

A Longitudinal Investigation of Psychosocial
Mediators of Allostatic Load in a Multi-Ethnic Sample of Midlife Women:
Findings from the Study of Women's Health Across the Nation

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ABSTRACT

Objective: The focus of this research was to assess racial and socioeconomic status (SES) differences in level and change in allostatic load (AL) over a 7 year time period and to test a set of predictive pathways of mediating psychosocial variables. Specifically, we examined the contributions of discrimination, perceived stress and hostility on level and change in AL and estimated the extent to which they explain racial and SES differences in a multi-ethnic sample of midlife women.

Methods: Longitudinal data obtained from the Study of Women's Health Across the Nation (SWAN) were used (N = 2063; mean age at baseline = 46.0). Confirmatory factor analysis (CFA) and latent growth curve (LGC) models were employed to estimate level and change in AL over an 8 year period.

Results: Higher discrimination and hostility were predictive of higher AL level ($p \leq .05$). Higher perceived stress was predictive of faster rate of increase of AL ($p \leq .05$). Racial and SES differentials were present, with African American race, lower income, and lower education predictive of higher AL level ($p \leq .001$ for each). In addition, the results identified several significant pathways through which race and SES indirectly predict level and change of AL over time.

Conclusion: This was one of the first studies to investigate longitudinally AL and results supported AL as a cumulative phenomenon, affected by multiple psychosocial and demographic factors. The findings demonstrated the utility of a dynamic biopsychosocial model to better understand racial and SES differentials in AL among midlife women.

Key Words: allostatic load, race/ethnicity, socioeconomic status, discrimination, perceived stress, hostility

Acronyms: AL = allostatic load. BMI = body mass index. CFA = confirmatory factor analysis. CFI = comparative fit index. CRP = C-reactive protein. DHEA-S = dehydroepiandrosterone. EM = expectation and maximization. FIML = full information maximum likelihood. HDL = high-density lipoprotein. HT = hormone therapy. LGC = latent growth curve. LM = LaGrange multiplier. MCAR = missing completely at random. RMSEA = root mean square error of approximation. SES = socioeconomic status. SWAN = Study of Women's Health Across the Nation. Y-B χ^2 = Yuan Bentler scaled chi-square.

INTRODUCTION

There is continuing interest in identifying and characterizing relations among environmental, social, and economic conditions and health differentials (1-4). Recent theoretical developments have proposed integrated models that capture the contributions of social conditions and the possible psychosocial processes through which they operate to affect health (1, 3, 5). Allostatic load (AL) has been a useful construct in the clarification of how person-environment interactions “get under the skin” and accumulate over time to affect health (6-9). The focus of the current study is to assess racial and socioeconomic status (SES) differences in AL level and change over time, and for the first time, to test a set of predictive pathways of *several* mediating psychosocial variables in a multi-ethnic sample of midlife women.

Allostatic Load, Social Conditions, Race, and Psychosocial Mediating Factors

Allostatic load. AL is conceptualized as a multi-system, cumulative burden of physiological dysregulation (10, 11). Repeated exposure to environmental and social stressors over time can lead to overaction and dysregulation of physiological systems (12) and higher AL has been shown to be predictive of subsequent morbidity and mortality (7, 13, 14). A multi-system approach, as exemplified by AL, is often preferred over investigation of a single biological system when trying to ascertain the *multiple* and *synergistic* influences of racial, SES, and psychosocial factors on health and physiology (1, 3, 15). AL is hypothesized to be cumulative in nature and several studies show higher AL is associated with older ages (16), but few studies have examined AL change over time within individuals.

Social conditions. Lower SES is associated with higher levels of AL among adults (17-21). There is evidence for a SES gradient in AL which emerges at young ages and is maintained through later life (22). Social and economic conditions have been theorized as ‘fundamental

causes' of health differences (1-3). The stresses of lower SES reflect a lack of multiple types of resources that contribute to health disadvantages (1, 3, 5). AL has been proposed as one possible mechanism by which SES differences in health proliferate (19).

Race. Independent of SES, race is associated with AL, with African Americans having higher AL than other groups (19, 23-25). Chronic exposure to social adversity, including discrimination, is hypothesized to produce a long-term stress response leading to earlier health deterioration (or “weathering”) (24, 26). These forces are distinct from the resources linked to SES in that they additionally constrain the opportunities of groups on the basis of their race or ethnicity (27).

