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Cardiovascular and Metabolic Risk in Women in the First Year Postpartum: Allostatic Load as a Function of Race, Ethnicity and Poverty Status

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Abstract

Objective: Allostatic load (AL) represents multi-system physiological “wear-and-tear” reflecting emerging chronic disease risk. We assessed AL during the first year postpartum in a diverse community sample with known health disparities.

Methods: The *Eunice Kennedy Shriver* National Institute for Child Health and Human Development Community Child Health Network enrolled 2,448 predominantly low income African-American, Latina and White women immediately after delivery of liveborn infants at ≥ weeks gestation, following them over time with interviews, clinical measures, and biomarkers. AL at six and twelve months postpartum was measured by body mass index, waist:hip ratio, blood pressure, pulse, hgbAlC, hsCRP, total cholesterol and HDL, and diurnal cortisol slope.

Results: Adverse AL health-risk profiles were significantly more prevalent among African-American women compared to non-Hispanic Whites, with Latinas intermediate. Breastfeeding was protective, particularly for White women. Complications of pregnancy were associated with higher AL, and disparities persisted or worsened through the first year postpartum.

Conclusions: Adverse AL profiles occurred in a substantial proportion of postpartum women, and disparities did not improve from birth to one year. Breastfeeding was protective for the mother.

Keywords

Postpartum health; allostatic load; cardiovascular risk; metabolic risk

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INTRODUCTION

The negative effects of pregnancy on mother's own cardiometabolic risk post-delivery has not been well studied. How well women minimize this risk will affect their health in subsequent pregnancies (and beyond) and the health of subsequently born children.[1,2] Postpartum physiological recovery may differ among racial/ethnic groups, potentially explaining the continued health disparities through the life course.

A normal pregnancy induces major maternal physiological adaptation to support a developing fetus. Dramatic changes in the immune and cardiovascular systems include an overall decrease in pro-inflammatory cytokines and an increase in counter-regulatory cytokines as a normal pregnancy progresses,[3] and a dramatic increase in blood volume and cardiac output to assure adequate perfusion of the uterus.[4] However, in healthy women, maternal physiology gradually returns to normal after delivery, with the greatest improvement in the first six months, and a return to baseline by one year postpartum. [5]

The literature on postpartum health focuses largely on recovery from high-risk pregnancy due to co-morbid conditions (e.g. gestational diabetes or preeclampsia) in women drawn from clinic-based samples.[6] In contrast, studies on postpartum health in a community-based sample are rare. Several studies have shown the role of inflammation during pregnancy and the increased risk of poor pregnancy outcome.[7] Likewise, women who had gestational diabetes (but not type 2 diabetes) may have inflammatory or cardiovascular changes in long term postpartum follow-up.[8,9]

Prior work in health over the life course has shown that extraordinary and/or persistent stressors can shift health trajectories leading to cardiometabolic risk. Pregnancy itself is a major physiological stress to which the mother must adapt and then from which she must recover.[5] AL was developed to study the physiological "wear-and-tear" that accrues because the body must adapt to experiences of chronic and high levels of stress over the life course. [10] AL thus represents the accumulated risk for chronic disease, as well as early and all-cause mortality.[11,12]

This paper from the Eunice Kennedy Shriver National Institute of Child Health and Human Development's (NICHD)'s Community Child Health Network (CCHN) presents findings from a community-based longitudinal investigation of mostly low-income women, diverse race-ethnically and geographically, who were enrolled immediately following delivery. Here we describe the cohort's postpartum cardiometabolic risk and recovery in the first year following delivery as a function of race-ethnicity and poverty status, and taking into account prior pregnancy-related risk factors.

METHODS

Participants

CCHN was a community-based participatory research study. There were three urban sites (Washington, DC; Baltimore, Maryland; and Los Angeles County, California); one suburban (Lake County, Illinois); and one rural (seven counties in eastern North Carolina). In total,

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2,448 mothers were recruited, generally on postpartum hospital units. Inclusion criteria were maternal age of 18–40 years; self-identification as "African-American/African- American/ non-Hispanic," "Hispanic/Latina," or "White;"[a] and infant live-born at ≥20 weeks gestation. Low income mothers and those delivering preterm were oversampled. Exclusion criteria were speaking neither English nor Spanish; inability to provide informed consent; child's birth order 4th or higher; study area residence under six months; incarceration; or plans for surgical sterilization. Research staff were trained to standardization, conducted home interviews, and collected clinical measures and biospecimens. Medical chart review was completed under clinical direction. See Ramey *et al.* for study overview.[13] IRB approvals were obtained in all sites.

