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An Ecological Approach to Understanding the Developing Brain: Examples Linking Poverty, Parenting, Neighborhoods, and the Brain

Luke W. Hyde, Arianna M. Gard, and Rachel C. Tomlinson
University of Michigan

S. Alexandra Burt
Michigan State University

Colter Mitchell and Christopher S. Monk
University of Michigan

We describe an ecological approach to understanding the developing brain, with a focus on the effects of poverty-related adversity on brain function. We articulate how combining multilevel ecological models from developmental science and developmental psychopathology with human neuroscience can inform our approach to understanding the developmental neuroscience of risk and resilience. To illustrate this approach, we focus on associations between poverty and brain function, the roles parents and neighborhoods play in this context, and the potential impact of developmental timing. We also describe the major challenges and needed advances in these areas of research to better understand how and why poverty-related adversity may impact the developing brain, including the need for: a population neuroscience approach with greater attention to sampling and representation, genetically informed and causal designs, advances in assessing context and brain function, caution in interpretation of effects, and a focus on resilience. Work in this area has major implications for policy and prevention, which are discussed.

Public Significance Statement
Millions of youth grow up in poverty and are exposed to an unequal share of adversity which impacts brain and behavior development. An ecological approach to developmental neuroscience can help to articulate the active ingredients associated with poverty that impact brain development. Better understanding how and why various adversities, including harsh parenting and neighborhood poverty, impact brain development can inform policies to prevent negative outcomes.

Keywords: poverty, parenting, neighborhood, developmental neuroscience, brain function

Nearly 12 million children (16.2% of the U.S. population under age 18) live at or below the U.S. federal poverty line, which was $25,100 annually for a family of four in 2018 (Semega, Kollar, Creamer, & Mohanty, 2019). Moreover, 32% of children live in families that are "near poor" (200% of the poverty line), a benchmark that identifies families

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Authors' note. Luke W. Hyde, Department of Psychology and Institute for Social Research, University of Michigan; Arianna M. Gard, Institute for Social Research, University of Michigan; Rachel C. Tomlinson, Department of Psychology, University of Michigan; S. Alexandra Burt, Department of Psychology, Michigan State University; Colter Mitchell, Institute for Social Research, University of Michigan; Christopher S. Monk, Department of Psychology, Institute for Social Research, and Department of Psychiatry, University of Michigan.

Luke W. Hyde and Arianna M. Gard contributed equally to the article.

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Correspondence concerning this article should be addressed to Luke W. Hyde, Department of Psychology, University of Michigan, 2229 East Hall, 530 Church Street, Ann Arbor, MI 48109. E-mail: LukeHyde@umich.edu

likely struggling to meet basic needs (Semega et al., 2019). Poverty predicts an array of maladaptive outcomes including lower academic achievement and future earnings, greater challenges with mental and physical health, and higher rates of criminal behavior (McLoyd, 1998). Moreover, poverty is often propagated across generations via these maladaptive outcomes (Kendig, Mattingly, & Bianchi, 2014). Although poverty is often described as a category, income and wealth distributions are continuous with a graded effect. Moreover, experiences of families living in impoverished contexts are diverse especially when considering other demographic factors (e.g., race, ethnicity, family structure, geographical context, urbanicity). One major question that developmental scientists have tackled is delineating *how* poverty affects children and their developmental trajectory. An array of mechanisms and poverty-related adversities have been identified to explain why poverty undermines development, including limited access to educational resources (Bradley & Corwyn, 2002), exposure to environmental toxicants (Trentacosta, Davis-Kean, Mitchell, Hyde, & Dolinoy, 2016), parenting stress and family conflict (Conger, Rueter, & Conger, 2000), exposure to violence (Margolin & Gordis, 2000), and exposure to instability and chaos (Doom, Vanzomeren-Dohm, & Simpson, 2015). However, at some level, poverty must "get under the skin" to impact socioemotional and cognitive outcomes. One major way in which experience is biologically embedded is by shaping brain structure and function across development, potentially "tuning" important brain systems in a way that is adaptive in the short term or in some contexts (e.g., greater attention to threat in a dangerous environment), but that may lead to negative outcomes in the longer term or in other contexts (e.g., greater stress-related health problems; Varnum & Kitayama, 2017).

Ecological Models of Development and the Brain

As neuroimaging has advanced as a field, researchers are moving from human brain mapping and connecting individual variability in brain structure and function to *outcomes* (e.g., psychopathology), to focusing on identifying *predictors* of individual differences in brain structure and function. Individual variability in brain structure, function, and development likely arises from a complex interplay of genetic variation and experience (Hyde, 2015). Models of experience-dependent plasticity (Blumberg, Freeman, & Robinson, 2010; Gottlieb, 2007; Oyama, Griffiths, & Gray, 2003; Wiesel & Hubel, 1963) have highlighted the profound role that environmental experiences play in sculpting brain circuits across development (Lupien, McEwen, Gunnar, & Heim, 2009; McEwen, 2012). Thus, there is an increasing emphasis in developmental neuroscience on identifying *experiences* that predict differences in brain development, which in turn, helps us to understand the diverse array of brain and behavioral trajectories across the life span.