Psychosocial factors. There is a large literature demonstrating relations between psychosocial factors and AL (4, 7, 18, 20, 28-32). One set of factors emphasizes the relevance of stressful life events and perceived stress on higher AL (29, 33). Lower SES individuals report higher levels of perceived stress and stressful life events (34, 35). Potentially salient dimensions of social stressors not yet fully investigated with regard to AL are discrimination and interpersonal mistreatment (36). Racial and ethnic minorities report higher levels of discrimination, which in turn, contribute to poorer physical and mental health outcomes (e.g., (37-40)). A second set of factors refers to personality traits, especially hostility and cynicism, which are positively associated with AL (18, 20, 33). Lower SES and racial and ethnic minority individuals also report greater hostility (18, 20). To date, however, few studies have examined explanatory pathways of psychosocial factors on AL (see (15, 18, 20, 28) as exceptions). The current research takes the next step to investigate the explanatory pathways of *multiple* psychosocial factors on level and rate of change of AL simultaneously.

Women at Midlife: A Critical Stage in the Life Course

The current study incorporates a dynamic biopsychosocial model to investigate longitudinally the racial and SES effects on AL over time and to estimate the extent to which discrimination, perceived stress, and hostility mediate their effects. We focus on midlife because it is during these ages that the first indications of health declines frequently occur (41) and health differentials across race and SES groups are the largest (41, 42). We employ LGC models to capture the level and change in AL among a cohort of midlife women (ages 42-52 at baseline) at baseline and 7 subsequent annual visits using data from SWAN.

Guided by previous research, we hypothesize that AL will increase as women age, African American women will have higher AL than Asians, and lower SES women will have higher AL than those who are more advantaged. We also hypothesize that higher levels of reported discrimination, stress, and hostility will each increase AL. Last, we hypothesize that African American women will report higher levels of discrimination and women of lower SES will have higher stress and hostility. Thus, we anticipate significant mediation effects.

METHODS

Study Design

The Study of Women's Health Across the Nation (SWAN) is a community-based, multi-ethnic sample of midlife women designed to investigate the biological and psychosocial characteristics of the menopausal transition. Details of recruitment procedures and study design have been described in detail elsewhere (43). From 1995 through 1997, 16,065 women were screened for a longitudinal cohort at each of the 7 sites. Each site screened one racial/ethnic minority population (African Americans in Pittsburgh, Pennsylvania, Boston, Massachusetts, Detroit, Michigan and Chicago, Illinois; Japanese in Los Angeles, California; Chinese in Oakland, California; and Hispanics in Newark, New Jersey) and one Caucasian population using

a community-based sample. The SWAN protocol was approved by each site's institutional review board, and all participants provided written informed consent.

Baseline eligibility criteria. Women were eligible for the longitudinal cohort study if they: were aged 42-52 years, had an intact uterus and at least one ovary, not currently using exogenous hormones affecting ovarian functioning, had at least one menstrual period in the previous 3 months, not currently pregnant or lactating, and self-identified with one of each site's racial/ethnic groups (43). Of eligible women, 51% (N = 3302) enrolled, all of whom were pre- or early perimenopausal according to bleeding criteria (44). The current study included data drawn from baseline through follow-up visit 07. Due to administrative issues at the New Jersey Medical School, data collection was halted during follow-up visit 06 through visit 10, thus Hispanics were not included in the current study

(<http://www.edc.gsph.pitt.edu/swan/research/Documents/DataQuality/RetentionandTransitionTables/>). The final analytic sample included women who had valid values of all 11 biomarkers at baseline (N = 2743) and at least two additional AL scores available during the 8 years of assessment (N = 2063).

Data collection. Baseline and annual study visits were conducted by trained, certified staff, who supervised the collection of the self-reported data, administered in-person or telephone interviews, conducted the physical measures and performed biological specimen collection (43). Blood samples were collected annually. Laboratory assays have been detailed elsewhere (<http://www.edc.gsph.pitt.edu/swan/research/Documents/PublicationsPresentations/StandardWorking/>). Blood pressure readings were taken seated after a 5-minute rest period and two assessments were taken and averaged. Waist circumference was measured in undergarments using a tape measure at the natural waist (narrowest part of torso). Hip circumference was

measured in undergarments at the maximum extension of the buttocks. Height and weight were measured without shoes and in light indoor clothing.

Single Biomarker Measures and Composite Allostatic Load Score

Eleven biomarkers were used to create the summary AL score and were selected from available SWAN data based on their representation of multiple physiological systems, biomarkers used to operationalize AL in prior research, data availability, and pertinent to disease risk (8, 9, 11, 31, 45, 46). Cardiovascular markers included systolic and diastolic blood pressure (BP). Metabolic markers included total cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, body mass index (BMI), waist-to-hip ratio, and fasting serum glucose. Inflammatory markers included C-Reactive protein (CRP) and fibrinogen. The neuroendocrine marker was dehydroepiandrosterone (DHEA-S).