Study variables

The study's main outcome was maternal AL calculated from 10 biomarkers collected at six months and one year postpartum. Participants who did not provide biomarkers at six months postpartum were retained to preserve the possibility that they could still provide biomarker data at one year postpartum. The AL measures were: *Body Mass Index (BMI)* was weight (kg) measured using the LifeSource® UC-321 ProFIT Precision Personal Health digital scale (A&D Medical, San Jose, CA, maximal 350 lbs) divided by height squared (meters). Women stood against a vertical wall marked for height measurement. World Health Organization (WHO) categories were used to classify participants as underweight <18.5 kg/m, normal weight 18.5−−24.9 kg/m^2, overweight 25.0−−29.9 kg/m^2, and obese ≥30.0 kg/m^2.[14] *Waist:Hip Ratio:* Waist and hip circumferences (centimeters) were measured standing, with a high risk clinical cut-off of 0.85.[15] *Systolic and Diastolic Blood Pressures:* Systolic and diastolic blood pressure (BP) readings (mmHg) were each recorded three times while mothers were seated during a home visit using an OMRON HEM-711DLX or HEM-907XL Pro standardized digital sphygmomanometer (OMRON Global, Osaka, Japan); the three readings of each type of BP were averaged to create composite measures of systolic and diastolic BP. Clinical cut-offs were 140 for systolic and 90 for diastolic BP (WHO criteria).[16] *Pulse:* Pulse readings (beats per minute [BPM]) were displayed during BP readings. High resting pulse was defined as 91 BPM.[17]

Blood and saliva specimens were analyzed by ZRT laboratory, Portland, OR. Whole blood spots on Guthrie paper were collected by finger prick with a 14 gauge spring-loaded lancet and dried. *C-Reactive Protein (hsCRP; mg/L)* pro-inflammatory state was defined as above 3.[18] *HgbA1c (%)* clinical cutoff was 5.4%, higher values representing pre-diabetes or diabetes.[19] *HDL* clinical cutoff was 40 mg/dL (lower values were less optimal). *Total cholesterol:HDL ratio* clinical cutoff was 5.9 (higher values less optimal).[20] *Cortisol slope:* Participants collected their own saliva at three times (immediately upon awakening and before getting out of bed; 30 minutes later; and just before going to bed in the evening). Completed packets were mailed by the participant or retrieved by staff. Cortisol values in the PM saliva samples were subtracted from the values in the AM sample, and then divided by the number of hours between these collections. As no "clinical" cut point exists, the value

[a]Throughout the paper, these groups will be described as "African-American", White", and "Latina".

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corresponding to the top (flattest) quartile of cortisol slopes was used (−.01). Flatter slopes are associated with negative health outcomes and earlier mortality.[21]

Calculation of AL scores[22] : AL was computed two ways for both six and twelve months and postpartum. The first "quartile" method assigned a score of "1" to each of the 10 component biomarker values in the top quartile. The second "clinical" method assigned a score of "1" to each component meeting or exceeding its clinical cutoff and 0 if below the cutoff. HDL was reverse-scored because higher values are advantageous. The ten component values were added for each method. Computed either way, AL ranged from 0–10 (10 being worst). When at least seven of 10 biomarkers were available for a participant, their sum was scaled to 10 by dividing the sum by the total number of non-missing biomarkers and multiplying by 10. Twenty-two percent of the sample at six months postpartum, and 23% at one year postpartum, had fewer than seven biomarkers available and were dropped. Insufficient biomarker data were largely due to field conditions and assay requirements.

We collected data on several demographic variables during home interviews: *Maternal Age; Education* (4 categories: <high school graduate; high school graduate; some college; college graduate or more), *Marital Status* (three categories: married; not married but in a relationship; not married or in a relationship), *Race-Ethnicity* (three categories: African-American; Latina; White), and *Poverty Status* (three categories: poor=household income below the federal poverty level, (FPL); near poor=household income between 100% and 200% of the FPL; not poor=at least 200% of the FPL).

