Childhood poverty and adult psychological well-being

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Childhood disadvantage has repeatedly been linked to adult physical morbidity and mortality. We show in a prospective, longitudinal design that childhood poverty predicts multimethodological indices of adult (24 y of age) psychological well-being while holding constant similar childhood outcomes assessed at age 9. Adults from low-income families manifest more allostatic load, an index of chronic physiological stress, higher levels of externalizing symptoms (e.g., aggression) but not internalizing symptoms (e.g., depression), and more helplessness behaviors. In addition, childhood poverty predicts deficits in adult short-term spatial memory.

childhood disadvantage | psychological health | stress | helplessness | memory

An increasingly large body of work in public health and medicine reveals that childhood disadvantage is prospectively linked to adult physical morbidity and mortality (1–4). Moreover, early disadvantage may shape the long-term health trajectories of individuals in ways that are difficult to dislodge by subsequent upward social mobility. The present study builds upon and extends this life-course research on childhood disadvantage and adult physical health in two respects. First, we investigate psychological rather than physical health outcomes among adults as a function of childhood poverty. We examine multimethodological indices of adult psychological adjustment including mental health symptoms, short-term memory, helplessness behavior, and allostatic load. Second, because we have measures of these constructs early in childhood, we conduct prospective, longitudinal analyses, testing whether adults from more disadvantaged backgrounds manifest lower levels of psychological well-being 15 y later, independently of childhood well-being assessed at age 9.

Thus, several measures of psychological well-being were investigated between ages 9 and 24 in relation to childhood poverty. The first of these, short-term memory, describes the number of bits of information individuals can hold in immediate memory. Short-term memory capacity is important because it influences the encoding of information into long-term memory and is associated with cognitive development among children (5, 6). Cross-sectional studies with elementary school children in two different samples reveal a positive association between household SES and short-term memory span (7, 8), and a field experiment shows that Mexican preschool children in households randomly receiving cash transfers of differing amounts, conditional on healthcare utilization and school attendance, yields a positive cash transfer dose–response for short-term memory (9). An important shortcoming in the poverty and memory literature is the paucity of longitudinal data, and no studies have investigated childhood SES and adult short-term memory.

An aspect of psychological health that has garnered little attention in the poverty literature is helplessness. Disadvantaged children face a plethora of uncontrollable psychosocial (e.g., family turmoil) and physical (e.g., substandard housing) stressors that may erode a sense of mastery or self-efficacy, rendering lower SES children vulnerable to helplessness (10). Considerable research shows that chronic exposure to uncontrollable stressors induces helplessness (11–14). A few studies reveal a positive association between poverty and helplessness during childhood (15, 16) and later in adolescence (16). For the first time, to our knowledge, we examine whether childhood disadvantage is related to helplessness in adulthood.

A voluminous literature documents elevated psychological distress concomitant with childhood disadvantage (17–21), but little of this work has investigated whether mental health inequalities carry forward into adulthood. Two studies indicate that early SES households can lead to elevated externalizing symptoms (e.g., aggression) in adulthood (22, 23). The life-course data on social inequalities in internalizing symptoms (e.g., depression) are more equivocal with mixed results (23, 24).

One of the prime, underlying mechanisms believed to account for physical health inequalities is chronic physiological stress (25–28). Increasingly scientists operationalize chronic physiological stress by allostatic load. Allostatic load reflects accumulated wear and tear on the body from repeated mobilization of physiological response systems to environmental demands (28–30). Allostatic load is assessed by the accumulation of biomarkers across multiple environmental response systems such as cardiovascular, sympathetic nervous system, hypothalamic pituitary adrenal axis (HPA) axis, and metabolic processes. Allostatic load has generated considerable interest in the health and life sciences because it predicts morbidity and mortality better than singular risk factors (28–31). Furthermore, childhood disadvantage is associated with greater allostatic load in children (25, 27, 28), but little is known about adult allostatic load in relation to childhood risk factors such as poverty.

Thus, in the present article, we evaluate whether childhood disadvantage predicts subsequent, adult psychological health. Specifically we test whether childhood poverty at age 9 prospectively predicts multimethodological indicators of psychological well-being at age 24, holding constant the same construct at age 9.

Results

Table 1 provides descriptive information and the zero-order correlation matrix for all of the major variables, including the wave 1 baseline measures and gender as a covariate. Multiple imputations were used to address missing values. With the exception of our measure of spatial short-term memory, all psychological dysregulation measures were assessed at age 9 at baseline and at age 24 within the same individual.