Operationalization of AL was based on an algorithm developed by Seeman et al. (9) and has since been used extensively in the literature. (e.g., (7, 9, 13, 16, 23-25, 31, 32, 47)). For each of the 11 biomarkers, the highest risk quartile value was determined based on the distribution of each parameter within the SWAN cohort (i.e., 75% quartile for all biomarkers except HDL cholesterol and DHEA-S, for which the 25% quartile represents high risk). The high risk quartile for each biomarker was determined by the cutoff value established at baseline. AL was measured by summing the number of biomarkers that fell into the high risk quartile.¹ AL was computed for baseline and each of 7 follow-up visits.² These procedures reflect empirically-driven cutoffs

¹ There is a question of how to score AL for individuals on medications that might impact biomarker values. Following previous studies and because AL theory is concerned with actual physiological regulation, we did not make an adjustment of AL values according to medication status.

² At FU 02, total cholesterol, HDL, triglycerides, glucose, CRP, and fibrinogen were not assessed. At FU07, fibrinogen was assessed for 50% of the sample. We imputed missing fibrinogen values using a linear mixed effects model where fibrinogen was a linear function of age. The mixed effect model also included a random intercept and slope. If a participant had ≥ 2 observations for fibrinogen, the missing value was imputed by the predictor based on the mixed effects model (correlation of observed vs. predicted = 0.74, $p \leq 0.001$). For the remaining missing

based on sample values. Although alternative methods for summarizing biomarker scores have been investigated, comparable results have been found regardless of method (8, 48). Both initial status and change over time were used as outcomes in the latent growth model.

Distributional qualities of each of the 11 biomarkers and cutoff points, based on baseline values, and baseline AL score are shown in Table 1. Baseline AL scores varied from 0 to 11 and the mean was 2.57 (SD=2.27).

Table 1

Demographic Measures and Menopausal Status

Baseline demographic indicators. Age was coded as a continuous variable (ages 42-52). Race was coded by dichotomous variables (African American 1=yes, 0=no; Caucasian 1=yes, 0=no). Japanese or Chinese was the combined reference category because of similar and lowest AL scores. Educational attainment was coded on a 1-5 scale (<12 years; high school graduate; some college; college graduate; and education beyond 4-year college). Household income was coded on a 1-8 scale (<\$10,000; \$10,000-19,999; \$20,000-34,999; \$35,000-49,999; \$50,000-74,999; \$75,000-99,999; \$100,000-149,999; and \geq \$150,000). Marital status was coded as a dichotomy (married/cohabiting vs. not) and categorical for descriptive statistics (single/nevermarried, married/cohabiting, separated/divorced/widowed).

Menopausal transition stage was coded as time-varying and followed standard guidelines (44). Categories were: premenopausal (bleeding in the previous 3 months with no change in cycle predictability in the past year); early perimenopausal (bleeding in the previous 3 months with decrease in cycle predictability in the past year); late perimenopause (3-11 months amenorrhea); postmenopausal (12 or more months amenorrhea). The postmenopausal stage was

biomarkers in FU02, the values were imputed by averaging values at FU01 and FU03. Analyses conducted using the non-imputed sample produced similar results.

further subdivided into those who were current users of hormone therapy (HT) and those who were not. Women who used HT before postmenopause were excluded in the follow-up visits when it was used but reinstated in those who stopped HT after an 18 month HT wash-out period (as established by the SWAN Coordinating Center). Women who had hysterectomy without bilateral oophorectomy before postmenopause were dropped at the follow-up visit when it occurred. Premenopausal women who had bilateral oophorectomy (with or without hysterectomy) were coded as surgically postmenopausal. Women who were pregnant or breastfeeding were excluded in the follow-up visits it occurred and were reinstated once they were no longer pregnant or breastfeeding. The final *menopausal transition stage* variable was structured as a latent growth variable scaled 0-4 with both an initial intercept and a growth component. At baseline all women were scored either premenopausal or early premenopause.

Psychosocial Intervening Measures

To the extent possible from the data available, the intervening measures were time-varying. *Discrimination* was assessed with a modified version of the Detroit Area Study Everyday Discrimination Scale (36). This 10-item scale asked participants to rate the frequency they experienced various types of interpersonal mistreatment over the past 12 months (e.g., “You are treated with less respect than other people.”) using a 1-4 response scale. The scale has demonstrated high levels of internal consistency (36, 49). The current study used scale items collected at baseline and 3 years of follow-up. Items were averaged within each year and then used as 4 indicators of discrimination.