We used medical record data abstracted via chart review to construct several pregnancy-related and infant health and feeding variables (all coded "yes" or "no"): *History of Prior Pregnancy;* diagnosed *Gestational Diabetes;* diagnosed *Pre-eclampsia; Pre-Term Birth (<37 weeks gestation); Low Birthweight (<2500 grams);* and *Very Low Birthweight (<1500 grams);"Ever breastfed during this pregnancy "* and *"Breastfeeding at six months ".*

Analyses

Data analyses were carried out using one-way analysis of variance (ANOVA) with follow up t-tests for continuous variables, and Chi-Square tests for categorical variables. Individual AL components and the composite AL measures were calculated separately by race- ethnic group, by poverty status, and by breastfeeding, gestational diabetes, preeclampsia, preterm birth, delivery of a low birth weight infant, and parity. To test differences in AL values among the subgroups, we used one-way ANOVA with follow up *t*-tests for normally distributed biomarkers and one-way non-parametric ANOVA with follow up Wilcoxon rank-sum tests for biomarkers with skewed distributions (HS-CRP, HgbA1C and HDL cholesterol).

We first analyzed the total available sample for each time point, and then, included only participants with available data at both time points. Since we found the two approaches were similar, we only report the results for the first set, explaining the changes in available number of participants for different variables. We report statistically significant differences at $p<.05$. We used a Bonferroni correction factor for multiple comparisons. Since there were

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significant site- differences with regard to poverty and race-ethnicity composition, so we are not reporting site- specific data.

RESULTS

Descriptive statistics:_

Table 1 describes baseline maternal demographic and infant feeding variables. White women were significantly older, most likely to have completed college and be married. Latinas were significantly most likely to have had a prior pregnancy and initiate breastfeeding, although Whites were most likely to continue to breastfeed through six months postpartum. African-American women were significantly most likely to be poor, have diagnosed preeclampsia, and deliver a low birth weight infant. Mothers who were not poor were significantly older, and most likely to have completed college be married, initiate breastfeeding, and continue breastfeeding through six months postpartum.

AL component values:

Table 2 shows values on each of the individual AL component biomarkers, split by race-ethnicity and by poverty status, with significant differences at six and twelve months and postpartum, summarized in the final two columns. Median BMI was “overweight” for all race-ethnic and poverty status categories, and approached “obese” among the poor. Almost half of the African-Americans were obese. Median resting pulse was also significantly elevated among the poor and near poor compared to mothers who were not poor.

Individual AL biomarkers showed the same pattern of race-ethnic group differences, and this pattern was stable from six to twelve months postpartum: African-Americans had significantly worse health outcomes than both Whites and Latinas, who did not differ, including higher BMI, higher systolic and diastolic BP, higher resting pulse rate, higher HgbAlc and a flatter cortisol slope Similarly, among the three groups, African-Americans had significantly worse diastolic BP readings both at six and twelve months postpartum. Although the mean diastolic BP at six months postpartum was significantly higher in Whites than in Latinas, by one year postpartum, this discrepancy was no longer significant.

Race-ethnic group differences on the remaining biomarkers were largely consistent with overall patterns, with slight departures. Notably, for cholesterol and HS-CRP, differences were not evident at six months postpartum, but emerged by twelve months. (Table 2)

As shown in Table 2, at six months postpartum, poor and near-poor mothers, who did not differ from each other, had significantly higher BMIs, waist:hip ratios, and mean resting pulse rates as well as significantly lower HDL cholesterol than did non-poor mothers At twelve months postpartum, these disparities by poverty remained significant. Moreover, several new poverty disparities emerged for AL components that had not been significant at six months postpartum: poor mothers now demonstrated significantly higher diastolic BP than did near poor mothers poor and near poor mothers had significantly worse values for HS-CRP and Hgb-A1C than did mothers who were not poor and mothers who were poor had significantly flatter cortisol slopes than mothers who were near-poor or not poor.

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Composite AL measures:

Table 3 Panel A presents means for the two composite measures of AL (i.e., top quartile AL score and clinical AL score) at each time point, separately by race-ethnicity. African-Americans had higher AL scores than Latinas, who in turn had higher scores than Whites, and this pattern of race-ethnicity differences was stable from six to twelve months postpartum.