As shown in Table 1, income at age 9 is positively correlated with adult short-term memory and inversely related to helplessness

Significance

Childhood poverty in a prospective, longitudinal design is linked to deficits in adult memory; greater psychological distress, including a behavioral marker of helplessness; and elevated levels of chronic physiological stress. These findings extend prior cross-sectional data during childhood and are largely parallel to existing life course work on physical health sequelae of childhood poverty.

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Table 1. Descriptive data and zero order correlation matrix

Variables	*M*	*SD*	2	3	4	5	6	7	8	9	10	11
1. Gender, 49% male			–0.03	–0.06	0.03	–0.10	0.02	0.29**	0.03	–0.02	0.08	–0.15*
2. $Income_9$	1.67	1.10		0.15*	0.14*	0.12*	–0.12*	–0.11	–0.28**	–0.18**	–0.17**	–0.14*
3. Short-term $memory_{24}$	9.07	2.72			0.02	0.17**	0.01	–0.00	–0.20**	–0.08	–0.12*	–0.05
4. $Helpless_9$	0.51	0.29				0.14*	0.08	0.11	–0.04	0.09	–0.07	–0.06
5. $Helpless_{24}$	0.71	0.28					0.02	–0.03	–0.10	–0.07	–0.04	–0.02
6. $Internalizing_9$	0.43	0.34						0.15*	0.24**	0.12*	–0.06	0.02
7. $Internalizing_{24}$	0.36	0.27							–0.01	0.58**	–0.08	–0.01
8. $Externalizing_9$	0.27	0.35								0.11	0.05	0.04
9. $Externalizing_{24}$	0.36	0.23									0.01	0.13*
10. Allostat $load_9$	1.43	1.21										0.22**
11. Allostat $load_{24}$	1.60	1.35										

Subscripts refer to age of measure (i.e., 9 or 24 years).
*$P < 0.05$. **$P < 0.01$.

concurrently and subsequently at age 24. Childhood income is inversely related to externalizing symptoms at age 9 and later on at age 24, with a similar but weaker pattern for internalizing symptoms. Finally, income during childhood is negatively correlated with allostatic load during childhood and later on in adulthood.

Because we have longitudinal data on all of the outcome measures, except for short-term spatial memory, a series of prospective, longitudinal analyses was conducted predicting adult psychological outcomes from childhood poverty, holding constant each respective childhood measure. Each of these equations, other than spatial short-term memory, contains the childhood measure at age 9 as a covariate in the model. All of these analyses also included gender as an additional covariate. The prospective analysis with gender as a covariate found that income at age 9 was a significant predictor of adult short-term spatial memory $\beta = 0.15$, $b = 0.36$ (SE = 0.15), $P < 0.05$.

Adult helplessness behavior, assessed as the proportion of total time devoted to solving a challenging puzzle, was greater as a function of childhood poverty. The poorer the household of origin, the less time the adult persisted on the puzzle as he or she matured over the 15-y period between the ages of 9 and 24, $\beta = 0.14$, $b = 0.04$ (0.02), $P < 0.05$.

The prospective, longitudinal analysis of childhood income on adult internalizing symptoms such as anxiety and depression was not significant, $\beta = –0.07$, $b = –0.02$ (0.01), whereas the same analysis for externalizing symptoms was significant, $\beta = –0.16$, $b = –0.03$ (0.01), $P < 0.05$. The poorer the adult's childhood, the greater the levels of externalizing symptoms at age 24.

The final set of prospective, longitudinal analyses examined allostatic load, a marker of chronic physiological stress. Consistent with the prospective, longitudinal helplessness findings and those for externalizing symptoms, adults growing up in poor households showed higher levels of allostatic load, $\beta = –0.11$ $b = –0.14$ (0.07), $p < 0.05$. Allostatic load increases with maturation but does so more if the individual was born into a low-income household. Allostatic load (0–6) is the sum of 0 = no risk and 1 = risk, wherein risk is defined as the upper quartile of resting diastolic and systolic blood pressure; overnight urinary cortisol, epinephrine, and norepinephrine; and body mass index.

To assess the sensitivity of the results, the original regression models were also repeated with additional covariates including parental education, occupation, and single-parent status. As indicated in Table S1, the significant prospective effects of childhood income on adult psychological well-being were unchanged by the addition of these statistical controls.

Discussion

A robust epidemiological literature indicates that childhood disadvantage is linked to subsequent physical morbidity among adults (1–4). Although most of this literature is prospective only, the few prospective longitudinal studies with baseline, childhood disease markers in the models replicate the simpler designs reducing the plausibility of alternative explanations for the childhood disadvantage effects. Any confounding factor with disadvantage would have to influence the change in health but not the baseline health measure. We show herein that early childhood disadvantage is linked prospectively to long-term changes in multimethodological indices of psychological well-being in adulthood.