Perceived stress was measured with the 4-item shortened version of the Perceived Stress Scale (50) and has been validated in several studies. The items assessed stress in the past 2 weeks (e.g., “Felt unable to control important things in your life.”) using a 1-5 response scale. Because

of substantial missing data at the first follow-up visit, the current study averaged baseline and subsequent scores for 5 years of follow-up. These 6 items were indicators of a latent variable representing perceived stress. *Hostility* was measured at baseline from a subscale of 13-items with dichotomous 0-1 responses from the Cooke-Medley Questionnaire (51). A sum score was used.³

Analysis

The distributional qualities including mean, quartiles, range, standard deviations, and the empirical cutoff values evaluated at baseline for each of the 11 biomarkers were computed. The baseline percentage distributions of the demographic and menopausal status variables were estimated. Standard χ^2 was used to test associations between each baseline variable and baseline mean AL. Bivariate correlations of all variables in the final model and significance levels were computed. Means, standard deviations, and factor loadings for each of the intervening variables for each year were estimated for the CFA.

An initial CFA was performed among the hypothesized independent measured and latent variables with each hypothesized latent construct predicting its measured indicators. This analysis assessed the adequacy of the proposed factor structure (measurement model) and the relationships among the latent and measured variables. All latent constructs and the single-item variables were correlated with no assignment of temporal ordering. Then an LGC model was tested in which the demographic variables predicted discrimination, perceived stress, and hostility which in turn predicted the AL intercepts over time and trajectory of the slope. In LGC

³ The original model specification also included latent variables for social support and stressful life events. Social support included 4 measures selected from the Medical Outcomes Study Social Support Survey indicating instrumental and emotional support (Sherbourne and Stewart 1991). It was collected annually through follow-up 06. Stressful life events included 20 items modified from the Psychiatric Epidemiology Research Interview (Dohrenwend, Krasnoff et al. 1978). Stressful life events was examined as a total number and also categorized based on items that were 'very stressful.' None of these variables were significant in earlier model development and so were dropped from the final model.

modeling, the *intercept* corresponds to the initial status of women's AL at baseline. The *slope* represents the rate of change over the period of observation. In addition, menopausal status was treated as another control variable and its intercepts and slope predicted the AL latent variables. Covariances were allowed between age and menopausal transition stage intercept and slope. Correlations (covariances) between adjacent error residuals within the latent intercept and slope variables were considered for addition to the model using the LaGrange Multiplier (LM) test (52). The LM test reports significant additional paths or covariances that can improve the fit of the model.

The EQS structural equations program (53) was used to assess the latent growth curve (LGC) models and provided information on the relationships among AL, demographic, menopausal status, and psychosocial variables. Goodness-of-fit of the models was assessed with the robust Yuan-Bentler scaled chi-square statistic (Y-B χ^2), the comparative fit index (CFI), and the root mean square error of approximation (RMSEA) (53, 54). Robust statistics were used due to the non-normality of the data ((55); normalized estimate = 65.36). The RMSEA should be < 0.06, and values $\geq .95$ for the CFI are desirable (54).

Due to multiple assessments over many years, relatively few participants had entirely complete data. Thus, the full information of maximum likelihood (FIML) missing data method available in EQS that uses an expectation and maximization (EM) algorithm was employed (53). In EM imputation parameter estimates are obtained by iterating an expectation step and a maximization step. FIML is the recommended data imputation method when using the EQS structural equations modeling program. Diagnostics indicated that the missing data points were missing completely at random (MCAR).

RESULTS

Baseline Descriptive Results

Baseline demographics and menopausal transition stage are presented in Table 2. Associations between each covariate and mean AL assessed at baseline are also shown. The average age at baseline was 46 (not shown). Over half of the sample was Caucasian, 29% African American, and the rest were Chinese or Japanese. In general, women in the sample were well-educated (49% had 4 year college degrees or higher) and lived in relatively affluent households (31% lived in households with annual incomes of \$75,000 or higher). Each baseline demographic variable was significantly associated with mean AL at baseline ($p \leq .001$). Age was positively associated with higher mean AL. African American women had the highest mean AL and Chinese and Japanese similar and the lowest mean ALs. Higher education was significantly associated with lower mean AL. Similarly, higher household income was associated with lower mean AL. Married/cohabiting women had lower mean AL than other marital statuses. Women who were in early perimenopause at baseline had higher mean AL than premenopausal women.

Table 2

Confirmatory Factor Analysis

Table 3 reports the means (or percentages), standard deviations, ranges, and factor loadings of the measured variables in the CFA. All factor loadings were significant ($p \leq .001$). Fit indexes for the CFA model are reasonable: Y-B $\chi^2 = 1641.15/400$ *df*; CFI = .96, RMSEA = .046. Correlations among the variables in the model are reported in Table 4. Of note, several predictors were correlated with the AL intercept but very few variables were significantly associated with the slope (only perceived stress and menopausal transition stage slope) and those associations were relatively weak. All variables in the model were significantly associated with the AL intercept latent variable.

