

Running Head: POV, LOAD, AND WORKING MEM

The Cost of Living in Poverty: Long-Term Effects of Allostatic Load on Working Memory

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Abstract: Compared to their middle-income peers, adolescents reared in poverty perform less accurately on neurocognitive tasks. Previous research has not delineated an underlying mechanism that would account for these deficits in cognition. Allostatic load is a likely contributor as physiological stress in the brain disrupts neurogenesis, decreases the connections between dendrites, and induces atrophy in areas essential for working memory. The interplay between poverty, allostatic load, and working memory has not been previously studied. The current investigation examines the influence of cumulative, physiological “wear and tear” (at ages 9 and 13) on working memory functioning (at age 17), in a low- and middle-income sample (N= 68 and 102 per group, respectively). As predicted, proportion of life spent in poverty was inversely related to working memory functioning, with decreased accuracy for individuals living in persistent poverty. This pathway was fully mediated by increased allostatic load in childhood and early adolescence. These findings have implications for future health and education policies, and have provided preliminary evidence for the role of allostatic load on neurocognitive functioning.

The Cost of Living in Poverty: Long-Term Effects of Allostatic Load on Working Memory

Over the past half century, the United States has witnessed a surge of economic growth. Issues of equity have also risen during this time period—the rich seem to be getting richer, while the poorest people appear to remain in a state of financial stagnation. Pervasive economic problems persist despite the fact that the federal government contributes 1.6 billion dollars, or 12% of its gross domestic product, to public assistance programs (such as Welfare, Medicaid, and food stamps). Currently, 36.5 million people in the United States live in poverty (DeNavas-Walt, Proctor, & Smith, 2007). The U.S. Census Bureau defines poverty according to family size and composition using a series of threshold values for income—“If the total income of a person’s family is less than the threshold appropriate for that family, then the person is considered poor, together with every member of his or her family” (U.S. Census Bureau, 2000, p. 1044). Out of approximately 72.5 million children living in the United States, more than thirteen million (17.9%) are currently living at or below the federal poverty line (DeNavas et al., 2007). These children comprise 35.2% of the entire nation’s poverty population. With such a substantial percentage of people under the age of 18 living in poor households, it is essential to understand how the environment influences health and human development. Poverty has deleterious effects on cognitive development, but the literature fails to explain *how* poverty relates to deficits in cognition. How might the environment of poverty manifest in the brain? This paper seeks to untangle the mechanisms underlying poverty and neurocognitive functioning by exploring chronic stress in the lives of poor and middle-class children.

Poverty and Cognitive Development: An Overview

Early childhood exposure to poverty is negatively associated with cognitive development (Heckman, 2006; Duncan & Brooks-Gunn, 1997; Duncan, Yeung, Brooks-Gunn & Smith, 1998;

McLoyd, 1998). Exceptionally poor children living in households that are 50% below the poverty threshold score six to thirteen points lower on standardized cognitive tests—measuring verbal ability, IQ, and achievement—than children with a family income 150-200% above the poverty line (Smith, Brooks-Gunn, & Klebanov, 1997). Using longitudinal data from the Infant Health and Development Program (IHDP), Duncan and colleagues (1994) compared low- and high-income families, and found substantially lower IQ scores amongst five-year-olds in persistent poverty. Even after controlling for maternal education, ethnicity, and family composition, adjusted mean IQ scores were roughly nine points lower for chronically impoverished children (Duncan, Brooks-Gunn, & Klebanov, 1994). This difference could affect the type of schooling a child receives, and specifically whether the child is included in a regular classroom (with other children of average or above average intelligence) or placed in special education setting.

Early influences of income may also lead to more pervasive developmental outcomes than later influences. For instance, for the first five years of life, a \$10,000 average increase in income results in nearly one additional year of completed school for low-income children (Duncan, Yeung, Brooks-Gunn, & Smith, 1998). More specifically, children were 2.8 times more likely to complete high school if the increase in income occurred before the children turned five years old. No similar benefit was found for increased family income when children were between 5-10 and 11-15 years old (Duncan et al., 1998). Evidence also suggests an intensification of income-related developmental differences during adolescence and adulthood (Duncan & Brooks-Gunn, 1997; Bradley & Corwyn, 2002). As children age, those growing up in poverty are exposed to an increasingly large number of stressful life events, compared to middle-income children. The achievement gap present in childhood widens over time between those who

are poor and those who are not poor, as a function of these cumulative stressors. From second through seventh grades, achievement scores for mathematical competency increased in children from middle and high-income families, and decreased in low-income children (Pungello, Kupersmidt, Burchinal, & Patterson, 1996).

In addition to the contribution of developmental timing, duration of poverty also moderates changes in cognition. Income measures assessed over multiple years, rather than over the course of twelve months better predict developmental outcomes (Blau, 1999). Korenman, Miller and Sjaastad (1995) used the National Longitudinal Survey of Youth (NLSY) to assess the effects of poverty exposure over time. Verbal, mathematical, and reading abilities were found to be 2-3 times lower for children living in long-term poverty as compared to those living in concurrent poverty (Korenman et al., 1995). In 2005, the National Institute of Child Health and Human Development (NICHD) investigated duration of poverty by targeting a diverse income sample across ten different locations in the United States. Six to nine year old children growing up in persistent poverty had the weakest performance on school readiness measures and language tests, compared to children who were never poor, as well as those experiencing transient poverty (NICHD, 2005).

Neurocognitive Correlates of Income and Socioeconomic Status

Despite well-documented cognitive correlates of income (and SES), only a handful of studies have looked at the possible underlying neurocognitive systems responsible for observed deficits. There is some evidence suggesting an association between poverty and decreases in neurocognitive performance—particularly with regard to tasks involving attention, cognitive control, and memory.

