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Authors
Slopen, Natalie
Chen, Ying
Priest, Naomi
et al.

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Emotional and instrumental support during childhood and biological dysregulation in mid-life

Natalie Slopen[a], **Ying Chen**[b], **Naomi Priest**[c,d], **Michelle A. Albert**[e], and **David R. Williams**[b,f]

[a]Department of Epidemiology and Biostatistics, University of Maryland College Park, School of Public Health

[b]Department of Social and Behavioral Sciences, Harvard H. T. Chan School of Public Health

[c]Center for Citizenship and Globalisation, Deakin University

[d]Melbourne School of Population and Global Health, University of Melbourne

[e]Division of Cardiovascular Medicine, Department of Medicine, University of California, San Francisco

[f]Department of African and African American Studies, Harvard University

Abstract

Objective—To determine whether greater emotional and instrumental support during childhood is associated with less dysregulation across multiple physiological systems in midlife.

Methods—Data are from participants in the second wave of the Midlife in the United States study (2004–2005) who participated in a clinic-based assessment of health status. Emotional and instrumental support was measured using a seven-item scale (α=0.89) based on participant retrospective self-report. Biological dysregulation was assessed using an allostatic load (AL) score constructed from 24 measures across seven physiological systems (N=1,236, aged 34–84 years).

Results—Emotional and instrumental support in childhood was associated with lower AL in a monotonic fashion: compared to individuals in the lowest quartile of support, respondents in the second, third, and fourth quartiles had −0.08 (standard deviation (SD)=0.08), −0.13 (SD=0.08) and −0.21 (SD=0.08) units lower AL, adjusting for age, sex, and race. This pattern was maintained after adjustment for reporting bias, childhood socioeconomic disadvantage, past-year depression, and physician-diagnosed cardiovascular disease or diabetes ($p<.01$). The inflammation and metabolic-lipid subscales showed the strongest associations.

The corresponding author is Natalie Slopen. Contact information is as follows: Natalie Slopen, ScD, Department of Epidemiology and Biostatistics, University of Maryland College Park, School of Public Health, 255 Campus Drive, College Park, MD 20742, nslopen@umd.edu Phone: (001) 301-405-6598.

Conflict of Interest: The authors declare there is no conflict of interest.

Conclusions—Greater emotional and instrumental support in childhood was associated with less biological dysregulation in mid-life, even after accounting for socioeconomic disadvantage in childhood and other potential confounders.

Keywords

allostatic load; physiological dysregulation; childhood; parental support; emotional support; instrumental support; life course

INTRODUCTION

A large and compelling body of research shows that individuals who experience adversity during childhood and adolescence face increased risk for a wide range of chronic diseases of aging (Johnson et al., 2013; Shonkoff et al., 2009). To date, less research has focused on protective factors during childhood that may promote good health or decrease susceptibility to chronic diseases later in life. Social support, which refers to the perception that one is cared for and can rely on others for assistance, is recognized as a determinant of morbidity and mortality (Berkman and Krishna, 2014; Uchino, 2009). In children and adolescents, support from the family and others is associated with positive psychological and behavioral outcomes (Resnick et al., 1997; Viner et al., 2012). However, we have limited evidence about whether the benefits from supportive relationships during childhood or adolescence extend to protect against adult chronic diseases of aging, and the specific biological processes that are influenced by supportive relationships early in life.

A few prospective studies show that feelings of warmth and closeness with parents (Russek and Schwartz, 1997) and parental academic involvement (i.e., a form of instrumental support) (Westerlund et al., 2013) predicts health-related outcomes in midlife including cardiovascular diseases, alcoholism, and allostatic load (AL) (i.e., a measure of cumulative dysregulation across physiological systems (McEwen B, 1993)). Other research has shown that positive parental relationships can buffer against the impact of low childhood socioeconomic status (SES) on pro-inflammatory signaling (Chen et al., 2011) and metabolic syndrome (Miller et al., 2011b) in adulthood. We are not aware of any prior studies that have examined the association between emotional and instrumental support during childhood and AL in midlife. We hypothesized that individuals with greater emotional and instrumental support would have lower AL, and that this relationship would be evident across physiological systems. Confirmation of this hypothesis could provide evidence to support increased attention to protective factors within childhood social environments for the primary prevention of adult diseases.

