Research Report

Psychological Science
XX(X) 1–5

DOI: 10.1177/0956797612441218
http://pss.sagepub.com

Childhood Poverty and Young Adults' Allostatic Load: The Mediating Role of Childhood Cumulative Risk Exposure

Gary W. Evans[1,2] and Pilyoung Kim[3]
[1]Department of Design and Environmental Analysis, Cornell University; [2]Department of Human Development, Cornell University; and [3]Department of Psychology, University of Denver

Abstract

Childhood poverty is linked to a host of physical and psychological disorders during childhood and later in life. In the study reported here, we showed that the proportion of childhood spent in poverty from birth to age 9 was linked to elevated allostatic load, a marker of chronic physiological stress, in 17-year-olds. Furthermore, this prospective longitudinal relationship was mediated by cumulative risk exposure at age 13. The greater the duration of early life spent in poverty, the greater the exposure to cumulative risk. This, in turn, leads to elevated allostatic load. Multiple psychological, biological, and neurological pathways likely account for the social patterning of psychological and physical disease.

Keywords

poverty, stress reactions

Received 8/16/11; Revision accepted 2/8/12

Early childhood poverty appears to set individuals on trajectories of physical (Adler & Rehkopf, 2008; Shonkoff, Boyce, & McEwen, 2009) and psychological disorder (Bradley & Corwyn, 2002; Grant et al., 2003). In the study reported here, we examined whether poverty from birth to age 9 predicted elevated allostatic load, a marker of chronic physiological stress, in 17-year-olds. Finding that it did, we then tested whether this poverty-related allostatic load was mediated by elevated cumulative risk exposure at age 13.

Poverty, Cumulative Risk, and Chronic Stress

Economic deprivation is linked to increased childhood morbidity (Chen, Matthews, & Boyce, 2002) and to elevated morbidity and mortality in adulthood (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Shonkoff et al., 2009). Associations between early-life socioeconomic status (SES) and adult health hold even after factoring in upward mobility during the life course. Health inequalities may be due in part to elevated stress in low-SES children (Matthews & Gallo, 2010; Shonkoff et al., 2009). Low-SES relative to middle-SES children manifest higher blood pressure (Chen et al., 2002) and elevated neuroendocrine stress hormones (Cohen et al., 2010).

Allostatic load is a prominent marker of chronic physiological stress. It reflects dysregulation across multiple physiological systems mobilized in response to enduring environmental demands (Ganzel, Morris, & Wethington, 2010; McEwen & Gianaros, 2011). When these mobilizations are prolonged over time, set points are altered such that basal levels become recalibrated. The elasticity of the system also changes, responding to challenges less robustly and recovering less efficiently (McEwen & Gianaros, 2011; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Lower-SES children (Worthman & Panter-Brick, 2008), adolescents (Evans & Schamberg, 2009) and adults (Seeman et al., 2008) have the highest levels of allostatic load.

Relations between childhood poverty and allostatic load may be mediated by cumulative risk exposure. Low-income children are exposed to greater cumulative risks than are middle-income children (Evans, 2004; Evans & English, 2002), and children exposed to higher cumulative risk manifest elevated allostatic load (Evans, 2003; Evans, Kim, Ting, Tesser, & Shanis, 2007).

Current Study

In the current study, we used a prospective longitudinal design to examine whether childhood deprivation was related to elevated allostatic load in young adulthood. We then tested

Corresponding Author:
Gary W. Evans, Cornell University, College of Human Ecology, Martha Van Rensselaer Hall, Ithaca, NY 14853
E-mail: gwe1@cornell.edu

whether childhood cumulative risk exposure can account for some of the ill effects of childhood poverty on subsequent allostatic load. Finally, we explored the developmental timing of poverty, risk exposure, and allostatic load. The effects of childhood risks associated with deprivation may become embedded in the organism early on and alter lifetime health trajectories. Alternatively, SES-related risks may continue to accumulate over the life course to affect adult health (Cohen et al., 2010; Shonkoff et al., 2009).