In 2004, Mezzacappa investigated the relationship between attention and socio-demographic differences in a sample of 249 children, ages four to seven years. The Attention Network Test (ANT) was used to explore alerting, orienting, and executive attention capabilities. Children were asked to complete a modified Flanker task; arrows bordering a center arrow either pointed in the same direction as a center arrow (congruent condition) or in the opposite direction (incongruent condition). Supplying an asterisk cue prior to some of the trials improved both accuracy and reaction time in the socially advantaged group, but not for children who were socially disadvantaged. On incongruent tasks, socially advantaged children were more proficient at ignoring the oppositely-positioned flankers—marginal increases in reaction time and slight decreases in accuracy were observed in the socially advantaged children compared with the socially disadvantaged children who experienced more profound difficulties. Mezzacappa (2004) concluded that socioeconomic status has an effect on fundamental processes of learning, which may affect global cognition (such as IQ), achievement on tests, and retention in grade level.

Noble, Norman, and Farah (2005) examined socioeconomic status (SES) as a predictor of multi-system neurocognitive functionality in sixty African American, kindergarten children. The sample was evenly distributed into low and middle SES groups, with thirty children per group. Low SES was based on three factors: parental educational attainment of less than or equal to high school; having a parent score between four and seven on the Hollingshead Occupational Status Scale (indicating a technical, clerical, or unskilled position); and an income-to-needs ratio that did not exceed 1.2, which is a cutoff slightly above the poverty threshold of an income-to-needs equal to 1. Conversely, the middle SES group was classified by at least one parent who had completed, at minimum, two years of college; having a parent score between one and four on the Hollingshead Scale; and an income-to-needs ratio of at least 1.5. Advances in cognitive

neuroscience (such as the advent of neuroimaging techniques) enabled the researchers to hypothesize the predictive value of SES on differences in both prefrontal/executive and perisylvian/language systems. Neuropsychological tasks based on anatomical and functional criteria were designed to delineate differences in neurocognition by taxing five brain systems: occipitotemporal/visual cognition, parietal/visuospatial processing, medial temporal/memory, perisylvian/language, and prefrontal/executive functioning. Subjects completed paper-based and computerized tasks during three separate, 30 minute sessions (for complete description of the experimental procedure, see Noble et al., 2005). As predicted, children from low SES families scored more poorly on tasks targeting the perisylvian/language and prefrontal/executive regions of the brain. Composite scores on all other neuropsychological tasks were unrelated to SES. Lastly, global measures (such as IQ) also differed in low and middle SES—a finding that is replicated in studies on income and cognition (e.g., Smith, Brooks-Gunn, & Klebanov, 1997; Duncan, Brooks-Gunn, & Klebanov, 1994).

In 2006, Farah and colleagues built upon the findings gathered by Noble et al. (2005) and refined their neurocognitive testing parameters. Sixty high and low SES children between the ages of 10 and 13 were given a more comprehensive battery of neurocognitive tests that specifically target the prefrontal cortex. Three neural processing systems were tested: the lateral prefrontal cortex/working memory, anterior cingulate/cognitive control, and ventromedial prefrontal cortex/processing of reward. Compared to the short-term memory tasks (e.g. test of recognition given immediately following incidental learning task) implemented in the 2005 sample by Noble and colleagues, Farah and her research team (2006) presented an incidental recognition task thirty minutes after initial viewing of the pictures. Level of SES was significantly related to disparities in memory, even when controlling for gender, age, and

ethnicity. A difference in performance level was illustrated between SES groups, with lower income children having the lowest accuracy. As expected, pervasive differences were also found in each of the three neural systems (Farah et al., 2006).

Allostasis: A Biological Pathway

Although these three studies illustrate linkages between SES and neurocognition, they do not address why or how this may occur. Trying to understand how this pathway might arise requires contextual understanding of the environment of poverty. Children of low-income families often arrive in the world with health vulnerabilities (Duncan & Brooks-Gunn, 1997). Compared to middle-income children, those from low-income households are 1.7 times more likely to have a low birth weight or die in childhood, 3.5 times more likely to develop lead poisoning, and twice as likely to spend a short amount of time in the hospital (Duncan & Brooks-Gunn, 1997). These and other early environmental and psychological stressors (such as chaotic households, increased noise, and violence) interrupt the balance of multiple bodily systems (Evans and Kim, 2007; Evans, 2003). Adaptation is therefore necessary to maintain physiological stability when environmental or behavioral states are demanding and variable. In 1988, Sterling and Eyer labeled this process *allostasis*. An older conceptualization of the stress response focused on the maintenance of physiological “set points” (Cannon, 1935; Selye, 1956, 1973). Unlike the constancy of Cannon’s homeostatic survival mechanism, *allostasis* is an attempt by the brain to readjust physiological parameters within neuroendocrine, autonomic nervous, metabolic, cardiovascular, and immune systems in order to meet the changing demands of the environment (Sterling & Eyer, 1988). Neuroendocrine hormones released from the hypothalamic-pituitary-adrenal (HPA) axis primarily mediate allostasis. Disruption in the balance of these primary mediators—with excessive secretion of some, but not others—results in

over- or under-active physiological systems. Koob and Lemoal (2001) named this process *allostatic state*. With respect to children growing up in poverty, negative outcomes in neurocognitive functioning might result from a chronically active allostatic state, known as *allostatic load*.