METHODS

Sample

Participants were men and women from the second wave of the Midlife in the United States (MIDUS) study. MIDUS was initiated in 1994–1995 to investigate the dynamics between social, psychological, behavioral factors and health, and enrolled 7,108 non-institutionalized individuals, aged from 25 to 74 years, from across 48 states through random digit dialing.

The sample included twin pairs and siblings (Brim et al., 2004). Among the original participants, 4963 (70%) individuals were followed-up at the second wave (2004–2005), and 592 African Americans from Milwaukee were recruited at this time (Radler and Ryff, 2010). Participants who completed the MIDUSII survey and were able to travel (N= 3,191) were invited to participate in a biomarker project, and 1255 agreed to participate. Participants stayed overnight at a research clinic. On Day 1, participants completed the medical history and physical exam, and the collection period for the 12 hour urine specimen began at 7 p.m. On Day 2, participants completed the 12 hour urine specimen collection (7 am) and provided a fasting blood specimen (Love et al., 2010). A comparison of these participants to the overall sample is detailed elsewhere (Dienberg Love et al., 2010).

Of the 1,255 participants, 13 had missing data on AL, 3 were missing information on childhood emotional and instrumental support, and 3 had missing data on covariates. Excluding participants with missing data yielded a sample of 1236, with 392 of the participants being siblings or twins. See Table A1 for comparison of included and excluded participants. Participants provided informed consent, and the study was approved by Institutional Review Boards at participating institutions.

Measures

Childhood emotional and instrumental support—Experiences of emotional and instrumental support during childhood and adolescence were retrospectively assessed with seven items from the Childhood Trauma Questionnaire (CTQ) (Bernstein and Fink, 1998; Bernstein et al., 1994) administered at the biomarker project (see Table A2 for items) which asked participants to reflect on experiences as child or teenager (no ages specified). These questions asked respondents to report on emotional and instrumental support from family as well as other people outside of the home. All response options ranged from 1 (never true) to 5 (very often true). Specifically, emotional support was measured using five items from the Emotional Neglect subscale that reflected positive experiences of nurturance and affection (e.g., family as source of strength; family members looked out for each other; α=.89). Instrumental support was measured using two items from the Physical Neglect subscale that assessed positive experiences of direct assistance (e.g., someone to take care and protect child; to take child to the doctor; α=.62). We combined the emotional and instrumental support items, and using factor analysis we established the presence of a single factor with good internal consistency reliability (α=.89). Responses were averaged to derive an overall score (range: 1 to 5). Quartiles were created such that the bottom quartile reflected low childhood emotional and instrumental support and the top quartile represented high support.

Allostatic load—AL, a multisystem dysregulation index, was calculated as the sum of risk scores across seven physiological systems including the sympathetic, the parasympathetic, the hypothalamic-pituitary adrenal axis, the inflammation system, the cardiovascular, the glucose metabolism, and the lipid metabolism. We operationalized AL following prior MIDUS studies (Chen et al., 2012; Gruenewald et al., 2012), and the biomarker indicators for each system are listed in Table S3. Details of the computation of AL are reported elsewhere (Chen et al., 2012; Gruenewald et al., 2012). The seven physiological systems included in the MIDUS AL score have substantial overlap with indices of cumulative

biological risk in other studies with different samples (Bird et al., 2010; Juster et al., 2010; Merkin et al., 2009). Furthermore, the selected indicators/systems have been shown to be associated with chronic disease (Cooney et al., 2009; Cooney et al., 2010; Danesh et al., 1998; de Koning et al., 2007; Muntner et al., 2005; Prospective Studies Collaboration, 2007; Stamler et al., 1993).