Table 3 also contains mean AL by race-ethnicity for subgroups of mothers as a function their breastfeeding status, pregnancy morbidities, and parity. Breastfeeding was protective overall, but the magnitude of the protective effect varied as a function of likelihood of breastfeeding initiation and breastfeeding duration, such that Whites derived the largest benefits, Latinas derived smaller benefits, and African-Americans deriving little if any benefit. Gestational diabetes and preeclampsia were both associated with AL decrements across race- ethnicity groups, though the magnitude of the decrement was much larger for African-Americans and Latinas than for Whites, and these patterns were consistent at six and twelve months postpartum. In general, delivering a low (vs. normal) birth weight infant was associated with worse AL scores at both time points, but again this relationships was stronger among African- Americans and Latinas than in Whites. Neither preterm birth nor parity were significantly related to AL scores

Results by poverty status in panel B of Table 3 indicate that whereas poor and near-poor mothers did not differ from each other in AL, both groups had significantly higher AL scores than non-poor mothers, and this was true at both six and twelve months postpartum.

DISCUSSION

We describe postpartum cardiovascular and metabolic risks in a geographically, race-ethnically and socio-economically diverse US cohort. Overall, adverse postpartum cardiometabolic risk profiles show race-ethnic and socio-economic gradients: African-American women worse than Latina, who were worse than White, and poor women worse compare to near-poor and not poor. We also found the higher risk were retained or magnified for groups with a pregnancy morbidity. Among the AL component measures, being White and “not-poor” generally reflected better health throughout the first postpartum year. The protective effects of breastfeeding were evident but varied by race-ethnicity in parallel with difference among those groups in breastfeeding initiation and duration. Generally, the groups with high risk AL profiles retained that standing from six months to one year postpartum. The AL profiles were similar between the nulliparous as well as multiparous women.

To our knowledge this is the first longitudinal AL study among a large group of women during the postpartum period. Compared to the women in the 15th wave of the CARDIA study,[23] our mean pulse, HDL and hsCRP were higher and cortisol slopes flatter. By enrolling a non- clinical sample and testing the effects of the major pregnancy morbidities and of lactation as they occurred, we show clear evidence of SES and race-ethnic disparities in sub-clinical and early clinical physiological factors among young women who, without intervention, may be at risk for future disease.

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Breastfeeding was protective at twelve months postpartum, though primarily for Whites and to a lesser extent Latinas. This could reflect a direct benefit from longer durations of breastfeeding compounded by better a socioeconomic status of those women who can afford to breastfeed for longer periods. Thus, this observed benefit may be cumulative. In CARDIA, breastfeeding for at least three months attenuated adverse pregnancy-associated metabolic changes in the first year postpartum (HDL and fasting insulin).[24] The lifetime duration of lactation across all pregnancies (up to four, cumulative duration 1.2 years) was inversely associated with the likelihood of metabolic syndrome at midlife.[25] Frei et. al,'s analysis of NHANES data suggests a cross-sectional relationship between low vitamin D levels and higher biological risk, including AL.[26] Our own data suggest that adding vitamin D sufficiency to the AL index increases the strength of prediction for adverse perinatal outcomes.[27] In general, women who breastfeed are more likely to be supplementing their vitamin D intake (although supplementation also varies by SES), another possible contributor to breastfeeding's protective effect. These findings support and extend the evidence on the protective role of breastfeeding in maternal health.

Cardiovascular and metabolic risk gradients by race-ethnicity and by poverty status varied directly with the high rates of overweight and obesity overall, and especially among African-Americans and Latinas. Substantial research supports these findings. More than one half of pregnant women are overweight or obese prior to pregnancy.[28] Similarly in our sample, the median BMI at one year postpartum was in the obese range for African-Americans and overweight for Latinas and Whites. Compared with weight gain at other times, postpartum weight retention is associated with long-term weight gain and increased risk for becoming or remaining overweight.[29] Excess postpartum weight retention tends to be preferentially deposited centrally, as visceral fat, and is associated with declines in HDL,[30] and production of pro- inflammatory cytokines. Obesity-related inflammatory changes may predispose a woman who becomes pregnant to adverse outcomes, including pre-eclampsia, diabetes and preterm delivery, as well as newborn complications.[31] Thus, consistent with the wear-and-tear hypothesis, postpartum weight retention contributes to obesity and a pro-inflammatory state, which may raise life course chronic disease risk.