Table 3

Table 4

Latent Growth Curve Results

Figure 1 presents the significant predictive paths in the final trimmed LGC model. For readability, the figure does not depict the significant relationships among the predictors. They are similar to those reported in Table 4. The fit indexes of the path model are acceptable: $\chi^2 = 1449.46/438$ *df*; CFI = .97, RMSEA = .039.

Direct effects. AL intercept was significantly predicted by several variables. Two of the mediating variables, discrimination and hostility, were predictive of higher AL intercept. African American race (compared to Japanese and Chinese) had higher AL intercept (there was no difference for White race). Older age, lower income, less education, and menopausal transition stage intercept were predictive of higher AL intercept. Perceived stress and menopausal transition stage slope were predictive of higher AL slope.

Among the mediating variables, higher discrimination was predicted by African American race (versus Japanese and Chinese) and lower income. Higher stress was predicted by less income and education and was lower among Caucasian women (versus Japanese and Chinese). Higher hostility was predicted by African American race, less income, lower education, and was lower among Caucasian women. Marital status was included in the model but it was not significant.

Figure 1

Indirect effects. The AL intercept was indirectly and significantly predicted by African American race ($p \leq .001$; standardized total effect = .25, indirect effect = .02), less income ($p \leq .001$; total effect = -.165, indirect effect = -.013), and lower education ($p \leq .05$; total = -.08,

indirect effect = -.01). The AL slope was predicted by less income ($p \leq .05$; no direct effect; indirect effect = -.01). (The other possible indirect pathways depicted in Figure 1 were not significant.)

DISCUSSION

This is one of the first longitudinal studies to investigate racial and SES differences in AL over time and identify predictive pathways of multiple psychosocial factors. Specifically, we find persistent racial and SES differentials on AL, with African American women and women of lower SES having higher AL. In addition, we find support for the influences of reported discrimination, perceived stress, and hostility on level and change in AL among of midlife women. Our results also identify several significant pathways through which race and SES indirectly predict level and change of AL through these psychosocial mediators. Therefore, our findings support the utility of using a dynamic biopsychosocial model to better understand racial and SES differentials in AL among midlife women.

A unique contribution of this work is that it examines AL over a 7 year time interval. The majority of the covariates are predictive of the AL intercept versus the slope. The intercept is representative of women's starting values measured at baseline. AL intercept reflects the lifetime of exposure to social and environmental stressors and the slope reflects the change over a much shorter duration. However, AL slope is also positive for each year of observation, and mean level of AL increases each year (see Table 3). Importantly, the one mediating variable we found to be predictive of rate of change of AL was perceived stress, also the construct most closely linked to theories about the factors that affect accumulation of AL (9-12). It is perhaps not surprising that this effect is more modest than those of the AL intercept given the differences in time scale. The finding that older age is predictive of AL intercept further supports the idea of the cumulative

pattern of physiological dysregulation that AL exhibits. Overall, these findings are consistent with the proposition that AL accumulates over the life course.

We also show that discrimination is predictive of AL in adult women. As expected, women reporting higher levels of discrimination have higher AL levels during midlife. There is a large literature linking higher discrimination to poorer health outcomes (for reviews see (37-39)), but little work has focused specifically on AL. One exception is a recent study of adolescents (28). The authors found higher level of discrimination is predictive of higher AL, as we also show. Moreover, our results demonstrate that two demographic factors (i.e., being African American, lower income) are predictive of higher discrimination, suggesting the need to further investigate the ways in which the multiple dimensions of social placement may result in discrimination (e.g., race, income, age, gender, and the like).

Significantly, the longitudinal results indicate that, consistent with theories linking stressful experiences to AL accumulation, women reporting higher perceived stress experience a faster rate of increase in AL over time than their less stressed counterparts. Perceived stress represents individuals' interpretation of life events and environmental demands. It reflects not only these 'objective' stressors but also personal experiences, resources, coping strategies, and availability of social supports (56). Previous research using cross-sectional AL data also found a significant relationship between higher perceived stress and higher AL (29, 33). Our findings support and extend this earlier work by demonstrating that differences in perceived stress represent a significant pathway affecting the rate at which AL *accumulates* over time. As our model also indicates, such perceptions are strongly linked to central characteristics that affect individuals' positions and experiences within our society – namely being non-Caucasian, of lower education, or lower income. In contrast to some earlier work (32, 33); we did not find

stressful life events predictive of AL independent of perceived stress, although Gleib et al. (2007) note that the effects were small. And more recently, a study of US midlife adults found no relationship between either perceived stress or stressful life events and AL (18). Clearly, the interrelationships between perceived stress and more objective stressors are complex and their impact on AL is incompletely understood and warrants further investigation.