Allostatic Load and Brain Development

Pioneered by Bruce McEwen and his colleagues 15 years ago, *allostatic load* refers to the costs of trying to adapt to chronically stressful life events; biologically speaking, this process manifests as cumulative wear and tear on regulatory systems in the body (McEwen & Steller, 1993). Unpredictability in the environment requires immediate physiological response (allostasis) to promote adaptation. If a predator is about to attack, acute allostasis enhances functioning in multiple systems. When a situation is perceived as stressful, the brain responds by activating the hypothalamus to release corticotrophin-releasing factor (CRF), which in turns triggers the pituitary gland to release adrenocorticotropin hormone (ACTH) into the blood stream. Traveling peripherally, ACTH arrives in the adrenal glands and targets the release of glucocorticoids to enable fight or flight behavior. Short-term release of glucocorticoids promotes adaptive responses. In such circumstances, the immune system responds by increasing pathogen resistance; the cardiovascular system adjusts blood pressure, and redistributes blood toward muscles to help meet any physical demands; in a similar vein, glucocorticoids provide glucose to muscles and other organs to facilitate an escape plan, and also replenish energy reserves following activity (Dhabhar & McEwen, 1999; McEwen, 2004). Frequent arousal of these responses and dysregulated activity (e.g., failure to shut off or failure to adequately turn on) of the HPA axis, on the other hand, have negative consequences on multiple physiological systems (McEwen & Steller, 1993). Exposure to elevated hormones over time results in pathologies, such

as hypertension, atherosclerosis, hypercholesterolemia, resistance to insulin, and suppression of the immune system (Munck, Guyre, & Holbrook, 1984), as well as neurocognitive impairment (Lupien, Gillin, & Hauger, 1999; Lupien et al., 2006; Lupien, Maheu, Tu, Fiocco, & Shurmaek, 2007). Allostatic load thus illustrates cumulative health effects resulting from multi-system dysregulation.

As previously mentioned, the brain is particularly sensitive to the primary mediators of allostatic load (McEwen, 1998). Cortisol in humans and corticosterone in animals comprise the stress hormones released by the HPA axis. The amount of circulating glucocorticoids shifts throughout the diurnal cycle, with cortisol peaking in the AM hours and showing a slow decline throughout the afternoon and evening (Lupien, Maheu, Tu, Fiocco, Shurmaek 2007). Type I (mineralocorticoid) and Type II (glucocorticoids) receptors allow for binding of glucocorticoids, and vary by location and degree of uptake in the brain, with the former having high affinity in the hippocampus, and the latter specifically distributed in the prefrontal cortex (Lupien et al., 2007).

Most of the studies focusing on the effects of stress on cognition have particularly emphasized the hippocampus and changes in declarative memory (Lupien et al., 2006; McEwen, 2000; Sapolsky, 2004; Squire & Zola-Morgan, 1991; Squire, 1992). Adrenal steroid receptors in the CA1 and CA3 regions of the hippocampus are particularly sensitive to infused glucocorticoids (Sapolsky, 2004; McEwen, 2000). Populations with elevated cortisol levels, such as those with Cushing's disease and among people who experience chronic stress are also particularly susceptible to the effects of cortisol in the brain, and show cognitive deficits as a result (Lupien et al., 2006; Sapolsky, 2004; McEwen, 2000). Additionally, animal and human studies have revealed a correlation between chronically elevated glucocorticoids and decreased

volume of the hippocampus (Lupien, Maheu, Tu, Fiocco, Shymaek, 2007; McEwen, 2000), as well as impairments in neurogenesis and dendritic remodeling of hippocampal pyramidal neurons (Lupien, 2006). Furthermore, in their 2006 review, Kim and colleagues showed how persistent, early life stress leads to elevated glucocorticoids and disrupts neuronal growth in the hippocampus (Kim, Song, & Kosten, 2006).

Allostatic Load and the Prefrontal Cortex: Implications for Working Memory

Despite the wealth of literature implicating stress and the hippocampus, only a handful of studies have looked at the prefrontal cortex as a possible target for allostatic load. For instance, Sapolsky (2004) has shown how stress and glucocorticoids increases the release of neurotransmitters—dopamine and norepinephrine—in the prefrontal cortex, and disrupts frontal functioning via impaired dendritic signaling (distally) and increased noise in proximal dendrites (Sapolsky, 2004). A disruption of this nature has implications for working memory—a type of information storage that takes place in the prefrontal cortex. Specifically, working memory is the maintenance and activation of information over short periods of time (Pelosi, Slade, Blumhardt, & Sharma, 2000). Lupien and colleagues also tested the acute effects of glucocorticoids on neurocognitive processes, and found greater deficits in working/prefrontal cortex than declarative/hippocampal memory (Lupien, Gillin, & Hauger, 1999), which suggests differences across brain areas in response to stress.

Long-term exposure to the primary mediators of allostatic load may also influence working memory via the hippocampus. There is evidence to suggest a neuronal linkage between the two cortical areas, tracing a pathway between CA1 and the medial prefrontal cortex (Thierry, Gioanni, Degenetais, & Glowinski, 2000). In a study with rodents, stress affecting the hippocampus induced dendritic atrophy in the medial prefrontal cortex (Cerquerira, Mailliet,

Osborne, Jay, & Sousa, 2006). Hippocampal projections terminate in upper layers of the medial prefrontal cortex, suggesting a highly influential stress pathway between the hippocampus and the prefrontal cortex in rats (Cerquerira et al., 2006). Wellman (2001) has also shown reorganization of prefrontal neurons following glucocorticoids administration to the hippocampus. As dendritic complexity decreased in the hippocampus, atrophy occurred in the proximal areas of prefrontal apical dendrites (Wellman, 2001). Furthermore, a lesion study in neonatal rats related early hippocampal damage to resulting deficits in working memory (Lipska, Aultman, Verma, Weinberger, & Moghaddam, 2002).