Adults growing up in poverty have diminished spatial short-term memory. This result comports with cross-sectional studies indicating that lower SES is associated with deficits in childhood short-term memory functioning (7–9). This is an important result because the ability to retain information in short-term memory is fundamental to a host of basic cognitive skills, including language and achievement. Furthermore, there is some evidence that spatial short-term memory functioning resides in the hippocampus, a subcortical region known to be highly sensitive to chronic stress exposure (26, 29, 30) and recently found to be associated with childhood poverty (32–34). One hypothesis worthy of further exploration is whether alterations in hippocampal structure or function associated with early experience of disadvantage might underlie deficits in spatial short-term memory associated with childhood poverty. It is also worth noting that the association between early poverty and spatial memory is the strongest of the four different outcomes. On average, the difference between a low- and middle-income childhood (i.e., income-to-needs ratio of 1 to 3) yields about three quarters of an additional item in short-term spatial memory. The greater correlation between allostatic load at age 9 relative to concurrent allostatic load and adult spatial memory also suggests early impacts of experience on memory development. Unfortunately, because we have no childhood assessment of memory, interpretation of the adult poverty-related deficits in spatial short-term memory is less rigorous compared with the remaining prospective, longitudinal data.

One of the consequences of long-term exposure to uncontrollable environmental demands is a diminished sense of mastery indicated by greater susceptibility to helplessness (11–14). A few childhood poverty studies have shown that both children and adolescents from more disadvantaged backgrounds manifest more helplessness (15, 16). Herein we show that adult helplessness is associated with childhood poverty. Adults growing up in poverty persist ~8% less relative to those from a middle-income background on challenging puzzles, and this tendency happens independently of similar behaviors at age 9. Given the importance of working hard and persistence in the face of challenge for multiple competencies in life, such shortcomings may prove prescient in signaling long-term challenges associated with childhood poverty.

A large and well-developed cross-sectional literature shows elevated psychological distress in relation to childhood disadvantage (17–21), but only a handful of studies have explored this topic over

the life course. Consistent with the child literature, we find a prospective, longitudinal relation between childhood poverty and externalizing symptoms among adults of about 0.06 on a 0–2 scale in comparison with adults from a middle-income background. However, we uncover little association with internalizing symptoms. This pattern of results matches two prior life-course studies on externalizing symptoms (22, 23) as well as null internalizing effects in one prior study (23) but not another (24).

We also show that childhood disadvantage predicts concurrent allostatic load, matching several prior studies (25) but indicating that more elevated levels of allostatic load occur over the life course as a function of childhood disadvantage. On a 0–6 range of allostatic load, movement from a poor to a middle-class childhood would reduce allostatic load in adulthood by about a third of a risk factor. Given the rapidly expanding evidence that elevated allostatic load marks accelerated aging (2, 30, 31), this prospective longitudinal evidence for social inequalities in allostatic load may prove fundamental in understanding how poverty gets under the skin and stays there.

An important question raised by prospective longitudinal findings as herein is whether the long-term sequelae of early poverty simply reflect a lifetime of disadvantage or if there is something particularly salient to early life experiences of disadvantage. Unfortunately, because of the high degree of collinearity of income across the sample over the 15-y period, we cannot meaningfully disentangle the developmental timing of poverty exposure and adult psychological well-being. Although many studies suggest the critical importance of childhood disadvantage for subsequent physical morbidity in adulthood (35), caution is warranted given the high degree of income collinearity typically found in US samples between childhood and adulthood.

Some additional limitations of the study design warrant brief comment. Although the study is prospective and longitudinal with multimethodological indicators of psychological well-being, the design is correlational and causal conclusions are not warranted. Selective attrition by income and use of maternal and participant reports of mental health across time may have produced overly conservative results. The sample is European American and rural and thus may not generalize to more heterogeneous or urban populations. On the other hand, there is a marked paucity of research on rural poverty and physical or mental health. Rural poverty tends to be more persistent and deeper than urban poverty, which likely has implications for impacts over the life course. Another limitation is the allostatic load index, which reflects the simple summation of a set of dichotomous indicators of dysfunction across multiple, biological systems. This crude index, which appears to reflect some aspects of chronic wear and tear on the body, likely does not fully capture the more dynamic and systematic interplay between multiple biological systems as they are challenged by chronic environmental demands (26–31).

Children growing up in poverty face a daunting array of physical (e.g., substandard housing) and psychosocial (e.g., family turmoil) risk exposures that far exceed those of their more advantaged peers (10). Herein we show that similar to the epidemiological literature on physical morbidity, childhood poverty is associated with multimethodological indices of behavioral adjustment problems.