A risk score for each system was constructed as the proportion of biomarker indicators for that system that fell within the high risk quartile ranges. Consistent with prior MIDUS research, the seven physiological system risk scores were only calculated for participants with information on at least half of the system‘s biomarkers, and were scaled to range from 0 to 1. Specifically, the AL score was only calculated when we had information on at least one outcome in the SNS and the HPA systems (i.e., these two systems only included 2 markers), at least two outcomes in the cardiovascular, metabolic-glucose metabolism, and parasympathetic nervous system (i.e., these three systems included 3 or 4 markers), and on at least three outcomes in the metabolic-lipids and inflammation systems (i.e., these two systems included 5 markers). AL was computed by summing risk scores across all seven systems to create an overall score ranging from 0 to 7, with higher scores indicating greater risk. AL was only calculated for participants with data on at least six systems. A total of 144 participants lacked one or more biomarker for a specific system; this includes 119 participants who had AL calculated based on six instead of seven systems, and 25 participants whose AL was calculated based on all seven systems but had missing data on less than half of the biomarkers for a specific system.

Covariates

Childhood support reporting bias score: The Minimization/Denial subscale of the CTQ (Bernstein and Fink, 1998; Bernstein et al., 1994) is comprised of three items to assess tendency to exaggerate their reports of positive childhood experiences due to social desirability or other reasons (e.g., —I had the perfect childhood). Response options range from 1: never true to 5: very often true. The highest response (5) was scored as 1, and other responses were scored as 0. Items were summed to create an overall score (range: 0—3), with higher scores reflecting greater bias.

Childhood SES disadvantage score: Following prior MIDUS research (Gruenewald et al., 2012; Karlamangla et al., 2013; Tsenkova et al., 2014), a childhood socioeconomic disadvantage score was constructed by summing across three retrospectively-reported indicators of SES in participants‘ childhood and adolescence: family finances (worse off than others=2; same as average family=1; better off than others=0), highest parental education (less than high school=2; high school=1; college or more=0), and welfare for ≥6 months (ever=2; never=0).

Major depression: Past-year major depression was assessed using the Composite International Diagnostic Interview Short Form (Kessler et al., 1998), which is based on criteria specified in DSM-III-R (American Psychiatric Association, 1987). This measure has been validated and shows high test-retest reliability and criterion and construct validity (Aalto-Setala et al., 2002; Blazer et al., 1994).

History of cardiovascular diseases and diabetes: Medical history was queried as part of the in-depth clinical assessment. Participants who reported at least one of the following conditions were considered as having history of cardiovascular disease (CVD) and diabetes: physician diagnosed heart diseases, stroke or diabetes.

Demographics: Demographic covariates included participants‘ age at MIDUSII, sex, and race.

Statistical Analyses

All analyses were performed in SAS 9.3. Chi-square and analysis of variance tests were used to examine distribution of AL and covariates in the full analytic sample and across quartiles of childhood emotional and instrumental support.

To investigate whether higher levels of childhood emotional and instrumental support predicted lower AL in adulthood adjusting for covariates, generalized estimating equations (GEE) with identity link and normal distribution were used to model AL with quartiles of childhood emotional and instrumental support as the independent variable, accounting for family clustering. A series of GEE models were used to examine effect of potential confounding. The base model adjusted for demographic characteristics including age, sex, and race. The second model additionally controlled for the reporting bias score to account for participants‘ tendency to exaggerate reports of support due to social desirability or other reasons. In the third model, childhood SES was further added. Next, the model additionally adjusted for major depression. Last, we additionally included history of CVD and diabetes. Sensitivity analyses reanalyzed the primary sets of models with log link and normal distribution to account for the possibility that associations may not be linear. The primary models were also reanalyzed with log link and Poisson distribution given that AL may be considered as a count measure.