Purpose of the Current Investigation

If childhood poverty produces adverse consequences on cognitive processing, then it is paramount to answer the question of how poverty might get into the brain. Stress is one such pathway, but there are no studies examining the long-term stress outcomes of poverty on neural functioning. The present investigation seeks to delineate the potential role of physiological stress in the pathway between poverty and working memory functioning (see Figure 1).

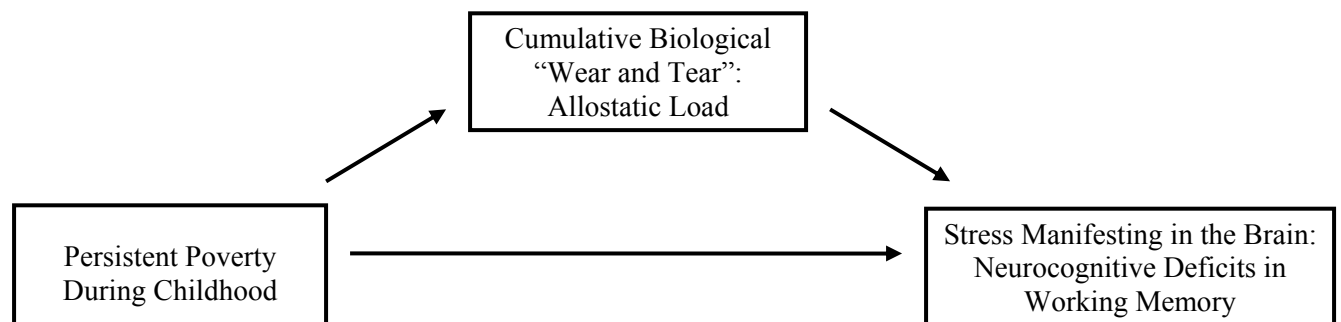


Figure 1. Proposed Mediation Model

Furthermore, no other study to date has looked at neurocognitive functioning in adolescents as a function of early childhood poverty. Within the limited literature on income and neurocognitive functioning in children, cross-sectional designs have predominated, thus limiting our understanding of this issue to a specific age cohort. Using longitudinal data on childhood poverty,

socioemotional development, and physiological stress, the present investigation evaluates Caucasian adolescents from both low- and middle-income groups who were previously assessed at ages 9, 13 and concurrently at age 17. For the first two Waves of the study, all subjects were assigned a 0-6 index of allostatic load, with 6 being the highest marker of physiological stress exposure. Additionally, a handheld, light-tone game was used to assess working memory in 17 year old subjects. Persistent poverty throughout childhood and early adolescence is expected to have negative effects on working memory in older adolescents. Increased allostatic load is predicted to mediate the pathway between poverty and deficits in working memory, and might be an important mechanism in understanding how income relates to changes in cognition.

Methods

Participants. This investigation is part of a longitudinal study on poverty, socioemotional development, and physiological stress that took place between 1997 and 2006, in three separate Waves. Three hundred and thirty-nine children (52% male, 97% Caucasian) living in rural areas of Upstate New York were initially recruited from public schools, Headstart and Cornell Cooperative Extension programs, and using information from wait lists for Section 8 Housing Assistance. From the original sample, only one hundred and seventy subjects participated in all three waves, had complete data on income, allostatic load, and working memory, and were therefore included in the current study. At Wave 1, subjects were nine years old (mean age = 9.21, SD = 1.12). Subsequent waves (W2 and W3) of data collection occurred when the subjects were thirteen (M = 13.37 years, SD = .99) and seventeen (M = 17.29, SD = 1.01), respectively. From birth of the target child until Wave 2, approximately half of the families were living at or below the federal poverty line. At the onset of the study, middle-income families were 2-4 times above the federal poverty line. Subjects and their families were blind to the ‘poverty’ component

of this study. They were told that the research focused on housing quality, stress, and wellbeing. In appreciation of their time, target parents were monetarily compensated with \$50 and the subject was given a toy or book. In subsequent waves, the subject received \$75 (Wave 2) and \$200 (Wave 3).

Materials. The present study assessed duration of poverty, six biological markers of stress (allostatic load), and working memory functioning.

Duration of Poverty, Birth-Wave 2: proportion of life spent in poverty from birth through 13 years of age was calculated. Income was assessed according to total family resources (cash entering into the home, including food stamps, housing supplements, child support, alimony, etc.). *Family* refers to the household composition and included individuals sleeping in the home at least three nights per week. For primary caregivers of the subject (usually the mother and father, or just the mother), “Family/Work History” calendars were used to document changes in type of work (paid, volunteer, unemployed, or actively seeking), hours worked per week, and average earnings (see Appendices A and B). Yearly earnings for each subjects’ family were calculated using Work History forms, and using a Demographic Update form (see Appendix C) to determine poverty status. Total resources (income, food stamps, Home Energy Assistance Program, alimony, etc.) per family were cross-referenced with the annually-adjusted Federal poverty threshold to determine poverty status for a given month. Duration of poverty was determined by counting the number of months between birth and Waves 1 and 2 that families were in poverty. This value was converted into the proportion of months spent in poverty during the adolescent’s lifetime (up to age 13).