As we hypothesized, hostility is predictive of higher AL among midlife women, confirming findings from two previous studies (20). There is a large literature illustrating the contribution of hostility for specific biological systems, such as cardiovascular (57) and metabolic (58). The current findings along with the earlier studies of AL suggest that hostility is also associated with biomarkers representative of multiple physiological systems.

Our findings highlight some of the multiple ways in which race and SES impact AL, as illustrated in Figure 1, and indicate several significant indirect effects. For African American women, indirect effects through higher discrimination and higher hostility are predictive of higher level of AL. For lower income women, indirect effects through increased discrimination and hostility are predictive of higher AL level and higher perceived stress predictive of more rapid increases in AL. Finally, for women with lower education, indirect effects through hostility are predictive of higher level of AL. Taken together, these results suggest the complex ways in which race, SES, and psychosocial factors operate to influence AL. They also suggest possible opportunities for intervention, such as stress management programs.

Although several psychosocial mediator variables were tested and we include three in our final model, significant racial and SES direct effects persist. African American women and women of lower SES have higher levels of AL. These findings are comparable to earlier studies of AL (17-21, 23-25). These results point to the need to cast a wider net in future work with

respect to potential mediating pathways. For example, the inclusion of not only psychosocial but also lifestyle measures may prove fruitful.

The strengths of this research have been highlighted, however, some limitations should be mentioned. Although the data are some of the most comprehensive available for the study of midlife women, SWAN is a community-based sample. However, attempts were made in SWAN to recruit participants who were representative of defined and diverse communities, and we note that our overall substantive findings are in line with many other national studies of AL. One recent study investigating women of all ages using NHANES, a nationally representative sample, reported similar findings for the demographic variables reported here (47). In addition, data limitations precluded analysis of Hispanic women. Although we used a specification of AL used widely in the literature, we note that we were limited by the biomarkers available in the dataset. Again, previous studies suggest that despite differences in operationalization of AL, substantive findings are robust. Last, we acknowledge the possibility of moderating effects of psychosocial factors, but did not examine them because of the complexity of the mediation model we present. It remains for future research to explore both mediating and moderating pathways for AL.

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Table 1. Biomarker and AL distribution at baseline, SWAN (N = 2063)

	Range	Mean	Standard Deviation	25%	Median	75%	Cutoff point ^a
<i>Cardiovascular</i>							
Systolic blood pressure (mm Hg)	(80, 227)	116.26	17.33	104	113	125	>125
Diastolic blood pressure (mm Hg)	(41,143)	74.31	10.46	68	73	80	>80
<i>Metabolic</i>							
Total cholesterol (mg/dL)	(92, 338)	193.41	34.14	171	191	213	>213
HDL (mg/dL)	(18, 138)	56.86	14.21	47	55	66	<47
Triglycerides	(31, 1185)	109.17	76.32	34	89	125	>127
Glucose	(52, 439)	97.35	29.81	86	91	98	>98
Body Mass Index (kg/m ²)	(14.99, 59.13)	27.84	7.18	22.49	26.01	31.76	>31.76
Waist Hip Ratio	(0.51, 1.14)	0.80	0.07	0.75	0.79	0.84	>0.84
<i>Inflammatory</i>							
C-Reactive Protein (mg/L)	(0.04, 9.90)	2.19	2.38	0.5	1.2	3.0	>3.0
Fibrinogen (mg/dL)	(122, 722)	291.72	66.16	246	282	325	>325
<i>Neuroendocrine</i>							
DHEA-S (µg/dL)	(0.29, 621.5)	133.75	81.13	76.9	117.7	172.7	<76.9
<i>Allostatic load</i>							
	(0, 11)	2.57	2.27	1	2	4	

Note: mm=millimeter; Hg=mercury; mg=milligrams; dL=deciliter; µg=microgram; L=liter

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^aQuartile cut-off indicates the cut-off value that distinguishes the high-risk quartile and is based on values in the sample at baseline. High risk was defined as the 75% quartile for all biomarkers except HDL cholesterol and DHEA-S for which the 25% quartile represents high risk.)