To estimate the effects on specific physiological systems, we modeled each of the seven physiological systems comprising AL with quartiles of childhood emotional and instrumental support as the independent variable. These exploratory models adjusted for age, gender, race, reporting bias and childhood socioeconomic disadvantage (we did not include depression or medical history in these models, as these variables could be on the causal pathway).

RESULTS

The participants were predominantly White (79.7%), and there were more females (56.5%) than males in the sample. The mean age was 54.5 years (standard deviation (SD)=11.73). The average emotional and instrumental support score was 4.18 (SD=0.82), and AL ranged from 0 to 5.03 (mean=1.75, SD=1.05). In bivariate analyses, AL showed a decreasing gradient across quartiles of support, but this pattern was not significant (p=0.35; see Table 1). In contrast, the mean inflammation and metabolic-lipid scores showed significant declines across quartiles of support (*p*-values <.05). Covariates of sex, reporting bias, childhood socioeconomic disadvantage, and major depression showed statistically significant patterning by quartile of support (*p*-values <.05).

In GEE models, emotional and instrumental support in childhood was associated with lower AL in a monotonic fashion: compared to individuals in the lowest quartile of support, respondents in the second, third, and fourth quartiles had −0.08 (SD=0.08), −0.13 (SD=0.08) and −0.21 (SD=0.08) units lower AL, adjusting for age, sex, and race (Table 2, Model 1). This pattern was maintained after adjustment for reporting bias, childhood socioeconomic disadvantage, past-year depression, and physician-diagnosed cardiovascular disease or diabetes (Table 2, Models 2–5).

In models to examine whether associations varied across individual physiological systems (adjusted for age, gender, race, reporting bias score and childhood disadvantage score), greater emotional and instrumental support in childhood was associated with lower scores on the inflammation and metabolic-lipid subscales (*p*-values for the top quartile of support <.05), but not with the five other physiological subsystem scores (see Table 3). Sensitivity analyses with alternative model specifications (i.e., a log link, and Poisson distribution) resulted in identical conclusions (see Tables A4 and A5).

DISCUSSION

In a population-based sample of adults in midlife, we found that AL was negatively associated with emotional and instrumental support during childhood. A decreasing graded pattern was maintained after adjustment for reporting bias, childhood socioeconomic disadvantage, depression, and physician-diagnosed cardiovascular disease or diabetes. Analysis of the seven physiological systems separately indicated the most pronounced patterns for the inflammation and metabolic-lipid subscales, thus suggesting that support in childhood may influence health in midlife through these systems most directly. Due to the cross-sectional nature of this study, we cannot make causal inferences about the relationship between support in childhood and biological dysregulation; however, these findings are consistent with prospective research that has found a protective effect of positive childhood family environment on later risk for chronic disease (Russek and Schwartz, 1997; Westerlund et al., 2013), as well as cross-sectional studies of retrospective report of parental warmth and chronic disease risk (Carroll et al., 2013; Miller et al., 2011b).

The results from this study provide an important extension to the expanding literature on adverse childhood experiences and poorer health in adulthood (Anda et al., 2006; Felitti et al., 1998) by suggesting that positive experiences in childhood also have enduring effects. Building on prior literature that has shown that maternal (or parental) support in particular is important, our measure may reflect support from other sources as well (i.e., it is non-specific). Researchers have made remarkable progress in developing biologically plausible models to link adverse childhood experiences to poorer mental and physical health outcomes (Miller et al., 2011a); moving forward, it will be important to evaluate whether positive childhood experiences function to protect health through similar or distinct social, behavioral, and biological (e.g., cellular, molecular, hormonal) pathways that lead to poorer health following childhood adversity. Parental support during childhood is associated with better early learning (Merlo et al., 2007) and long-term academic achievement (Cutrona et al., 1994), mental health (Stewart and Suldo, 2011), peer relationships (Benson et al., 2006), and less risk taking behavior (Schwartz et al., 2009); thus, it is seems likely that the

pathways that confer worse health following adverse childhood experiences may confer health advantages following emotional and instrumental support during childhood.