Materials and Methods

Participants were 341 Caucasian adults (M = 23.53 y, 51% female), but there was selective attrition, with more low-income households leaving the sample over the 15-y period, $t(339) = 3.25$, $P < 0.001$. However, there was no selective attrition in relation to any of the outcome variables. Low-income families were oversampled at recruitment so that approximately half the sample was at or below the US poverty line, an income-to-needs ratio of 1.0. The other half of the sample was 2–4 times the poverty line, the income of the majority of American families. At baseline, the mean income-to-needs ratio of the sample was 1.67.

Data were collected in the home by two experimenters working independently with the child and mother at ages 9, 13, and 17 and then with the adult participant only at 24 y of age. All of the psychological health measures except for spatial short-term memory were collected at baseline and at age 24.

Short-term spatial memory was assessed by the adult participant's ability to recall a sequence of stimuli presented for 500 ms on a four quadrant touchpad with monochromatic lights and four unique, corresponding tones. The trial began with one signal, and after correctly repeating the initial lit quadrant, each successive trial added one more signal to the sequence. The participant's task was to repeat the sequence of lighted quadrants in the correct order on the touchpad. Participants completed a practice run to ensure task comprehension and then two experimental trials separated by 1 h. Short-term spatial memory is the mean of the maximum number of signals correctly recalled in sequence at each of the two experimental trials.

At baseline (age 9) and at wave 4 (age 24), participants were given a standard helplessness behavioral protocol. At age 9, the participant was given a paper-and-pencil puzzle and instructed to visit each animal on the paper by tracing over the interconnecting lines between the animals. The child was instructed to not lift his or her pencil or double back over any line. The child was informed that he or she could continue working on the puzzle by starting over on another sheet until solved or until he or she felt unable to solve the puzzle. At this point, the child could move onto a second similar puzzle. Once the child had ceased working on the initial puzzle and began the second puzzle stimulus, he or she could not return to work on puzzle 1. A practice puzzle was used to ensure task comprehension. Unbeknownst to the child, the initial puzzle was unsolvable. The index of helplessness is time of persistence on the first puzzle. All children solved the second puzzle and were assured that they did a good job and that most children find the first puzzle very difficult. If after 10 min the child continued to persist on the initial, unsolvable puzzle, he or she was instructed to move onto the second puzzle. An analogous procedure was used at age 24 with the puzzle game called Traffic Jam. In this version of the puzzle, the task was to rearrange cars on a plastic base by slotting them into different spaces so that the target car could be moved from the starting point to the exit of the traffic jam. For the adult protocol, the participant was given up to 15 min before moving onto the second, solvable puzzle. Because the maximum persistence time varied across waves of data collection, the proportion of total time working on the initial, unsolvable puzzle is used as the index of helplessness. This helplessness protocol has been associated with beliefs about personal control, experimental manipulations of control, and chronic exposure to uncontrollable stressors (11–14).

Mental health was measured at both waves with maternal ratings of internalizing symptoms ($\alpha = 0.64$) and externalizing symptoms ($\alpha = 0.74$) at age 9 (36) and at age 24 by participant self-rated (0, not true; 1, somewhat or sometimes true; 2, very true or often true) internalizing (e.g., "I feel lonely"; "I am nervous or tense"; $\alpha = 0.91$) and externalizing (e.g., "I argue a lot"; "I am too impatient"; $\alpha = 0.85$) symptoms (37). Both of these scales have undergone extensive psychometric development and been widely used across a large number of heterogeneous samples throughout the world (36, 37).

Allostatic load represents physiological activity across a range of response systems including cardiovascular, HPA axis, sympathetic adrenal medullary system, and metabolic activity. For each system, the participant is assigned a score of 0 (no risk) or 1 (risk), wherein risk is defined as scoring within the top quartile of values for each of the following parameters. Resting blood pressure was the mean of the second through seventh reading of repeated monitoring (Dinamap Model Pro-100, Critikon) every 2 min while the participant sat quietly. Overnight urine was collected from 8 AM on the evening of the experimental protocol to 8 AM the next morning. Epinephrine and norepinephrine were assayed with HPLC with electrochemical detection (38) and cortisol with a RIA (39). The neuroendocrine assays controlled for creatinine. Body mass index was calculated as kg/m^2. Allostatic load was calculated separately at ages 9 and 24. This simple, additive model of allostatic load across multiple response systems predicts morbidity and mortality better than singular components of allostatic load (26–31).

Children provided assent and their parents provided informed consent. This research was approved by the Cornell University institutional review board.

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