Allostatic Load, Waves 1 & 2: assessed six biological markers of stress, including resting systolic and diastolic blood pressure, overnight cortisol (12-hr functioning of the HPA axis),

overnight epinephrine and norepinephrine (12-hr functioning of the sympathetic nervous system), and fat deposits [body mass index (kg/m^2)]. Blood pressure was assessed using an automatic blood pressure machine (Critikon Corp., Tampa, FL; Dinamap Model 1846SXP), with child or adult size cuffs (depending on upper arm circumference). *Resting blood pressure* was measured as an average of the second through the seventh reading, without any stressful provocation (e.g., while the subject sat quietly). Reactivity and recovery blood pressure were not included in the allostatic load index (for rationale, see Evans, Kim, Ting, Tesher, and Shannis, 2007). *Cortisol, epinephrine, and norepinephrine* stress hormones (μg) were measured from 12-hour urine samples (8pm-8am), deep-frozen at -80 degrees Celsius, and later analyzed by a technician who was blind to the poverty status of the subject. Cortisol was measured using radioimmune assay (Baxter Travenol Diagnostics, 1987), and catecholamines (epinephrine and norepinephrine) were assayed with high performance, liquid chromatography with electrochemical detection (Riggin & Kissinger, 1977). Creatinine was also assessed to control for incomplete voiding of urine and for variation in body mass (Tietz, 1976). *Body fat* was determined by calculating body mass index for each subject; as indicated above, BMI is weight divided by height squared (kg/m^2). Weight itself was measured using a standard mechanical (non-digital) scale, in pound units. Height was assessed with a tape measure, in inch units, while the subject was standing. United States English units were converted to metric units in order to calculate BMI. To calculate allostatic load, each physiological marker was defined using a 0/1 dichotomy. Subjects with physiological values (resting blood pressure, neuroendocrine hormones, and BMI) in the top quartile (above 75%) were given a score of 1; any value below the top quartile was given a score of 0. Allostatic load scores were summated across each physiological measure, and vary from 0-6 for each subject.

Working Memory Functioning, Wave 3: assessed working memory using the hand-held game SIMON® (Hasbro, Pawtucket, Rhode Island). This commercially available electronic game has a yellow plastic exterior, is oval in shape, and measures 10.6 inches (height) by 10.6 inches (width) by 2.2 inches (depth). The original SIMON has four colored plastic panels evenly distributed in the center of the game. In the current study, the panels were modified to be monochromatic (white, translucent) to remove the possibility of color influencing memory functioning. The plastic panels each present auditory and visual stimuli, and are also used to document sequence responses. One of four possible tones corresponds with each panel. An initial sequence will include only one randomly assigned panel that lights up and emits one of the four tones. For every correctly remembered panel, another tone is added to the sequence, thus increasing the length and complexity of the memory task. Each tone and corresponding visual cue is presented for less than 500 ms before changing to the next stimulus in the sequence. During the memory recall session, subjects must correctly press the panels in the specified order. Failure to repeat the sequence (either by incorrectly pressing a panel or by failing to respond) causes the trial to end.

Procedure. Subjects were assessed in three separate Waves of data collection, at 9, 13, and 17 years of age. A standard protocol was used to collect data in the home of each participant (Evans, 2003; Evans, Kim, Ting, Tesher, & Shannis, 2007; Evans, unpublished procedure for W3). Two experimenters worked independently with the subject and his or her mother to assess family poverty level and degree of physical stress experienced by the target youth.

For the hour prior to the home interview in Waves 1 and 2, the subject was asked to not engage in physical activity. Resting cardiovascular functioning was measured after the subject and his or her mother completed Family/Work history and Demographic Update forms. The

experimenters measured subjects' standing height and weight, inquired as to which hand they use when writing, and placed an appropriately sized blood pressure cuff above the antecubital region of the non-dominant arm. The subject sat quietly for 14 minutes while resting blood pressure was recorded. A total of seven blood pressure readings were recorded at two minute intervals.

In Wave 3, seventeen year old subjects participated in two separate SIMON trials throughout the course of a two hour interview. The second trial was repeated to account for practice effects. For each trial, SIMON was placed on a table (usually kitchen or living room) in front of the subject. The following standardized instructions were then read:

Now we are going to play a game called Simon. Simon is going to make a noise and light up a sequence of panels. Your job is to press the same panels that Simon lights up in the same exact order that Simon shows them to you. Simon will repeat the sequence again but will then add another panel to the order. You must again repeat the sequence exactly as Simon has just showed it to you, including any new panels that Simon adds each turn. Do this for as long as you can. Do you have any questions? *If necessary, the experimenter clarified the instructions.* I'll demonstrate the game for you just to make sure you understand the rules. *The experimenter demonstrated four sequences of the game as the subject watched.*

Okay, now why don't you practice?

The experimenter pressed START to reset the game. Each subject practiced for two sequences to ensure their understanding of the instructions. Following the practice run, further instructions were provided:

Do you have any questions about how to play the game? *If necessary, the experimenter again clarified the instructions.* Now let's play the game for real.

I'm going to restart the game now. Remember, try to follow the sequence for as long as you can.

The subject listened to the tones and repeated each sequence provided by SIMON. Every sequence repeated correctly was documented by the experimenter. A buzzer sounded when an incorrect panel was pressed or if the subject failed to continue the sequence within a specified time period (3 seconds following the previous stimulus). When the buzzing sound terminated the game, the experimenter confirmed the number of correctly repeated panels and documented this number.

The second trial occurred about 45 minutes later with the same instructions and procedure used in the first trial.

Results

Prior to analyzing the data, I averaged the two SIMON trials ($r = .17$) to better capture working memory functioning for each subject. Allostatic load scores from Waves 1 and 2 were also averaged to create a summary index of stress level over time. Also, two statistical outliers were found during analyses with values three standard deviations above the mean for SIMON; these outliers were removed from the data set before performing further statistical tests. Lastly, sex did not significantly contribute to any of the models and was therefore excluded from all final analyses.