Women with gestational diabetes or hypertension during pregnancy are at a higher risk of developing cardiovascular and metabolic diseases as they age.[32] A few women in our sample were already in treatment for diabetes or hypertension. We would expect treatment to lower AL scores and potentially reduce our observed disparities.

A history of pregnancy morbidity was generally associated with higher AL that was stable or worsened, suggesting their next fetus might be exposed to greater maternal cardiovascular and metabolic dysregulation. Early maternal cardiovascular disease and preterm birth may be related. Recent evidence shows that both maternal and fetal vascular resistances are higher in pregnancies that result in preterm delivery,[33] and that the fetus may be genetically programmed by exposure to this dysregulation, a potential contributor to life course risk of cardiovascular disease. [34]

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We compared the effects of prior pregnancies and found that despite pregnancy morbidities, parity was did not influence AL. This issue has not been adequately addressed in other studies.

Our findings are partly consistent with Gunderson et al's[31] prospective longitudinal study of lipid changes in African-Americans and Whites enrolled in CARDIA, as a function of parity. Whites had improvements in total cholesterol and LDL with later pregnancies but African-Americans did not. As opposed to our study, the CARDIA study did not include poverty status in their models and there were no Hispanic women.

Some of our study limitations include a lack of information about adverse health issues in women prior to current postpartum period. This limitation can be addressed through future studies of preconception influences on later maternal and child outcomes. We did not evaluate the relationship of obesity or central adiposity to cardiovascular and metabolic risk because these are part of the AL index, the outcome in our study design. However, we have shown the prevalence of significant postpartum weight retention in our sample, especially among African-Americans.[35] Although our cohort of postpartum women represented diverse socioeconomic and race/ethnic groups, this sample is not nationally representative; thus caution is needed in developing national priorities to improving health of pregnant and postpartum women. We have also not reported the effects of nativity and acculturation among the Latina women in this study. This complex topic needs to be studied in future research.

How persistent postpartum elevation of maternal AL could influence the next prenatal environment, affecting both generations simultaneously needs to be studied. Such knowledge could lead to interventions which can efficiently and effectively serve the double purpose of mitigating chronic disease risk for the mother and her future children. CCHN benefitted from community partnership processes and resources, as should future public policy initiatives and intervention design.

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Table 1.

Characteristics of CCHN Sample Mothers by Race/Ethnicity and Poverty Status

	African Americans (n=1,029)	Whites (n=429)	Latinas (n=450)	P-values Differences by race/ethnicity	Poor (n= 816)	Near Poor (n=529)	Not Poor (n=563)	P-values Differences by poverty
Baseline Demographics								
Age (mean±SD)	24.3 ± 5.0	29.7 ± 6.0	25.8 ± 5.2	<0.001	23.7 ± 4.3	24.9 ± 5.1	29.9 ± 5.9	<0.001
Poverty status (%)								
poor	53.5	19.8	40.0	<0.001	N/A	N/A	N/A	N/A
near poor	25.1	17.0	44.0		N/A	N/A	N/A	
not poor	21.4	63.2	16.0		N/A	N/A	N/A	
Education (%)								
< High school graduate	15.8	5.4	35.1	<0.001	26.5	20.8	3.2	<0.001
High school graduate	49.7	24.5	44.4		52.6	50.1	21.7	
Some college	26.0	23.1	12.4		16.7	23.3	29.0	
College graduate or more	6.4	44.1	5.1		1.6	4.5	42.8	
Other, No information	2.1	3.0	2.9		2.7	1.3	3.4	
Marital status (%)								
Married	13.9	67.6	40.5	<0.001	15.3	25.9	63.6	<0.001
Not married, in relationship	56.9	25.7	49.7		58.0	55.0	27.0	
Not married, not in relationship	29.2	6.7	9.8		26.7	19.1	9.4	
Pregnancy-Related Factors (%)								
Any prior pregnancy	50.6	52.2	67.3	<0.001	54.3	57.1	53.8	0.494
Gestational diabetes	4.6	5.0	6.5	0.313	4.5	5.4	5.8	0.540
Pre-eclampsia	11.3	3.5	4.2	<0.001	8.9	8.0	5.8	0.126
Infant Health and Feeding (%)								
Preterm (<37 wks)	12.1	11.7	12.1	0.971	11.6	10.1	14.3	0.101
Low Birthweight (<2500 gms)	12.4	8.0	7.3	0.007	10.6	9.3	10.1	0.778
Very Low Birthweight (<1500 gms)	2.3	0.9	1.9	0.297	1.6	1.6	2.7	0.349
Ever breastfed baby	59.5	79.8	88.5	<0.001	60.8	72.6	84.2	<0.001
Breastfeeding at 6 mo	51.2	73.1	63.9	<0.001	49.7	59.0	73.5	<0.001

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Table 2.