At the same time, models from developmental science and developmental psychopathology have outlined the multilevel, transactional ecology in which children develop. Specifically, ecological models of human development place individuals within multiple environmental contexts, from proximal microsystems (e.g., family) to distal macrosystems (e.g., cultural values; Bronfenbrenner, 1977). Transactional models have highlighted that the child and these nested ecologies are in constant interaction, shaping each other across time and development (Sameroff, 2009). These models of development have then been well-articulated in developmental psychopathology with a focus on identifying mechanisms and pathways to typical and atypical development (Cicchetti, 1993). Although these models have transformed developmental science, the application of these models to human neuroscience has been limited. The current article outlines an ecological model for understanding neural function and structure across development. That is, our goal is to describe an *ecological neuroscience* approach that integrates science on the multilevel, transactional contexts in which children develop with our understanding of the cellular and systems-level development of the human brain to articulate how the brain develops in unfolding physical and social contexts (Figure 1). This ecological neuroscience approach highlights that brain development is nested within multiple ecological systems, sources of contextual influences vary by developmental stage, maturational timing of the developing brain modulates environmental effects on certain systems (e.g., region-dependent sensitive periods), and associations are fluid and may change in strength and direction across development precisely because the brain adapts to environmental influences. To better articulate this approach, we focus on a specific example of interest: How does the ecology of poverty becomes "biologically embedded" to shape neural development? The goal is not an exhaustive review of the area, nor ecological neuroscience broadly, but rather to highlight ways in which developmental theories can inform our search for mechanisms, and to identify steps forward toward a truly ecological model of neuroscience.

Neural Embedding of Poverty

Early animal models have shown that the brain is plastic and, in some cases, dependent on specific environmental inputs during critical or sensitive periods (Hensch, 2005; Wiesel & Hubel, 1963). Moreover, basic cellular work on learning has shown that physical and chemical changes in the brain mediate the brain's ability to learn and remember (Malenka, 1994). That is, the brain is constantly changing as we learn, encode experiences into memory, and adapt to our environment. Thus, for experience to alter behavior, it must

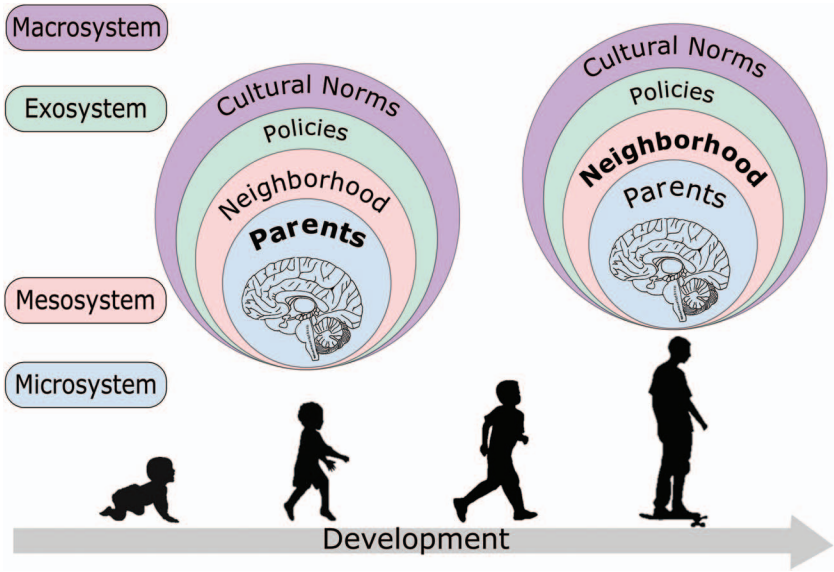

Figure 1. An ecological developmental neuroscience approach: The developing brain is embedded within a multilevel, transactional system that changes over time and across development. Figure adapted from Falk et al. (2013) with permission and builds on work from developmental psychology and developmental psychopathology (Bronfenbrenner, 1977; Hyde, 2015; Sameroff, 2009). See the online article for the color version of this figure.

alter the brain; highlighting the brain as a mediator of our adaption to a variety of contexts.

Building on this research, human neuroimaging studies have begun to link multiple components of socioeconomic status (SES) to differences in brain structure and function. In one line of research, lower SES has been associated with several neural outcomes, including smaller volume of the prefrontal cortex (PFC) and altered functional activity within the middle and inferior frontal gyri and the anterior cingulate cortex (Blair & Raver, 2016; Farah, 2017; Noble et al., 2015). As these frontoparietal networks support executive function and self-control (Dosenbach, Fair, Cohen, Schlaggar, & Petersen, 2008), these findings provide potential neural mechanisms through which low SES may lead to cognitive and achievement-related outcomes.