The present study has several limitations to consider. First, our measure of emotional and instrumental support was retrospective and self-report, which may have resulted in measurement inaccuracies. Second, the study participants are not representative of the U.S. national population, and thus generalizability of the results may be limited. Third, our results may be confounded by unmeasured factors during childhood that would predict both our exposure and outcome (e.g., shared genetics that would predict parenting and increased physiological dysregulation in adulthood); notably, our results were sustained after adjustment for childhood socioeconomic deprivation (i.e., an obvious potential confounder). In future studies, it would be ideal to adjust for prenatal and childhood factors that were not available in the MIDUS (e.g., mother‘s health during pregnancy, breast feeding duration, or physical activity or nutrition in childhood). Fourth, we were not able to disentangle family support from support obtained outside of the home. Fifth, it is possible that individuals with higher AL are biased towards less positive memories of childhood; however, at this time, we could not find evidence in the literature to support this form of bias. Finally, the seven subscales of the AL score share interrelated pathways; future research is needed to better refine which pathways are most important for measuring accelerated aging.

CONCLUSIONS

Our data suggest that emotional and instrumental support in childhood is associated with less biological dysregulation in midlife, even after accounting for socioeconomic disadvantage in childhood and several other potential confounders. Further research is needed in order to a) replicate this association using a prospective sample; b) examine potential social, behavioral, and biological pathways, including physical activity and nutrition in adulthood; c) consider aspects of the environment in childhood and beyond that could modify the observed association (e.g., school environment (Spriggs et al., 2009), neighborhood context in adulthood (Slopen et al., 2014b), and other features of the family environment); d) delineate specific effects based on source of support (e.g., parents, siblings, teachers/originating from inside or outside of the home); and e) establish whether specificity to the inflammation and metabolic-lipid subscales is driven by adiposity. The prevention literature shows that psychosocial interventions with families can lead to improvements in child behavior, hypothalamic pituitary adrenal axis activity, and parental attachment (Fisher et al., 2006; Leve et al., 2012; Slopen et al., 2014a), and can have a lasting impact on inflammatory outcomes in adolescence (Miller et al., 2014). If the present findings are substantiated by prospective studies, targeted efforts to promote positive social environments during childhood, and particularly, supportive child-parent relationships, should be considered for the primary prevention of adult chronic diseases.

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Abbreviations

AL	allostatic load
CTQ	Childhood Trauma Questionnaire
CVD	cardiovascular disease
GEE	generalized estimating equations
MIDUS	Midlife in the United States
SES	socioeconomic status
SD	standard deviation

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Highlights

- We examine report of support in childhood in relation to allostatic load in midlife
- Childhood emotional and instrumental support is associated with lower allostatic load
- The inflammation and metabolic-lipid subscales showed the strongest associations

Table 1

Distribution of participant characteristics according to level of childhood emotional and instrumental support: Midlife in the United States study, Wave II (2004–2005)