Biomarkers by Race/Ethinicity and Poverty Status, Time 2 and 3, Median (Mean±SD)

A. RACE/ ETHNICITY	***African Americans (n=1,029)****		***Whites (n=429)***		***Latinas (n=450)***		***Significant Differences*** (p<.05)***	
	Time 2	**Time 3**	**Time 2**	**Time 3**	**Time 2**	**Time 3**	**Time 2**	**Time 3**
BMI	29.5 (30.9±8.4)	30.0 (30.9±8.8)	26.0 (28.1±7.1)	25.7 (27.6±7.3)	28.0 (28.8±6.0)	27.7 (28.6±6.3)	AA>W,L	AA>W,L
Waist-hip ratio	0.86 (0.86±.09)	0.85 (0.86±.09)	0.86 (0.86±.07)	0.86 (0.86±.08)	0.89 (0.89±.07)	0.90 (0.89±.07)	L>AA,W	L>AA,W
Systolic BP	113.0 (113.3±12.0)	111.7 (112.7±12.5)	108.7 (109.2±11.5)	109.3 (109.3±10.7)	106.3 (107.4±10.4)	108.7 (109.5±10.9)	AA>W,L	AA>W,L
Diastolic BP	74.7 (75.7±10.4)	75.0 (75.1±10.3)	72.0 (72.7±9.0)	71.7 (72.1±9.2)	69.7 (70.1±8.3)	69.0 (70.3±9.0)	AA>W>L	AA>W,L
Pulse	78.0 (78.5±10.3)	78.7 (78.5±10.0)	74.3 (74.2±10.9)	74.7 (75.0±11.1)	73.8 (74.6±9.8)	74.7 (75.5±9.3)	AA>W,L	AA>W,L
HS-CRP	2.8 (4.2±3.9)	2.8 (4.3±4.3)	2.3 (3.7±3.6)	2.1 (3.8±4.2)	2.7 (4.0±3.5)	2.8 (4.4±4.3)		L>W
Hemoglobin Ale	5.3 (5.4±0.9)	5.3 (5.5±1.1)	5.0 (5.1±0.5)	5.1 (5.1±0.7)	5.4 (5.4±0.6)	5.4 (5.4±0.7)	AA,L>W	AA,L>W
HDLcholesterol	40.0 (42.5±14.6)	41.0 (43.3±14.4)	43.0 (45.3±15.9)	42.0 (43.6±14.3)	38.0 (40.6±13.9)	37.0 (40.1±13.6)	W>A A, L	AA,W>L
Total-HDLratio	4.2 (4.4±1.7)	3.9 (4.1±1.5)	4.3 (4.4±1.6)	3.9 (4.2±1.5)	4.3 (4.6±1.5)	4.2 (4.5±1.7)		L>AA,W
Cortisol slope	−0.02 (−0.02±.03) n=465	−0.01 (0.02±.02) n=435	−0.03 (−0.03±.03) n=262	−0.03 (−0.03±.02) n=209	−0.02 (−0.02±.02) n=206	−0.03 (−0.03±.02) n=188	W,L>AA	W,L>AA
B. POVERTY STATUS	***Poor (n=816)***		***Near Poor (n=529)***		***Not Poor (n=563)***		***Significant Differences (p<.05)***	
	Time 2	**Time 3**	**Time 2**	**Time 3**	**Time 2**	**Time 3**	**Time 2**	**Time 3**
BMI	29.0 (30.5±8.2)	29.3 (30.5±8.4)	28.9 (29.9±7.5)	28.6 (29.7±7.9)	27.0 (28.5±7.2)	26.0 (28.2±7.4)	Poor,Near>Not	Poor,Near>Not
Waist-hip ratio	0.88 (0.88±.09)	0.87 (0.87±.08)	0.87 (0.87±.07)	0.88 (0.88±.08)	0.86 (0.86±.08)	0.85 (0.85±.08)	Poor,Near>Not	Poor,Near>Not
Systolic BP	111.7 (111.3±11.6)	110.7 (111.2±12.2)	110.0 (111.0±12.2)	110.3 (111.2±12.0)	109.8 (110.4±11.6)	110.7 (111.1±11.4)		
Diastolic BP	73.7 (74.0±10.0)	73.7 (73.9±10.1)	71.7 (73.3±10.3)	71.0 (72.3±10.0)	73.0 (73.5±9.3)	73.3 (73.3±9.7)		Poor > Near
Pulse	78.0 (78.2±10.3)	77.7 (77.9±9.8)	76.7 (76.8±10.5)	76.3 (77.2±10.1)	73.7 (74.0±10.3)	75.3 (75.6±10.7)	Poor,Near>Not	Poor > Not
HS-CRP	2.8 (4.2±3.6)	2.9 (4.5±4.3)	2.8 (4.0±3.6)	2.9 (4.5±4.7)	2.3 (3.8±4.0)	2.1 (3.6±3.7)		Poor,Near>Not
Hemoglobin Ale	5.3 (5.3±0.7)	5.3 (5.4±0.9)	5.3 (5.4±0.7)	5.4 (5.4±0.8)	5.2 (5.3±0.8)	5.2 (5.3±1.0)		Poor,Near>Not
HDLcholesterol	40.0 (41.4±14.1)	39.0 (41.1±13.3)	40.0 (42.1±14.3)	41.0 (42.7±13.9)	40.2 (45.2± 16.1)	42.0 (44.6±15.6)	Not>Poor,Near	Not > Poor
Total-HDLratio	4.3 (4.5±1.6)	4.0 (4.4±1.6)	4.2 (4.5±1.5)	3.9 (4.1±1.5)	4.0 (4.4±1.7)	3.9 (4.2±1.6)		
Cortisol slope	−0.02 (−0.02±.03) n=368	−0.02 (−0.02±.02) n=336	−0.02 (−0.02±.03) n=261	−0.02 (−0.02±.02) n=231	−0.02 (−0.02±.02) n=304	−0.03 (−0.03±.02) n=265		Near,Not>Poor