Table 1 illustrates the intercorrelations for proportion of life spent in poverty, allostatic load (0-6 indices), and working memory (accuracy on SIMON). To first examine the relationship between proportion of life spent in poverty and working memory functioning, average accuracy on SIMON was regressed onto poverty. Proportion of months spent in poverty from birth to Wave 2 (age 13) was inversely related to performance on SIMON, $b = -1.04$,

$t(168) = -2.30, p = .023$ (see Figure 2). Regressing allostatic load onto poverty revealed a significant, positive relationship between the two constructs, $b = .364, t(168) = 2.35, p = .02$ (see Figure 3). Lastly, subjects with higher childhood-early adolescent allostatic load had lower accuracy on the SIMON working memory task, $b = -.64, t(168) = -2.91, p = .004$ (see Figure 4).

Multiple regression analysis was conducted to evaluate whether the relationship between poverty (birth-Wave 2) and working memory functioning (Wave 3) is partially accounted for by exposure to accumulated allostatic load (Wave 1 and 2). Table 2 summarizes these results, showing the original raw b weight, its standard error, and the standardized β weight before partialing out the averaged allostatic load term (Step 1); and then the new raw weight, standard error, and standardized β weight after partialing out the averaged allostatic load term (Step 2). The R^2 term is also provided for step 1 and step 2. Using Baron and Kenny's (1986) model, mediation is indicated when the β weight for the poverty term becomes non-significant after partialling out the childhood-adolescent allostatic load term. Full mediation was suggested when the β weight became statistically non-significant in step 2. Specifically, inclusion of allostatic load reduces the effect of poverty on working memory by 20%.

Discussion

This study sought to examine how chronic poverty might have a long-term effect on working memory. Previous research has shown an inverse relationship between poverty and brain functioning (Farah et al., 2006; Mezzacappa, 2004; Noble et al., 2005), but has not delineated an underlying mechanism that would account for changes in neurocognition. Allostatic load is a likely contributor, as physiological stress in the brain disrupts neurogenesis, decreases the connections between dendrites, and induces atrophy in areas essential for declarative and working memory (Lupien, Maheu, Tu, Fiocco, Shraek, 2007; Lupien, 2006;

Kim, Song & Kosten, 2006, Sapolsky, 2004; McEwen, 2000). In concord with the allostatic load model, modest changes in physiological dysregulation across multiple systems—as opposed to changes in one system—increase risk for negative developmental outcomes over time (Evans, 2003). In this study, children who spent most of their lives in poverty displayed increased allostatic load at nine and thirteen years of age (Figure 4), and also performed more poorly on a task measuring working memory at seventeen years of age (Figure 2). The pathway between poverty and working memory resulted in an R^2 value of .030, indicating that poverty alone accounts for 3% of the variance in working memory accuracy. Together, poverty and allostatic load account for .067 or 6.7% of the variance. This increase in R^2 suggests that including allostatic load in this pathway explains an additional 3.7% of the variance in working memory accuracy. While these effects are not robust—with such small percentages—the values were still significant. The findings therefore suggest that poverty, over the course of childhood and early adolescence increases allostatic load, and this dysregulation, in turn, explains some of the subsequent deficits in working memory four years later.

What is it about the environment of poverty that substantially affects learning in children? The level of investment on the part of the parents, such as the provision of financial, educational, and social resources within and outside the home differs between low and middle-income households. While this latter fact accounts for some of the adverse cognitive outcomes of childhood poverty, it only explains about 10% of variance (Magnuson & Duncan, 2002). Environmental and socioemotional risk factors play an additional role, influencing the elevation of allostatic load over time. Exposure to one or two risk factors shows little detrimental effects on cognitive functioning compared to the exposure of multiple risk factors. When stressful stimuli converge, there is an increased probability of negative outcomes in development

(Ackerman, Izard, Schoff, Youngstrom, & Kogos, 1999; Sameroff, 1998; Rutter, 1981). Low socioeconomic status (SES) families are more frequently exposed to a number of risk factors (Evans & English, 2002). A series of experiments have isolated risk factors that cause stress more frequently in low SES families as compared to their middle SES counterparts. Evans and English (2002) found that exposure to physical and psychosocial stressors is correlated with elevated risk for socioemotional difficulties. Eight-10 year old low income children are found to have substandard housing, louder noise levels, and increased crowding, family turmoil, early childhood separation, and increased community violence (Evans & English, 2002). Middle income children may experience some of these physical and psychological risk factors, but this happens far less frequently. When comparing cumulative risk (0-6 score) between poverty and non-poverty samples, the average stressor index for children living in poverty was 2.82, while middle-income children experienced a mean of 1.53 stressors (Evans & English, 2002). In addition to showing increased risk for environmental and socioemotional stressors, low-income children display dysregulation of the HPA axis, measured by increased overnight cortisol levels (Evans, 2003; Evans & Kim, 2007). Comparatively, children and adolescents from middle-income families do not display the same elevated indices of environmental, psychosocial, or physiological stress outcomes.

In 2003, Evans added three additional risk factors to the original Cumulative Risk index-- an income-to-needs ratio, being raised by a single parent, and whether the target mother graduated from high school, and correlated these nine converging domains with physiological stress. He observed prolonged recovery of diastolic blood pressure in children with higher cumulative risk following an acute stressor (mental arithmetic). More time was needed for these children's diastolic blood pressure to return to baseline levels than for children with only one or

two risk factors. There is also a strong correlation between neuroendocrine stress hormones and cumulative risk--low-income nine year old children with high indices of cumulative risk had higher overnight levels of urinary cortisol (Evans, 2003). Evans and Kim (2007) conducted a follow-up investigation with the same population when the children were 13 years old. Low-income young adolescents raised in rural poverty showed increased exposure to cumulative risk factors along with increased overnight urinary cortisol levels (Evans & Kim, 2007). Comparatively, middle-income young adolescents did not display the same elevated indices of environmental, psychosocial, or physiological stress (Evans & Kim, 2007).

