



Time-varying effects of income on hippocampal volume trajectories in adolescent girls



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ABSTRACT

Children from lower-SES families exhibit smaller hippocampal volume than do their higher-SES peers. Few studies, however, have compared hippocampal developmental *trajectories* as a function of SES. Thus, it is unclear whether initial rank-order stability is preserved, or whether volumes diverge/converge over the course of adolescence. In a sample of 101 girls ages 10–24 years, we examined the longitudinal association between family income and parental education, proxies for SES, and changes in hippocampal volume. Hippocampal volume was obtained using MRI; using mixed modeling, we examined the effects of income and education on hippocampal volume across age. As expected, changes in volume were non-linear across development. Further, trajectories diverged in mid-adolescence, with lower-income girls exhibiting reductions in hippocampal volume. Maximal income-related differences were observed at 18 years, and trajectories converged thereafter. This interaction remained significant when accounting for maternal hippocampal volume, suggesting a unique contribution of environment over potential heritable differences. In contrast, the association between parental education and offspring hippocampal volume appeared to be stable across adolescence, with higher levels of parental education predicting consistently larger hippocampal volume. These findings constitute preliminary evidence that girls from lower-income homes exhibit unique trajectories of hippocampal growth, with differences most evident in late adolescence.

1. Introduction

Growing up in a home with fewer economic and educational resources has been shown to be a risk factor for a range of negative life outcomes. Compared with children raised in higher socioeconomic status (SES) households, children from lower SES backgrounds are more likely to perform poorly in school, exhibit behavioral problems, and develop psychopathology (Duncan et al., 1994; Hackman et al., 2010; McLoyd, 1998). Researchers have proposed several mechanisms through which this risk may be conferred, including a lack of cognitively stimulating experiences (Johnson et al., 2016; Weisleder and Fernald, 2013), increased stress in the home (Evans et al., 2005; Evans and English, 2002), unequal access to educational and health resources (Coleman, 1968; Graham, 2008), and unfair treatment within these systems (Alexander and Entwisle, 1987; Marks et al., 2006; McLoyd, 1998).

While there is a long history of psychosocial and epidemiological research examining the causes and consequences of disparities in SES,

there has been a recent impetus to examine the neural mechanisms through which risk factors may exert their adverse effects. Understanding the neural regions implicated in risk – and particularly the ages at which these effects have the greatest adverse impact on neural regions – may facilitate the development of interventions that utilize sensitive periods in children's development (Gabrieli and Bunge, 2016; Lawson et al., 2017). Indeed, it is clear that different neural systems undergo significant transformation during distinct periods of development, which may lead people to be particularly sensitive to relevant forms of environmental input at different times. For example, in the context of SES, it is noteworthy that critical connections between regions supporting executive function are developing rapidly during adolescence (Murty et al., 2016; Ordaz et al., 2013).

In this context, researchers have recently demonstrated that the hippocampus, a brain region that is particularly sensitive to stressful effects of the environment (Frodl and O'Keane, 2013; Lupien et al., 2009), is smaller in children and adolescents from lower- than from higher-SES homes (Hanson et al., 2015, 2011; Luby et al., 2013; Noble

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et al., 2012). Rao and colleagues found that in a sample of lower-SES children, parental nurturance at age 4, but not at later ages, was significantly associated with smaller hippocampal volume in early adolescence (Rao et al., 2010), suggesting a direct association between the early environment and hippocampal volume, although the effects were in the opposite direction than would be expected from other research. Further supporting an association between the environment and hippocampal development, Hanson and colleagues found that early life stress was significantly linked to smaller hippocampal volume, and that hippocampal volume partially mediated the relation between stress and behavioral problems (Hair et al., 2015). Recently, Noble and colleagues (2015) examined this association in a large, cross-sectional sample of children ages 3–20. They found a significant association between parental education and left hippocampal volume. Moreover, their analyses revealed that effects of education on the hippocampus was most pronounced for those children whose parents had the least formal education. Interestingly, there was no significant association between income and hippocampal volume in their sample, despite significant support for the link between these variables from other work (for a review, see Farah, 2017). Notably, consistent with prior studies (Mills and Tamnes, 2014), Noble and colleagues found that a quadratic model was the best fit for modeling hippocampal volume across this age range (Noble et al., 2015).

Importantly, the majority of research linking SES to hippocampal volume has thus far been cross-sectional, comparing hippocampal volume of low- and high-SES children at a single time point. This approach has critical limitations, including the difficulty of separating age-related differences from cohort effects or age-related measurement errors (Church et al., 2010). Longitudinal analyses can model between- and within-subject variation separately to describe growth processes more accurately. Indeed, a longitudinal approach is particularly useful in examining the relation between SES and hippocampal volume, given the documented inverted-U trajectory of hippocampal development (Gogtay et al., 2006; Mills and Tamnes, 2014). Not only does this protracted and nonlinear growth make it difficult to interpret the meaning of volumetric differences at a single time point during childhood, but the teenage years may be particularly significant, given that important connections between the hippocampus and prefrontal cortex that support cognitive control are developing rapidly during this time (Murty et al., 2016).