*N's vary for specific biomarkers depending on the amount of missing data; see text for details. Sample sizes for most biomarkers were ~70% or more of the total race-specific n's atthe top of the columns. The exception was cortisol slope, for which n's are shown separately.

**Statastical significance determine by one-way ANOVA with multiplecomparisons based on t-tests for all biomarkers except those with skewed distributions (HS-CRP, Hemoglobin Ale, and HDL cholesterol), for which significancewas determined by nonparmetric one-way ANOVA with multiple comparisons based on Wilcoxon rank-sum tests.

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Table 3.

Subgrouped Mean Allostatic Load Scores by Race/Ethnicity and Poverty Status

A. RACE/ETHNIC COMPARISONS	*African Americans*			*Whites*			*Latinas*			*Significant Differences (P<05)*	
	6 mos	*12 mos*	–	*6 mos*	*12 mos*	–	*6 mos*	*12 mos*		*6 mos*	*12 mos*
FULL SAMPLE	*n=788*	*n=778*		*n=349*	*n=330*		*n=346*	*n=355*			
Top quartile allostatic load score	2.97	3.00		1.91	1.98		2.34	2.48		AA> L>W	AA>L> W
Clinical allostatic load score	3.40	3.34		2.40	2.29		2.84	2.97		AA> L>W	AA>L> W
SUBGROUPED BY BREASTFEEDING											
Ever Breastfed	*n=452*	*n=437*		*n=269*	*n=253*		*n=296*	*n=296*			
Top quartile allostatic load score	2.95	2.8		1.76	1.84		2.32	2.44		AA> L>W	AA>L> W
Clinical allostatic load score	3.38	3.17		2.27	2.11		2.84	2.96		AA> L>W	AA, L>W
No Breastfeeding	*n=292*	*n=283*		*n=72*	*n=64*		*n=39*	*n=36*			
Top quartile allostatic load score	2.98	3.26		2.53	2.66		2.56	2.79		None	None
Clinical allostatic load score	3.39	3.53		2.91	3.08		3.02	3.11		None	None
SUBGROUPED BY PREGNANCY COMORBIDITIES											
Gestational diabetes	*n=38*	*n=32*		*n=17*	*n=16*		*n=21*	*n=23*			
Top quartile allostatic load score	4.18	4.16		2.04	2.14		3.23	3.86		AA> W	AA>W
Clinical allostatic load score	4.74	4.50		2.66	2.40		3.77	4.49		AA> W	AA, L>W
No Gestational diabetes	*n=748*	*n=722*		*n=332*	*n=303*		*n=325*	*n=330*			
Top quartile allostatic load score	2.91	2.97		1.90	1.96		2.28	2.39		AA> L>W	AA>L> W
Clinical allostatic load score	3.33	3.31		2.38	2.27		2.78	2.87		AA> L>W	AA>L> W
Preeclampsia	*n=74*	*n=74*		*n=12*	*n=9*		*n=13*	*n=13*			