Characteristic	Full sample N=1236	Childhood emotional and instrumental support quartiles				*p*
		Low: 1 *n* = 319	2 *n* = 283	3 *n* =382	High:4 *n* = 252	
Allostatic load (SD)	1.75 (1.05)	1.82 (1.04)	1.76 (1.01)	1.72 (1.08)	1.67 (1.03)	0.35
Sympathetic subscale (SD)	0.23 (0.35)	0.23 (0.34)	0.23 (0.36)	0.24 (0.35)	0.23 (0.35)	0.92
Parasympathetic subscale (SD)	0.24 (0.36)	0.24 (0.36)	0.22 (0.35)	0.24 (0.36)	0.25 (0.37)	0.79
HPA axis subscale (SD)	0.24 (0.30)	0.23 (0.29)	0.25 (0.31)	0.24 (0.31)	0.23 (0.30)	0.80
Inflammation subscale (SD)	0.28 (0.27)	0.31 (0.28)	0.28 (0.26)	0.26 (0.26)	0.25 (0.25)	0.03
Cardiovascular subscale (SD)	0.25 (0.30)	0.28 (0.29)	0.25 (0.29)	0.24 (0.30)	0.26 (0.29)	0.38
Metabolic-glucose subscale (SD)	0.28 (0.34)	0.29 (0.34)	0.28 (0.35)	0.27 (0.34)	0.26 (0.34)	0.65
Metabolic-lipids subscale (SD)	0.25 (0.25)	0.27 (0.26)	0.27 (0.24)	0.25 (0.25)	0.21 (0.23)	0.02
Mean age (SD), years	54.53 (11.73)	53.12 (10.72)	54.94 (11.87)	54.94 (12.08)	55.25 (12.15)	0.09
Sex						0.0003
Male	43.53%	36.68%	52.65%	45.81%	38.49%	
Female	56.47%	63.32%	47.35%	54.19%	62.51%	
Race						0.51
White	79.69%	76.18%	81.63%	81.41%	79.37%	
Black	17.31%	20.06%	15.55%	15.45%	18.65%	
Other	2.99%	3.76%	2.83%	3.14%	1.98%	
Reporting bias score (SD)	0.54 (0.91)	0.08 (0.30)	0.19 (0.48)	0.58 (0.86)	1.45 (1.14)	<0.001
Childhood disadvantage score (SD)	1.92 (1.45)	2.46 (1.66)	1.91 (1.43)	1.70 (1.27)	1.57 (1.23)	<0.001
Major depression	12.06%	22.57%	10.95%	7.33%	7.14%	<0.001
History of heart diseases or diabetes	23.46%	20.38%	28.62%	21.20%	25.00%	0.06

Note: Percentages refer to the proportion of individuals within each support category with that characteristic. *p* value is derived from χ^2 or analysis of variance tests.

Table 2

Parameter estimates (standard error) for the association between childhood emotional and instrumental support quartiles and allostatic load (N = 1236): Midlife in the United States study, Wave II (2004–2005)

Childhood emotional and instrumental support quartiles	Model 1 : base model [a]	Model 2: adjusted for reporting bias	Model 3: additionally adjusted for childhood disadvantage score	Model 4: additionally adjusted for depression	Model 5: additionally adjusted for medical history
1: bottom quartile	Reference	Reference	Reference	Reference	Reference
2	−0.08 (0.08)	−0.08 (0.08)	−0.06 (0.08)	−0.04 (0.08)	−0.08 (0.08)
3	−0.13 (0.08)~	−0.16 (0.08)*	−0.12 (0.08)	−0.10 (0.08)	−0.11 (0.08)
4: top quartile	−0.21 (0.08)**	−0.28 (0.10)**	−0.25 (0.10)**	−0.23 (0.10)*	−0.24 (0.10)**

Note: Generalized estimating equations with identity link and normal distribution were used in all models to adjust for clustering by family and were calculated using SAS PROC GENMOD.

[a] The base model was adjusted for age, gender, and race.

** $p \leq .01$,

* $p \leq .05$,

~ $p \leq .10$

Table 3

Parameter estimates (standard error) for the association between childhood emotional and instrumental support quartiles and allostatic load subscales: Midlife in the United States study, Wave II (2004–2005)