Method

Participants

The participants for this study were part of a longitudinal study of rural poverty and child development (Evans, 2003). The original sample (mean age = 9.18 years) was recruited in rural counties in upstate New York using records from public schools, the Cooperative Extension System of the U.S. Department of Agriculture, the federal Head Start program, subsidized housing, and other antipoverty programs. In this sample, 50% of the participants came from families that were at or below an income-to-needs ratio of 1, which is the U.S. federal poverty line. The income-to-needs ratio is an annually adjusted per-capita index. The other half of the sample at recruitment came from families with income-to-needs ratios two to four times the poverty line, which represents the level of most American families. Additional data were collected when subjects reached mean ages of 13.36 years (Wave 2) and 17.47 years (Wave 3). The final sample analyzed in the present study consisted of 173 participants (mean age = 17.40 years; 51% male, 49% female) out of 224 at Wave 3 who had complete information on poverty, allostatic load, and cumulative risk exposure.

Procedure

All data were collected in participants' homes by a pair of experimenters working independently with each participant and his or her mother. Only one child per household was tested. Each family's income-to-needs ratio was assessed at each wave of data collection. For each 6-month period of the participant's life, we also collected information on household composition and adult household members (job title, hours worked per week, and level of education). These two data sources were used to calculate the proportion of life from birth to age 9 that the individual had lived at or below the poverty line. We defined poverty in this manner because duration of poverty appears particularly critical in childhood development and because allostasis theory emphasizes that the duration of chronic environmental demands plays a significant role throughout the life course (McEwen & Gianaros, 2011).

Allostatic load was calculated to capture activity across a range of physiological response systems, including the cardiovascular system, hypothalamic-pituitary-adrenocortical axis, sympathetic medullary system, and metabolic system. To calculate resting blood pressure, we used a Dinamap PRO 100 (Critikon, Tampa, FL) to take automated readings every 2 min while subjects sat quietly. The mean of the second through seventh readings was used as the index of basal blood pressure (Kamarck et al., 1992). Urine was collected overnight, from 8 p.m. on the evening before the experimental protocol to 8 a.m. the following morning. Epinephrine and norepinephrine were assayed by high performance liquid chromatography with electrochemical detection (Riggin & Kissinger, 1977), and cortisol was measured with a radioimmune assay (Contreras, Hane, & Tyrrell, 1986). Body mass index (BMI) was calculated as kg/m^2. Allostatic load was calculated from 0 to 6 by summing the number of physiological parameters (resting diastolic and systolic blood pressure; overnight epinephrine, norepinephrine, and cortisol; and BMI) for which the participant scored above the median (0 = 1st to 50th percentile,1 = > 50th percentile). This additive model of allostatic load predicts morbidity and mortality end points better than singular components of allostatic load do and comparably with other aggregate indices of multiple physiological-system activity (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002; Seeman, McEwen, Rowe, & Singer, 2001). Separate allostatic-load calculations were made at Wave 1 (age 9) and Wave 3 (age 17).

Cumulative risk exposure was calculated at Wave 2 (age 13) by exposures to three physical risk factors (noise, crowding, housing problems) and three psychosocial risk factors (family turmoil, separation from family, exposure to violence). Noise was measured by a decibel meter over a 2-hr period in the home. Crowding was defined as the ratio of occupants to number of rooms in the home. Housing problems were assessed by trained raters on a standardized scale that included items regarding structural problems, poor maintenance, cleanliness and clutter, physical hazards, and poor climatic conditions (Evans, Wells, Chan, & Saltzman, 2000). Exposure to social risks was determined by combining mother's reports on the Life Events and Circumstances Checklist (Work, Cowen, Parker, & Wyman, 1990) and youth's reports on the Adolescent Perceived Events Scale (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). An event was counted a single time if it was reported by the youth, the mother, or both. Multiple yes/no items assessed each social risk factor. For each of the six individual risk factors, risk was coded dichotomously. A score of 1 was given if the youth's exposure was in the upper quartile for the entire sample's distribution of continuous-risk-factor exposure at Wave 2, and a score of 0 was assigned for all lower levels of exposure. Cumulative risk was then calculated from 0 to 6 by summing the six singular risk factors.

Results

Table 1 provides descriptive statistics about the variables as well as the zero-order correlations between them. The proportion of life lived in poverty to age 9 was positively correlated

Table 1. Descriptive Statistics and Zero-Order Correlations Among Study Variables

Variable	*M*	Correlations			
		1	2	3	4
1. Proportion of life lived in poverty from birth to 9 years	.38 (.48)	—	.21**	.20**	.49**
2. Allostatic load at 17 years[a]	2.93 (1.47)		—	.26**	.25**
3. Allostatic load at 9 years[a]	2.86 (1.58)			—	.19*
4. Cumulative risk at 13 years[a]	1.45 (1.35)				—

Note: Standard deviations are given in parentheses.
[a]The possible range for these scores was 0 to 6 (see the text for details).
*$p < .05$. **$p < .01$.

with allostatic load at age 9, as well as with allostatic load at age 17. The putative mediator, cumulative risk exposure at age 13, was also significantly associated with duration of childhood poverty as well as with both indices of allostatic load.