Top quartile allostatic load score	4.06	4.10		2.26	2.05		3.06	3.07		AA> W	AA>W
Clinical allostatic load score	4.47	4.42		2.86	2.67		3.70	3.65		AA> W	None
No Preeclampsia	*n=598*	*n=590*		*n=301*	*n=282*		*n=320*	*n=331*			
Top quartile allostatic load score	2.85	2.87		1.79	1.92		2.32	2.45		AA> L>W	AA>L> W
Clinical allostatic load score	3.28	3.23		2.29	2.21		2.82	2.93		AA> L>W	AA, L>W
SUBGROUPED BY INFANT BIRTH STATUS											

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Low birthweight	*n=80*	*n=69*		*n=21*	*n=19*		*n=21*	*n=24*			
Top quartile allostatic load score	3.26	3.50		1.53	2.29		2.93	2.97		A>W	None
Clinical allostatic load score	3.59	3.52		2.19	2.40		3.25	3.72		A>W	None
No low birthweight	*n=533*	*n=534*		*n=236*	*n=230*		*n=295*	*n=303*			
Top quartile allostatic load score	2.91	2.98		1.64	1.83		2.29	2.48		AA> L>W	AA>L> W
Clinical allostatic load score	3.37	3.38		2.14	2.19		2.80	2.97		AA> L>W	AA>L> W
Preterm birth	*n=89*	*n=82*		*n=41*	*n=32*		*n=44*	*n=39*			
Top quartile allostatic load score	3.15	3.44		1.50	1.60		2.01	2.34		AA> W, L	AA>W, L
Clinical allostatic load score	3.60	3.51		1.86	1.93		2.48	2.99		AA> W, L	AA>W
No Preterm birth	*n=603*	*n=604*		*n=282*	*n=271*		*n=298*	*n=313*			
Top quartile allostatic load score	2.96	2.91		1.89	1.97		2.39	2.50		AA> L>W	AA>L> W
Clinical allostatic load score	3.38	3.30		2.42	2.25		2.90	2.96		AA> L>W	AA>L> W
SUBGROUPED BY PRIOR PREGNANCY											
Prior pregnancy	*n=408*	*n=391*		*n=184*	*n=171*		*n=229*	*n=234*			
Top quartile allostatic load score	2.92	3.02		1.81	1.95		2.39	2.42		A>L> W	A>L>W
Clinical allostatic load score	3.35	3.36		2.28	2.20		2.91	2.95		A>L> W	A>L>W
No prior pregnancy	*n=380*	*n=387*		*n=165*	*n=159*		*n=117*	*n=121*			
Top quartile allostatic load score	3.03	2.97		2.02	2.02		2.22	2.60		A>L, W	AA, L>W
Clinical allostatic load score	3.45	3.31		2.53	2.39		2.71	3.00		A>L, W	AA, L>W
B. POVERTY COMPARISONS	***Poor***			***Near Poor***			***Not Poor***			***Significant Differences (p<.05)***	
	n=629	*n=628*	–	*n=410*	*n=400*	–	*n=444*	*n=435*		–	–
Top quartile allostatic load score	2.86	2.93		2.59	2.61		2.15	2.26		Poor, Near >Not	Poor>Near>Not
Clinical allostatic load score	3.31	3.27		3.06	3.06		2.61	2.59		Poor, Near >Not	Poor,Near >Not