Longitudinal data has shown consistent elevation of allostatic load in low-income individuals with high indices of cumulative risk, and has illustrated cumulative risk as a mediator between decreased income and increased cortisol levels (Evans & English, 2002; Evans, 2003; Evans and Kim, 2007). As the current study has illustrated, allostatic load accounts for some of the changes seen in working memory. A huge percent of the variance in working memory was not explained by the current model. Theoretically, exposure to multiple environmental and psychosocial stressors could also mediate the pathway between poverty and neurocognitive deficits. One possibility is to examine the relationship between poverty, cumulative risk, and allostatic load and measure how each variable individually, and as a complete model, influences outcomes in working memory.

Alternative Explanations for the Results

Accuracy slightly, but still significantly decreased on SIMON trials for low-income adolescents, with responses that were .02 less accurate for every additional unit of time (proportionately) spent in poverty up to age 13. Rather than discounting the importance of poverty on outcomes in working memory, it's possible that the measures used to initially

determine poverty status are faulty. The Federal poverty thresholds are based on an archaic method of estimating family needs and don't fully capture what it means to be poor. The difference between "poor" and "near poor" varies greatly in terms of services provided by the government; e.g., families with an income-to-needs value slightly above 1 (the Federal poverty value) are not eligible for some welfare benefits. The Federal government still calculates poverty based on the cost of a minimum required diet for survival, multiplied by three to account for other needed resources. This assessment is outdated and needs revision to reflect current expenditures that are more expensive than food. A more representative indicator of poverty should also account for rising costs of gas, heat, and electricity for families, as well as geographic differences in the costs of living. For the current study, poverty status was initially measured dichotomously for every month between birth and Wave 2, and as indicated in the Methods section, a proportion was calculated by dividing age in months at Wave 2 by time spent in poverty. An income-to-needs measure was not used and differs in values above or below the poverty line, and might illustrate how ratios slightly higher than one (e.g. 1.2) affect neurocognition. While these people are not technically "poor," allostatic load does not, on average, drop to zero when the income-to-needs ratio exceeds the poverty threshold; so, allostatic load might also be elevated in this subset of people, who in turn also display deficits in working memory. The main point here is to consider methodological differences between duration of poverty and an income-to-needs ratio, and how the former accounts for stability (or change) in poverty status over time, and the latter for individual differences between subjects at one period in time. Because life is not static, it seemed more important to measure the impact of poverty across the lifetime (up to age 13). Still, without a more accurate indicator of poverty,

studies measuring income-related effects may continue to underestimate the pervasiveness and negative consequences of poverty.

Additionally, SIMON may not have been as reliable as traditional neuropsychological tools used to assess working memory (such as an Object 2-back task). While SIMON requires the storage, maintenance, and activation of information over a relatively brief period of time, no studies have tested its effectiveness. Because this is a light-tone game, individuals with a musical background might be better at associating the changes in pitch than others without similar experiences. Even so, one of the four monochromatic panels emits a light with the corresponding tone on each trial, so visual learners would not be at any disadvantage. It's also important to realize that SIMON is not an extremely popular commercial item, and most, if not all of the subjects had never previously played the game prior to the day of data collection.

Another limiting factor is the failure to control for genetic differences in IQ between low- and middle-income adolescents. While this may seem relatively important, research has shown environmental factors having a greater influence on childhood IQ than genetic factors in lower SES populations (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). A true measure of IQ may not have been possible to obtain because at higher SES, genetics have a greater influence on childhood IQ than environmental factors (Turkheimer et al., 2003).

Furthermore, the sample used in this study comprised low- and middle-income adolescents living in upstate New York, and is limited in two regards. First, sixty-seven individuals spent some of their lives living in poverty, while a larger portion of the sample (102 people) never lived below the poverty threshold. Keeping in mind the aforementioned pitfalls of poverty classification, the unevenly distributed sample may have biased the results. Secondly, the sample was non-representative and studied a predominantly Caucasian population living in

rural areas. Low-income families in an urban environment might have access to resources (e.g., more social support, community involvement, etc) unavailable to those living in rural areas, and these outlets might help buffer against the rise in allostatic load.

Implications and Future Directions

Daily stressors (such as work pressures, interpersonal conflicts, in addition to other social factors like crowding in the home, etc.) generate chaos, lack of control, and learned helplessness, as well as anxiousness and depression. Irrespective of maladaptive coping mechanisms (e.g., alcohol consumption, smoking, or failure to exercise) which would cause further harm, allostatic load represents an endogenous dysregulation of multiple body systems over the course of time. This study has demonstrated how poverty might get “under our skin” and its findings should encourage scientists and policy makers to investigate primary preventions that might thwart the long-term, pervasive effects of poverty on neurocognition. One possibility involves the role of buffering effects that could moderate the pathway between poverty and allostatic load, via increases in circulating oxytocin (a neuropeptide that is released on social contact, and relaxes activity of the HPA axis). Some research has already been done on the efficacy of low-income mothers who are highly responsive to their children (Evans, Kim, Ting, Tesher, & Shannis, 2007), and while highly speculative, the attention and care provided by a caregiver might increase oxytocin in the child and down-regulate the release of the primary mediators (namely, glucocorticoids) of allostatic load. A secondary prevention might promote healthy coping techniques for impoverished families (via support-systems or community partnerships) which promote resilience amongst youth.

These policy suggestions might be more effective interventions than redistributing money to low-income families because they specifically influence aspects of the physiological stress

response. In the a similar vein, if “stress” is a major underlying reason for the cognitive divide between low- and middle income children, then academic remediation, alone, may not compensate for—and certainly cannot fix—architectural changes in the brain. This research illustrates how neurocognitive disparities exist between different populations and hopefully counters misconceptions regarding educational attainment, as well as provides a partial answer as to why poverty doesn’t really fluctuate amongst the rural poor.