The primary mediation test took advantage of the prospective longitudinal design of the data set by incorporating into the model allostatic load at Wave 1, when the youths were approximately 9 years of age. The prospective longitudinal relationship between the proportion of life lived in poverty from birth to age 9 and allostatic load at age 17 was significant ($b = 0.482$, $SE = 0.223$, $p < .03$). The elevation in allostatic load between ages 9 and 17 was greater the longer children lived in poverty from birth to age 9. The addition of cumulative risk exposure at Wave 2 shrank the beta weight for poverty 47% from 0.482 to 0.244 ($SE = 0.253$), which is no longer significant (95% confidence interval = [0.042, 0.502]; Preacher & Hayes, 2008). The significant prospective longitudinal relationship between childhood poverty and increased allostatic load was mediated by cumulative risk exposure (Table 2).

We also replicated the mediation analysis without controlling for prior allostatic load at Wave 1. The prospective relationship between the proportion of life lived in poverty from birth until Wave 1 (age 9) and allostatic load at Wave 3 (age 17) was significant ($b = 0.535$, $SE = 0.214$, $p < .01$), but the relationship became nonsignificant when Wave 2 (age 13) cumulative risk exposure was included in the model ($b = 0.323$, $SE = 0.256$).

Although our results suggest that childhood poverty leads to elevated allostatic load later in life, and this appears to be caused in part by increased risk exposure during childhood, an alternative explanation is tenable. Perhaps childhood poverty leads to continued elevations in cumulative risk exposure throughout life, including during early adulthood. Thus, elevated allostatic load at age 17 might reflect continued higher levels of cumulative risk at age 17 rather than prior exposure to elevated risks during childhood. To evaluate this alternative explanation, we repeated the original prospective longitudinal analysis (see Table 2), adding in a second mediator, cumulative risk exposure at age 17, to see whether it incrementally increased the predictive accuracy of the model beyond the predictive accuracy of age 13 cumulative risk exposure. It did not. Moreover, cumulative risk exposure at age 13 remained a significant mediator of the prospective longitudinal relationship between childhood poverty and young adult allostatic load

Table 2. Results of the Prospective Longitudinal Mediation Analysis

Model and variable	*b*[a]	β	R^2	*F*
Model 1				
Allostatic load at 9 years	0.17 (0.07)	0.19**	.05	$F(1, 183) = 9.65$***
Proportion of life lived in poverty from birth to 9 years	0.48 (0.22)	0.16*	.07	$F(2, 182) = 7.26$***
Model 2				
Allostatic load at 9 years	0.20 (0.07)	0.22**	.07	$F(1, 172) = 12.83$***
Cumulative risk exposure at 13 years	0.18 (0.09)	0.16*	.11	$F(2, 171) = 10.51$***
Proportion of life lived in poverty from birth to 9 years	0.26 (0.25)	0.08	.12	$F(3, 170) = 7.35$***

[a]Standard deviations are given in parentheses.
*$p < .05$. **$p < .01$. ***$p < .001$.

when cumulative risk at age 17 was added to the model. This pattern of results lends support to the idea that exposure to poverty early in life has a pervasive and long-term effect, setting one on a trajectory for elevated allostatic load throughout the life course, irrespective of subsequent risk exposure.

Discussion

Early life experiences of material deprivation are harmful for children. Elevated chronic physiological stress may help explain why childhood poverty is bad for human development. The objective of the present study was to verify in a prospective longitudinal analysis the hypothesized link between childhood poverty and elevated allostatic load among young adults. We also investigated whether the expected link between poverty and allostatic load was mediated by cumulative risk exposure during childhood. Finally, we explored the developmental timing of childhood poverty, risk exposure, and allostatic load as an index of chronic stress.