Table 2. Sociodemographic characteristics at baseline, SWAN (N=2063)

Characteristics at baseline	Percentage	Mean AL
Age		
42-45 years	45.95	2.37***
46-49 years	42.90	2.65
50-52 years	11.15	2.95
Race/ethnicity		
Caucasian	50.75	2.32***
African American	28.79	3.69
Chinese	9.45	1.59
Japanese	11.00	1.67
Education		
<12 years	3.12	3.27***
High school graduate	16.07	3.18
Some college	32.05	2.84
College graduate	22.65	2.12
Post-college	26.11	2.17
Household income		
<\$10,000	4.23	3.88***
\$10,000 - 19,999	5.33	4.20
\$20,000 – 34,999	14.93	2.94
\$35,000 – 49,999	18.72	2.68

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\$50,000 – 74,999	25.73	2.40
\$75,000 – 99, 999	14.34	2.23
\$100,000 – 149,999	11.75	1.97
≥\$150,000	4.98	1.55

Marital status

Single/Never Married	13.90	3.04***
Married/Cohabiting	68.09	2.38
Separated/Divorced/Widowed	18.01	3.01

Menopausal transition stage

Premenopausal	54.55	2.42***
Early perimenopausal	45.45	2.77
Late perimenopausal	--	
Postmenopausal, no HRT	--	
Postmenopausal, HRT	--	

Site

Detroit	18.18	3.63***
Boston	15.90	2.55
Chicago	14.83	3.25
Davis	17.16	1.81
Los Angeles	19.15	1.65
Pittsburgh	14.78	2.71

*** $p \leq .001$. Bivariate chi-square.

Table 3. Means or percentages, standard deviations, ranges, and factor loadings of measured variables in the Confirmatory Factor Analysis, SWAN (N = 2063)

<u>Latent and Measured Variables</u> (range)	<u>Mean (S. D.)/ percentage</u>	<u>Factor Loading*</u>
<i>Baseline demographic variables</i>		
African-American	29%	NA**
Caucasian	51%	NA
Asian (Japanese and Chinese)	20%	
Age (range = 42-52 years)	46.00 (2.70)	NA
Income (1-8)	2.62 (1.71)	NA
Education (1-5)	3.53 (1.13)	NA
Married or cohabiting (yes/no)	68%	NA
<i>Mediating psychosocial variables</i>		
Discrimination (1-4)		
Baseline	1.76 (0.47)	.79
Year 1	1.75 (0.47)	.84
Year 2	1.70 (0.48)	.85
Year 3	1.67 (0.49)	.84
Stress*** (4-20)		
Baseline	8.34 (2.86)	.55
Year 2	7.70 (2.86)	.74
Year 3	7.69 (2.88)	.75
Year 4	7.59 (2.86)	.81
Year 5	7.63 (2.91)	.79
Year 6	7.61 (2.99)	.74
Hostility (0-13)	3.77 (2.91)	NA
<i>Menopausal Transition Stage Latent Growth</i>		
		Intercept/Slope
Baseline	0.45 (0.50)	.77/NA
Year 1	0.83 (0.66)	.57/.35
Year 2	1.05 (0.85)	.45/.55
Year 3	1.35 (1.10)	.38/.69
Year 4	1.72 (1.28)	.33/.81

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Year 5	2.08 (1.39)	.27/.82
Year 6	2.46 (1.44)	.21/.78
<i>Allostatic Load Latent Growth Variables</i>		Intercept/Slope
Baseline	2.57 (2.27)	.92/NA
Year 1	2.57 (2.26)	.94/.07
Year 2	2.68 (2.29)	.93/.14
Year 3	2.67 (2.28)	.94/.21
Year 4	2.73 (2.19)	.97/.29
Year 5	2.81 (2.21)	.97/.36
Year 6	2.80 (2.13)	.98/.44
Year 7	2.76 (2.13)	.95/.50

* All factor loadings significant, $p \leq .001$. Factor loadings are standardized.

