-81.

Walsh SJ, Gilchrist A. Geographical clustering of mortality from systemic lupus erythematosus in the United States: contributions of poverty, Hispanic ethnicity and solar radiation. Lupus. 2006;15(10):662-70.

Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans: a biopsychosocial model. Am Psychologist. 1999;54(10):805-16.

Clark VR. The perilous effects of racism on blacks. Ethn Dis. 2001;11(4):769-72.

Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. Am J Public Health. 2008;98(9 Suppl):S29-37.

Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. J Behav Med. 2009;32(1):20-47.

Pons-Estel GJ, Alarcón GS, Scofield L, Reinlib L, Cooper GS. Understanding the epidemiology and progression of systemic lupus erythematosus. Semin Arthritis Rheum. 2010;39(4):257-68.

TABLE 1-Characteristics of the AA women in the GOAL cohort, Atlanta, GA, 2011 total and dichotomized by high versus low discrimination exposure

	Total Cohort	High Level of Discrimination	Low Level of Discrimination
Characteristic	n=399	n=114	n=285
[a] ***SLAQ score****	17.79 (9.17)	20.68 (9.30)	16.64 (8.87)
Age	48.45 (13.14)	48.30 (12.51)	48.51 (13.42)
[b] ***BMI****	29.78 (7.79)	31.13 (7.94)	29.32 (7.67)
Current Smoker** n (%)			
Yes	350 (88.38)	92 (81.42)	258 (91.17)
No	46 (11.62)	21 (18.58)	25 (8.83)
[c] ***Work Status* n (%)***			
Full time	90 (22.67)	25 (22.12)	65 (22.89)
Part time	87 (21.91)	16 (14.16)	71 (25)
Unemployed	220 (55.42)	72 (63.72)	148 (52.11)
[d] ***Education Level n (%)***			
< High School	43 (10.89)	18 (15.93)	25 (8.87)
High School	124 (31.39)	27 (23.89)	97 (34.4)
Some College	182 (46.08)	54 (47.79)	128 (45.39)
College graduate	46 (11.65)	14 (12.39)	32 (11.35)
Insurance status, n (%)			
Public	207 (53.77)	58 (53.21)	149 (53.99)
Private	71(18.44)	25 (22.94)	46 (16.67)
Uninsured	107(27.79)	26(23.85)	81 (29.35)
[e] ***Poverty Index (n, %)***			
Above one	171(48.72)	44(42.31)	127(51.42)
Less or equal to one	180(51.28)	60(57.69)	120(48.58)
Marital Status			
Never Married	141 (35.34)	41 (35.96)	100 (35.09)
Currently Married	122 (30.58)	26 (22.81)	96 (33.68)
Previously Married	136 (34.09)	47 (41.23)	89 (31.23)
[f] ***Disease Duration***	15.39 (9.70)	15.12 (9.34)	15.5 (9.86)

*p<.05 **P<.025, ***P<.005,* ***p<.0001. All categorical variables reported as Mean (SD).

[a] Continuous variable as measure by the Systemic Lupus Activity Questionnaire.

[b] Continuous variable equaled to Weight (kg) divided by height (cm) squared.

[c] Students and retirees in classified as part-time.

[d] Categorized by less than 12 years, 12 years, 13-15 years, greater than 16 years of schooling.

[e] Median income divided by US census 2011 poverty level based on family unit and children under 18

[f] Continuous variable representing years since diagnosis

TABLE 2-Multivariable models examining the relationship between discrimination and SLE disease activity.

Model 1 (Adjusted for baseline characteristics)	**Regression coefficient**	**95% CI**	
Discrimination Score[a]	0.41881 Ω	0.22952	0.6081
Age (Years)	-0.07205*	-0.1383	-0.0057
BMI [b]	0.05696	-0.0557	0.1697
Current Smoking Status	3.62971**	0.86873	6.39069
Model 2 (Adjusted for socioeconomic covariates)	**Regression coefficient**	**95% CI**	
Discrimination Score[a]	0.4565 Ω	0.25406	0.65894
Age (Years)	-0.065	-0.1505	0.02053
BMI [b]	0.0643	-0.0577	0.18636
Current Smoking Status	2.94238	-0.1571	6.0419
Part time	1.99115	-1.1129	5.09526
Unemployed	3.03523*	0.03858	6.03188
Less than Highschool education	-0.35635	-4.6715	3.95887
High School education	2.82858	-0.5612	6.21844
Some College	2.05344	-1.0524	5.1593
Public Insurance	-0.00436	-2.8020	2.79333
Uninsured	0.62284	-2.5121	3.75787
Poverty Index[e]	2.54139**	0.23828	4.8445
Never Married	-1.39155	-3.9615	1.17847
Previously Married	-0.2337	-2.6660	2.19864
Model 3 (Adjusted for Disease Duration)	**Regression coefficient**	**95% CI**	
Discrimination Score[a]	0.47069 Ω	0.2686	0.67279
Age	-0.04111	-0.1362	0.05407
BMI [b]	0.05156	-0.0703	0.17347
Current Smoking Status	2.62722	-0.4701	5.72455
Part time[g]	1.40417	-1.7362	4.54464
Unemployed[g]	2.51961	-0.4932	5.53246
Less than High school education[h]	-1.40957	-5.8145	2.99535
High School[h]	1.56935	-1.9771	5.1158
Some College[h]	1.16149	-2.0160	4.33904
Public Insurance[i]	-0.5545	-3.3820	2.27305
Uninsured[i]	0.13347	-3.0167	3.28369
Poverty Index[e]	-1.22505***	-2.0146	-0.4354
Never Married[k]	-1.43535	-3.9862	1.11554
Previously Married[k]	-0.31624	-2.7244	2.09191
Disease Duration[k]	-0.01607	-0.1224	0.0903

[a] Continuous variable as measure by the Systemic Lupus Activity Questionnaire.

[b] Continuous variable equaled to Weight (kg) divided by height (cm) squared.

[c] Students and retirees in classified as part-time.
[d] Categorized by less than 12 years, 12 years, 13-15 years, greater than 16 years of schooling.
[e] Median income divided by US census 2011 poverty level based on family unit and children under 18
[f] Continuous variable representing years since diagnosis
[g] Reference=fully employed
[h] Reference=completed >16 years school
[I] Reference=private insurance
[k] Reference=currently married
*p<.05 **P<.025, ***P<.005, [Ω] p<.0001.