As indicated in Table 2, the greater the proportion of childhood from birth to age 9 spent below the poverty line, the higher the increase in allostatic load at age 17. This result appears to be robust, as it includes a prior index of allostatic load at age 9 and replicates without the prior allostatic-load term in the model. These results fit with prior work indicating that low-SES children and youth (Evans & Schamberg, 2009; Worthman & Panter-Brick, 2008) as well as adults (Seeman et al., 2008) evidence heightened levels of allostatic load. The data reported here are novel in three ways. First, they show that childhood deprivation is related prospectively to elevated allostatic load in emerging adults. Second, they reveal that cumulative risk exposure in childhood accounts for some of the effects of childhood poverty on allostatic load in young adulthood. Finally, childhood poverty appears to have a pervasive and long-lasting effect, and this produces elevated allostatic load in young adults irrespective of their concurrent risk exposure.

The longer the duration of childhood spent living in poverty, the greater the subsequent exposure to cumulative risks. Lower-SES children are more likely to face psychosocial and physical environmental risk factors than their more affluent counterparts are (Evans, 2004). This elevated cumulative risk exposure, in turn, appears to mediate the prospective relationship between childhood poverty and elevated allostatic load in emerging adults. These data fit with prior cross-sectional evidence of elevated cumulative risk exposure among lower- relative to higher-SES children and youth (Evans & English, 2002) and with evidence suggesting that cumulative risk exposure is positively correlated with allostatic load in children (Evans, 2003; Evans et al., 2007). Here, we showed for the first time that elevated cumulative risk exposure is a viable explanatory mechanism linking childhood poverty to subsequent chronic stress as children emerge into adulthood.

Another contribution of the present study is to call attention to the potential role of chronic physiological stress in understanding why childhood experiences of poverty are inimical to health and well-being. Prior research on explanatory mechanisms for poverty and SES, on the one hand, and child development, on the other, has focused on unresponsive and harsh parenting (Bradley & Corwyn, 2002; Conger & Donnellan, 2007; Grant et al., 2003) as well as diminished cognitive enrichment (Bradley & Corwyn, 2002; Duncan & Brooks-Gunn, 1997; Hoff, Laursen, & Tardif, 2002). Elevated allostatic load, a marker of chronic physiological stress, is a complementary physiological mechanism that may also help explain how and why childhood disadvantage is harmful to human development.

The primary limitation of our study is its observational design. Although the results were derived from a prospective longitudinal analysis, it is conceivable that some variable other than childhood poverty drove the findings. We ran two additional sets of analyses to examine the plausibility of this alternative explanation. First, we added various personal characteristics as covariates to the analyses. These characteristics included maternal education, maternal mental health, maternal stress, and single-parent status, as well as the participant's birth weight, gender, ethnicity, and temperament. We found that adding these characteristics resulted in only marginal reductions in explained variance. We also reversed the order of terms in the mediation equation, examining whether allostatic load mediated the link between poverty and cumulative risk exposure—it did not. If one or more "third" variables were responsible for the pattern of results, then reversing the order of terms in the hierarchical regression equation would have yielded the same pattern of data. Ideally, an experimental intervention that altered exposures to cumulative risks could be evaluated in terms of chronic stress outcomes in relation to early experiences of poverty. Another valuable extension of this study would be to examine coping resources in concert with the environmental demands accompanying poverty. It is likely that the ecological context of poverty reflects not only higher levels of cumulative risk exposure but also diminished coping resources (Compas et al., 2001; Matthews & Gallo, 2010; Wadsworth & Berger, 2006).

Psychological well-being, cognitive skills, and physical morbidity all reveal a social gradient beginning in childhood and persisting throughout the life course. Multiple psychological, biological, and neurological pathways likely account for the social patterning of psychological and physical disease. Psychological science has much to offer in unraveling how and why lifelong socioemotional, cognitive, and health inequalities result from early experiences of childhood deprivation.

Acknowledgments

We are grateful to the families who participated throughout this research. We thank Jana Cooperman, Kim English, Missy Globerman, Matt Kleinman, Rebecca Kurland, Melissa Medoway, Tina Merilees, Chanelle Richards, Adam Rohksar, and Amy Schreier for their assistance with data collection.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Funding

This work was supported by the William T. Grant Foundation and the John D. and Catherine T. MacArthur Foundation Network for Socioeconomic Status and Health.

References

Adler, N. E., & Rehkopf, D. H. (2008). U.S. disparities in health: Descriptions, causes, and mechanisms. *Annual Review of Public Health*, *29*, 235–252.