		Childhood emotional and instrumental support quartiles			
	N	**Low: 1**	**2**	**3**	**High:4**
Sympathetic subscale	1222	Reference	0.02 (0.03)	0.02 (0.03)	−0.02 (0.03)
Parasympathetic subscale	1139	Reference	−0.05 (0.03)	−0.02 (0.03)	−0.01 (0.04)
HPA axis subscale	1236	Reference	0.03 (0.02)	−0.00 (0.02)	−0.04 (0.03)
Inflammation subscale	1236	Reference	−0.02 (0.02)	−0.05 (0.02)*	−0.09 (0.03)**
Cardiovascular subscale	1236	Reference	−0.02 (0.02)	−0.04 (0.02)~	−0.04 (0.03)
Metabolic-glucose subscale	1230	Reference	−0.01 (0.03)	−0.01 (0.03)	−0.02 (0.03)
Metabolic-lipids subscale	1234	Reference	−0.02 (0.02)	−0.03 (0.02)	−0.05 (0.02)*

Note: Generalized estimating equations with identity link and normal distribution were used in all models to adjust for clustering by family and were calculated using SAS PROC GENMOD. All models adjusted for age, gender, race, reporting bias score and childhood disadvantage score.

** $p \leq .01$,

* $p \leq .05$,

~ $p \leq .10$.

See Table S5 for the component indicators for each subscale.

Table A1

Comparison of participant characteristics for those included and excluded from the analysis: Midlife in the United States study, Wave II (2004–2005)

Characteristic	Included n=1236	Excluded n=19	*p*
Allostatic load (SD)	1.75 (1.05)	2.62 (1.12)	0.04
Childhood emotional and instrumental support (SD)	4.18 (0.82)	3.90 (1.16)	0.18
Mean age (SD), years	54.53 (11.73)	53.89 (11.51)	0.81
Sex			0.05
Male	43.53%	21.05%	
Female	56.47%	78.95%	
Race			0.01
White	79.69%	52.94%	
Black	17.31%	35.29%	
Other	2.99%	11.76%	
Reporting bias score (SD)	0.54 (0.91)	0.69 (0.95)	0.52
Childhood disadvantage score (SD)	1.92 (1.45)	1.78 (1.44)	0.68
Major depression	12.06%	15.79%	0.62
History of heart diseases or diabetes	23.46%	36.84%	0.17

Note: Percentages refer to the proportion of individuals within each inclusion category with that characteristic. *p* values were calculated using χ^2 or t-tests.

Table A2

Measurement of childhood emotional and instrumental support: Midlife in the United States study, Wave II (2004–2005)

	N	Mean	Range
Q1. There was someone in my family who helped me feel that I was important or special.	1235	4.05	1.00 – 5.00
Q2. I felt loved.	1233	4.28	1.00 – 5.00
Q3. People in my family looked out for each other.	1234	4.05	1.00 – 5.00
Q4. People in my family felt close to each other.	1226	3.87	1.00 – 5.00
Q5. My family was a source of strength and support.	1234	3.99	1.00 – 5.00
Q6. I knew that there was someone to take care of me and protect me.	1236	4.44	1.00 – 5.00
Q7. There was someone to take me to the doctor if I needed it.	1234	4.56	1.00 – 5.00

Note: higher score reflects higher level of support.

Table A3

Physiological systems, representative biomarkers, and high risk cut-points used in allostatic load score: Midlife in the United States study, Wave II (2004–2005)