Despite evidence of the protracted development of the hippocampus, most studies have focused on the relation between SES and hippocampal volume in childhood. This limits our understanding of this association, given the likelihood that SES exerts different levels of influence on the hippocampus throughout development. For example, maternal sensitivity influences trajectories of hippocampal growth when children are preschool-aged, but not older, suggesting a sensitive period for maternal sensitivity (Luby et al., 2016). Similarly, socioeconomic factors may be differentially salient during specific developmental periods. For example, when children shift in the relative importance of their peer group compared to their family at 12–13 years of age (Claes, 1992), SES-related differences in cortisol levels have been found to disappear (Dowd et al., 2009; Lupien et al., 2001). Moreover, higher-SES children experience more stress during school transitions than do lower-SES children (Lupien et al., 2001). In fact, a recent study showed no association between childhood SES and hippocampal volume in adulthood (Lawson et al., 2017). Thus, lower-SES children might exhibit hippocampal recovery over late adolescence and early adulthood. On the other hand, however, there is evidence that childhood poverty influences hippocampal function and associated memory-related functioning in adulthood (Duval et al., 2017). While some longitudinal research has documented SES-related differences in trajectories of brain growth in infants (Hanson et al., 2013), we know little about SES-related differences in hippocampal growth through adolescence. In fact, it may be that the null results reported by Noble and colleagues (2015) is due to varying effects of income on hippocampal

volume over this large age range. Elucidating whether these differences vary as a function of children's age or remain stable over development has critical implications for the generation of timely and sensitive interventions to improve child outcomes.

Importantly, more recent studies have examined the effects of SES on hippocampal development longitudinally, with mixed results. Hair and colleagues (2015) found that children living below the federal poverty level had hippocampal gray matter that was on average 6–8% below developmental norms across the ages of 4–22. Moreover, these differences partially mediated income-related differences in scores on academic tests, suggesting that hippocampal volume is associated with academic outcomes. These authors calculated developmental norms for hippocampal gray matter by modeling its developmental trajectory in their sample, strategically accounting for its nonlinear development over adolescence. The results that they presented in the paper, however, were based on an average of comparisons across ages, and did not specifically examine whether the effects of income on hippocampal development varied systematically across these age ranges. In another study, Whittle et al. (2017) explicitly tested possible interactions of age and SES in a longitudinal sample of adolescents. Contrary to expectations, there was no main effect of SES or interaction of age and SES on hippocampal volume. However, the authors' models only tested for linear effects, despite evidence of the nonlinear development of the hippocampus; a quadratic model may have yielded different results. Therefore, it remains an open question whether the association between SES and hippocampal volume varies as a function of age over adolescence.

Finally, SES-related differences in children may represent, in part, heritable characteristics acquired from their parents. Indeed, twin studies show that 40% of variance in hippocampal volume is due to genetic influences (Sullivan et al., 2001). Further, hippocampal volumes of mothers and daughters are strongly correlated, significantly more so than for father–daughter, mother–son, or father–son pairings (Yamagata et al., 2016), suggesting matrilineal patterns of transmission for this region. It is important, therefore, to control for the influence of matrilineal transmission in characterizing SES-related differences in hippocampal volume.

We address these issues by examining longitudinally the effects of family income and parental education, two distinct indicators of SES (Braveman et al., 2013), on trajectories of hippocampal volume in 10- to 24-year-old females. We examined these variables separately because evidence suggests that they are differentially associated with early experiences and subsequent outcomes (Duncan and Magnuson, 2012). Many participants provided multiple time points of data, allowing us to investigate changes in hippocampal volume over development. Our first aim was to examine whether trajectories of hippocampal development vary as a function of family income and parental education. We tested two potential hypotheses. One possibility is that trajectories of hippocampal volume would diverge as a function of family income and parental education across adolescence, with children from less wealthy or educated households exhibiting a steady reduction in hippocampal volume compared to their higher-SES peers. This finding would be consistent with research demonstrating the compounding effects of factors associated with low SES (Hart and Risley, 1995), and with animal models showing a lack of synapse production following early exposure to stress (Andersen and Teicher, 2004). Another possibility is that trajectories would converge over adolescence, mirroring findings of SES-related differences in cortisol in childhood but not in adulthood (Dowd et al., 2009; Lupien et al., 2001).

In addition, because a subset of the girls' biological mothers were also scanned, we were uniquely positioned to assess the matrilineal familial transmission of hippocampal volume. Therefore, our second aim was to examine the association between two metrics of SES (i.e., family income and parental education) and offspring hippocampal volume, controlling for maternal hippocampal volume. We conceptualize this relation as a proxy for both inherited traits and the shared

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