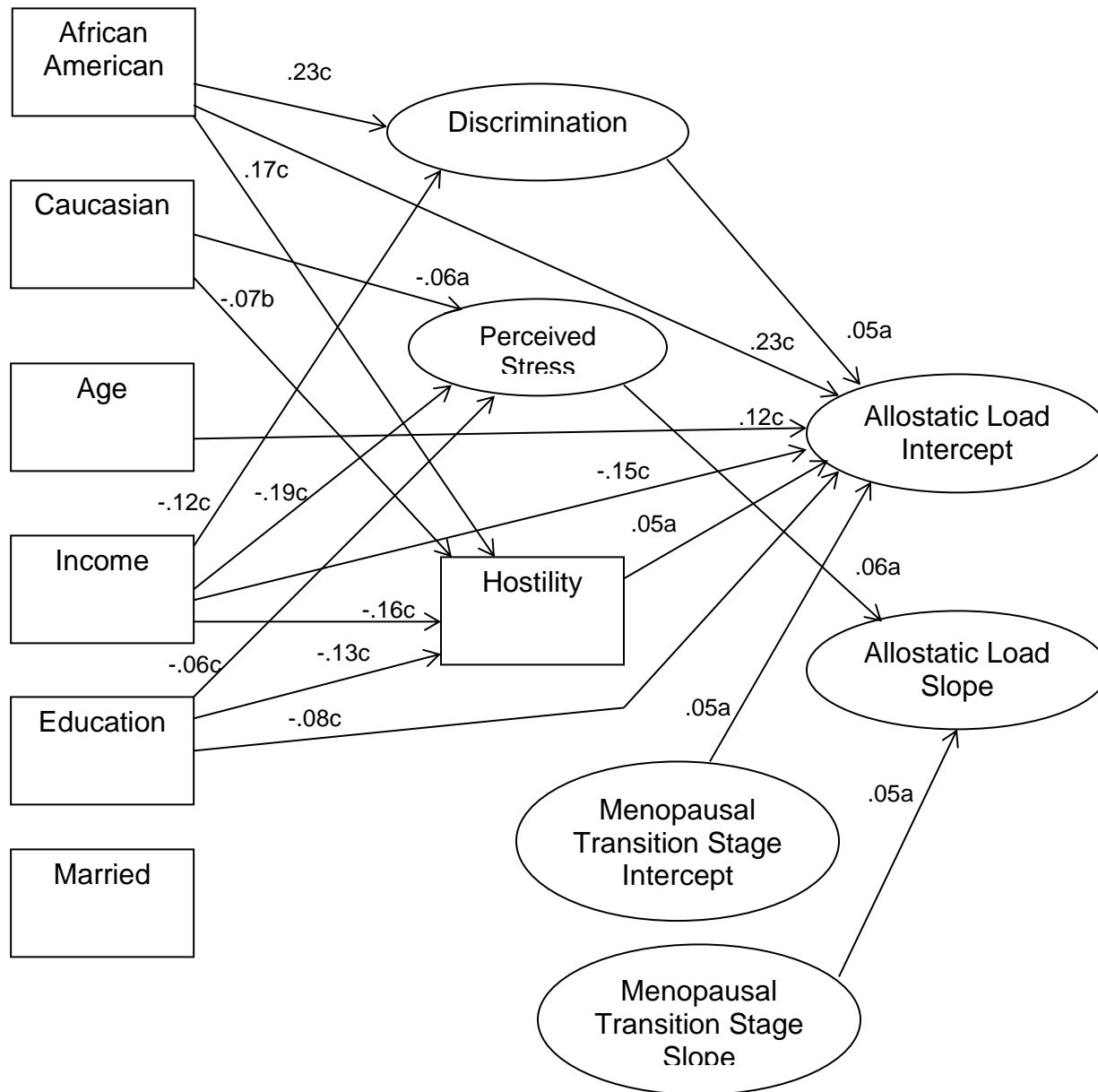
** NA = Not applicable. ***Not available Year 1, Year 7.

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Table 4. Correlations among variables in model ($N=2063$; * = $p \leq .05$, ** = $p \leq .01$, *** = $p \leq .001$).

	1	2	3	4	5	6	7	8	9	10	11	12
1. AL Intercept	—											
2. AL Slope	-.38***	—										
3. African-Amer.	.33***	-.04	—									
4. Age	.14***	-.05	-.02	—								
5. Income	-.25***	-.05	-.30***	.07***	—							
6. Education	-.19***	-.04	-.19***	-.02	.39***	—						
7. Married	-.15***	-.01	-.27***	.01	.47***	.05*	—					
8. Caucasian	-.12***	-.01	-.65***	-.01	.16***	.21***	.14***	—				
9. Discrimination	.17***	-.03	.24***	-.04	-.19***	-.04	-.15***	-.20***	—			
10. Stress	.08***	.06*	.02	-.03	-.22***	-.12***	-.08***	-.11***	.39***	—		
11. Hostility	.19***	.02	.28***	.00	-.28***	-.23***	-.16***	-.24***	.38***	.28***	—	
12. Menopausal transitions stage intercept	.11***	-.03	.08**	.20***	-.05	-.10***	-.02	-.02	.13***	.10***	.11***	—
13. Menopausal transitions stage slope	.13***	.06*	.08***	.52***	-.03	-.10***	-.04	-.08***	-.03	-.01	.09***	.13***

Figure 1. Latent growth curve analysis of demographic variables and mediating variables and pathways of AL, SWAN (N = 2063)



a = $p \leq .05$; b = $p \leq .01$; c = $p \leq .001$. See text for additional detail.