	High Risk Cut-Points[1]
1. Cardiovascular	
Resting SBP (mmHg)	≥143.00
Resting DBP (mmHg)	≥82.00
Resting heart rate (bpm)	≥77.00
2. Metabolic–lipids	
Body mass index (kg/m^2)	≥32.31
Waist to hip ratio	>0.97
Triglycerides (mg/dL)	≥160.00
HDL Cholesterol (mg/dL)	≤41.37
LDL Cholesterol (mg/dL)	≥128.00
3. Metabolic - glucose metabolism	
Glycosylated hemoglobin (HbA1c)	≥6.10
Fasting glucose (mg/dL)	≥105
Insulin resistance (HOMA-IR)	≥4.05
4. Inflammation	
C-Reactive protein (mg/L)	≥3.18
Interleukin 6 (pg/mL)	≥3.18
Fibrinogen (mg/dL)	≥390.00
sE-Selectin (ng/Ml)	≥50.58
Soluble intercellular adhesion molecule-1 (ng/Ml)	≥329.65
5. Sympathetic Nervous System	
Urine Epinephrine (ug/g creatine)	≥2.54
Urine Norepinephrine (ug/g creatine)	≥33.33
6. Hypothalamic Pituitary Adrenal Axis	
Urine Cortisol (ug/g creatine)	≥21.00
Blood DHEA-S (ug/dL)	≤51.00
7. Parasympathetic Nervous System	
Standard deviation of R-R intervals (msec)	≤23.54
Root mean square of successive difference	≤11.83
Low frequency spectral power	≤113.96
High frequency spectral power	≤54.16

[1] The high risk cut-points were defined as the top quartile for all biomarkers other than HDL cholesterol, DHEA-S and the 4 resting HRV variables (for these exceptions, high risk was defined as the bottom quartile). Risk scores for each system were constructed as the proportion of biomarkers within each system in the high risk quartile range (Gruenewald et al., 2012).

Table A4

Parameter estimates (standard error) for the association between childhood emotional and instrumental support quartiles and allostatic load (N = 1236): Midlife in the United States study, Wave II (2004–2005)

Childhood emotional and instrumental support quartiles	Model 1 : base model [a]	Model 2: adjust for reporting bias score	Model 3: additionally adjust for childhood disadvantage score	Model 4: additionally adjust for depression	Model 5: additionally adjust for medical history
1: bottom quartile	Reference	Reference	Reference	Reference	Reference
2	−0.05 (0.04)	−0.05 (0.04)	−0.03 (0.04)	−0.03 (0.04)	−0.05 (0.04)
3	−0.08 (0.04)~	−0.09 (0.05)*	−0.08 (0.05)~	−0.06 (0.05)	−0.07 (0.05)
4: top quartile	−0.13 (0.05)**	−0.17 (0.06)**	−0.15 (0.06)**	−0.13 (0.06)*	−0.14 (0.06)**

Note: Generalized estimating equations with log link and Poisson distributions were used in all models to adjust for clustering by family and were calculated using SAS PROC GENMOD.

[a] The base model was adjusted for age, gender, and race.

** $p \leq .01$,

* $p \leq .05$,

~ $p \leq .10$

Table A5

Parameter estimates (standard error) for the association between childhood emotional and instrumental support quartiles and allostatic load subscales: Midlife in the United States study, Wave II (2004–2005)

		Childhood emotional and instrumental support quartiles			
	N	**Low: 1**	**2**	**3**	**High:4**
Sympathetic subscale	1222	Reference	0.06 (0.12)	0.06 (0.12)	−0.07 (0.15)
Parasympathetic subscale	1139	Reference	−0.21 (0.13)	−0.11 (0.12)	−0.06 (0.15)
HPA axis subscale	1236	Reference	0.08 (0.10)	− 0.02 (0.10)	−0.16 (0.12)
Inflammation subscale	1236	Reference	−0.05 (0.07)	−0.17 (0.08)*	−0.32 (0.10)**
Cardiovascular subscale	1236	Reference	−0.10 (0.10)	−0.17 (0.09)*	−0.16 (0.11)
Metabolic-glucose subscale	1230	Reference	−0.06 (0.10)	−0.05 (0.10)	−0.09 (0.12)
Metabolic-lipids subscale	1234	Reference	−0.07 (0.07)	−0.10 (0.08)	−0.19 (0.10)*

Note: Generalized estimating equations with log link and Poisson distributions were used in all models to adjust for clustering by family and were calculated using SAS PROC GENMOD. All models adjusted for age, gender, race and childhood disadvantage score.

** $p \leq .01$,

* $p \leq .05$,

~ $p \leq .10$