Bradley, R. H., & Corwyn, R. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*, 371–399.

Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin*, *128*, 295–329.

Cohen, S., Janicki-Deverts, D., Chen, E., & Matthews, K. A. (2010). Childhood socioeconomic status and adult health. *Annals of the New York Academy of Sciences*, *1186*, 37–55.

Compas, B. E., Connor-Smith, J., Saltzman, H., Thomsen, A., & Wadsworth, M. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin*, *127*, 87–127.

Conger, R. D., & Donnellan, M. B. (2007). An interactionist perspective on the socioeconomic context of human development. *Annual Review of Psychology*, *58*, 175–199.

Contreras, L. N., Hane, S., & Tyrrell, J. B. (1986). Urinary cortisol in the assessment of pituitary-adrenal function: Utility of 24-hour and spot determinations. *Journal of Clinical Endocrinology & Metabolism*, *62*, 965–969.

Duncan, G., & Brooks-Gunn, J. (1997). *The consequences of growing up poor*. New York, NY: Russell Sage Foundation.

Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*, *39*, 924–933.

Evans, G. W. (2004). The environment of childhood poverty. *American Psychologist*, *59*, 77–92.

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*, *73*, 1238–1248.

Evans, G. W., Kim, P. K., Ting, A. H., Tesser, H. B., & Shanis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, *43*, 341–351.

Evans, G. W., & Schamberg, M. A. (2009). Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences, USA*, *106*, 6545–6549.

Evans, G. W., Wells, N. M., Chan, E., & Saltzman, H. (2000). Housing and mental health. *Journal of Consulting and Clinical Psychology*, *68*, 526–530.

Ganzel, B. L., Morris, P. A., & Wethington, E. (2010). Allostasis and the human brain: Integrating models of stress from the social and life sciences. *Psychological Review*, *117*, 134–174.

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, *129*, 447–466.

Hoff, E., Laursen, B., & Tardif, T. (2002). Socioeconomic status and parenting. In M. H. Bornstein (Ed.), *Handbook of parenting* (2nd ed., pp. 231–252). Mahwah, NJ: Erlbaum.

Kamarck, T. W., Jennings, J. R., Debski, T. T., Glicksman-Weiss, E., Johnson, P. S., Eddy, M. J., & Manuck, S. B. (1992). Reliable measures of behaviorally evoked cardiovascular reactivity from a PC-based test battery. *Psychophysiology*, *29*, 17–28.

Karlamangla, A. S., Singer, B. H., McEwen, B. S., Rowe, J. W., & Seeman, T. E. (2002). Allostatic load as a predictor of functional decline: MacArthur studies of successful aging. *Journal of Clinical Epidemiology*, *55*, 696–710.

Matthews, K. A., & Gallo, L. C. (2010). Psychological perspectives on pathways linking socioeconomic status and physical health. *Annual Review of Psychology*, *61*, 501–530.

McEwen, B. S., & Gianaros, P. J. (2011). Stress- and allostasis-induced brain plasticity. *Annual Review of Medicine*, *62*, 51–66.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*, 879–891.

Riggin, R., & Kissinger, P. (1977). Determination of catecholamines in urine by reverse phase liquid chromatography with electrochemical detection. *Analytic Chemistry*, *49*, 2109–2111.

Seeman, T. E., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Annals of the New York Academy of Sciences*, *1186*, 223–239.

Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences, USA*, *98*, 4770–4775.

Seeman, T. E., Merkin, S. S., Crimmins, E., Koretz, B., Charette, S., & Karlamangla, A. (2008). Education, income and ethnic differences in cumulative biological risk profiles in a national sample of US adults: NHANES III (1988–1994). *Social Science & Medicine*, *66*, 72–87.

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association*, *301*, 2252–2259.

Wadsworth, M. E., & Berger, L. E. (2006). Adolescent coping with poverty-related family stress: Prospective predictors of coping and psychological symptoms. *Journal of Youth and Adolescence*, *35*, 57–70.

Work, W., Cowen, E., Parker, G., & Wyman, P. (1990). Stress resilient children in an urban setting. *The Journal of Primary Prevention*, *11*, 3–17.

Worthman, C. M., & Panter-Brick, C. (2008). Homeless street children in Nepal: Use of allostatic load to assess the burden of childhood adversity. *Development and Psychopathology*, *20*, 233–255.