In addition to incorporating an index of cumulative risk, future research should also look at the role of perceived stress to delineate how emotional awareness of the environment affects the physiological stress response and allostatic load, and if the perception of a stressor versus actual physiological stress results in greater deficits in working memory. Lastly, because much of what we know about the brain, allostatic load, and working memory comes from studies with rats, it’s difficult to make homologous comparisons with human neural functioning. Therefore, future research should replicate the animal findings in humans by using standardized neuropsychological measures and neuroimaging techniques that more specifically target the prefrontal cortex.

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Year	1992		1993		1994		1995	
Month	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec
Daycare or School								
BioFather StepFather Boyfriend Single								
Siblings: Gender and Birthdate								

Year	1996		1997		1998		1999	
Month	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec
Daycare or School								
BioFather StepFather Boyfriend Single								
Siblings: Gender and Birthdate								

Did you record the birthdate of the target child? circle all males providing financial support?

Year	2000		2001		2002		2003	
Month	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec	Jan-June	July-Dec
Daycare or School								
BioFather StepFather Boyfriend Single								
Siblings: Gender and Birthdate								

Appendix B

work history 7/21/97

Date _____
Experimenter _____

Work History Continuation

Last time we were here, you and I filled out this scale. Today, I just want to update it from the last time we were here. Date circled is where you need to begin.

Mother's Work History

Year	1993		1994		1995		1996	
Month	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec
Type of Work								
Hours/Week Outside of Home								
Change resid. during this time?								

2

Work History Continuation

Year	1997		1998		1999		2000	
Month	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec
Type of Work								
Hours/Week Outside of Home								
Change resid. during this time?								

workhis.1 7/21/97

Experimenter _____

Father's Work History
[or whoever shares financial responsibility for your child]

Begin where you left off last time.

Year	1993		1994		1995		1996	
Month	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec
Type of Work								
Hours/Week Outside of Home								

Work History Continuation

2

Father's Work History

Year	1997		1998		1999		2000	
Month	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec	Jan-May	June-Dec
Type of Work								
Hours/Week Outside of Home								

(IF LIVING
AT HOME)

Appendix C

DEMO 10/21/97

DEMOGRAPHIC UPDATE MONTH/YEAR OF LAST VISIT _____

1. Have you moved since we were last here on _____?

No ____ [go on to question 2]

Yes ____

I need to know the number of rooms in your home.

Please tell me the number of:

bedrooms ____

bathrooms ____

living rooms ____

study or family rooms (separate from living room) ____

kitchens ____

separate dining or eating area ____

any other rooms you regularly use? [explain]

Note: we are not interested in storage areas like basements, attics, or garages unless they are used for sleeping, family room, etc. If uncertain write down what the room is and what its used for. Make sure its not already in above list.

2. Are you employed? No ____ Yes ____

What is your occupation?

[we need specific job]

3. Is your spouse employed? No ____ Yes ____
What is your spouse's occupation? [be specific]

A spouse is someone who stays here at least three nights per week.

4. What is your spouse's highest level of education?

5. Yours?

6. What was [child] weight at birth? Please feel free to look it up if you don't recall.

7. During the first three months after [child] was born, were you and he/she ever separated for more than one week?

No ____

Y e s - - -

Reason? _____

8. How long have you lived here (nearest half year)

9. What is your current marital status?

Married
Remarried
Not married but living with a partner
Single, never married

10. How many adults live in the home? [Live in the home means stay 3 or more nights per week].

11. How many children live in the home? [Live in the home means stay 3 or more nights per week].

12. Since the last time we were here, which was in [year] have any new adults or children been born, moved in or moved out?

Moved in: how many?

What are their ages/gender?

Moved out: How many?

Ages/gender?

13. Do any of the following biological/by blood relatives of [target child], have any history of heart disease, high blood pressure?

Mother Father Siblings Grandfather (One Both)
Grandmother (One Both)

For the following question: If they don't know monthly, do yearly and indicate as such.

14. What is your income before taxes for the household? Do it monthly or yearly, whichever you can do more accurately. Count your partner/spouse's income if he helps pay bills. [Encourage them to look at bills, receipts if needed for accuracy].

salary _____
disability or social security _____
social assistance _____
alimony or child support _____
housing subsidy _____
food stamps _____
any other sources _____

Yearly _____ Monthly _____

Author Note

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Table 1: Intercorrelations between poverty, allostatic load, and working memory (SIMON).

	1	2	3
1. Life Spent in Poverty, Birth-W2	--	.	
2. Allostatic Load, Waves 1 & 2	.179*	--	
3. Average SIMON Scores	-.174*	-.219**	--

One-tailed correlations, * $p < .05$, ** $p < .01$

Table 2: Regression results for working memory (SIMON), proportion of life spent in poverty (Birth-13 years), and child-early adolescent allostatic load (ages 9 & 13).

Step		<i>b</i>	<i>SE</i>	β
Working Memory				
1	Duration of Poverty	-1.04*	.452	-.174*
2	Duration of Poverty Allostatic Load	-.831	.452	-.140

R^2 for step 1 = .030, R^2 for step 2 = .0198, * $p < .05$

Figure Captions

Figure 1. Proposed theoretical model linking poverty, allostatic load, and outcomes in working memory function. (in text).

Figure 2. Regression plot showing the relationship between persistent poverty and working memory (SIMON) ($\beta = -.174, p = .023$)

Figure 3. Regression plot showing the relationship between persistent poverty and childhood-early adolescent allostatic load ($\beta = .179, p = .020$).

Figure 4. Regression plot showing the relationship between childhood-early adolescent allostatic load and working memory (SIMON) ($\beta = -.22, p = .004$).