In parallel, a growing literature has linked SES to the structure and function of regions within the corticolimbic system, including the amygdala, hippocampus, and medial regions of the PFC (including the anterior cingulate). For example, greater childhood poverty has been linked to weaker functional connectivity between the hippocampus and mPFC (e.g., Barch et al., 2016) and relative standing on a social ladder during childhood predicted individual variability in structure and function of the amygdala and anterior cingulate (Gianaros & Manuck, 2010). This neural circuit supports threat detection, emotion processing, fear learning, and salience detection (LeDoux, 2000; McEwen, Nasca, & Gray, 2016), and variability in this system has been linked to internalizing and externalizing psychopathology (Hyde, Shaw, & Hariri, 2013; Monk et al., 2008). Thus, the corticolimbic system is key in understanding stress-mediated pathways from poverty to maladaptive psychosocial outcomes (Barch et al., 2016). These studies also suggest neural adaptation to environmental demands: In the context of poverty, marked by lower resources and greater exposure to unpredictable and uncontrollable events (Bradley & Corwyn, 2002), brain systems may be tuned to enhance attention to salient cues to avoid threats and focus on interpersonal relationships to cope with adversity and stress (Joëls & Baram, 2009; Varnum & Kitayama, 2017), as well as shape cognitive functioning toward being present-oriented and adaptable to rapidly changing conditions (Frankenhuis, Panchanathan, & Nettle, 2016).

Neurobiological Mechanisms

Collectively, these studies highlight the associations between SES and brain structure and function. Moreover, these links are not to the brain broadly, but to specific regions of the brain including systems involved in executive function and stress/emotion processing (Figure 2). Studies have focused on these brain regions because they support behavioral processes often disrupted by poverty, namely, (a) executive function/self-regulation (proximally) and school achievement (distally), and (b) stress/emotion processing (proximally) and psychopathology (distally). Moreover, emerging studies suggest that there may be two routes through which poverty impacts brain development: (a) a

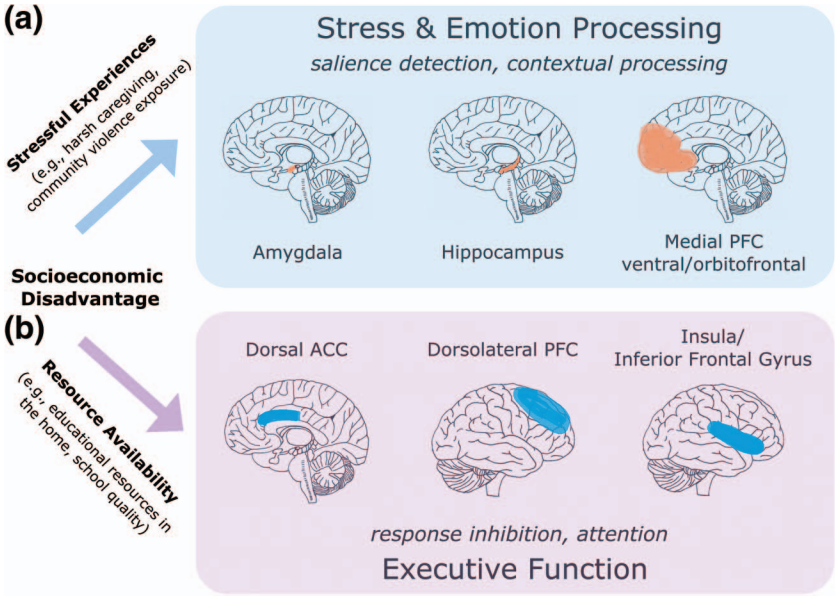

Figure 2. Ecological pathways through which socioeconomic disadvantage may impact youth brain development. We highlight two pathways through which socioeconomic disadvantage during childhood is likely to impact brain development in regions that underlie (a) stress and emotion processing, and (b) executive has been associated with structure and function within these neural regions (Hanson et al., 2013; Johnson et al., 2016; McLaughlin, Sheridan, & Lambert, 2014). See the online article for the color version of this figure.

lack of resources (or deprivation), and (b) stress exposure (or threat; see Johnson, Riis, & Noble, 2016; McLaughlin, Sheridan, & Lambert, 2014).

This work is supported by animal studies that identify the molecular and cellular processes through which resources and/or stress may affect neural development (Joëls & Baram, 2009; McEwen et al., 2016): Animal models of environmental enrichment that induce experimental manipulation in housing conditions (i.e., enhanced social, sensory, and/or cognitive stimulation) have shown that resource-rich environments promote cortical dendritic branching and length, the number of dendritic spines and size of synapses, as well as hippocampal neurogenesis (see Nithianantharajah & Hannan, 2006). Enrichment can also increase neurotrophin synthesis, further enhancing specific forms of synaptic plasticity such as long-term potentiation (Nithianantharajah & Hannan, 2006). By contrast, resource deprivation in the form of unpredictability in maternal sensory signals, as well as quality and quantity of maternal care, has been linked to loss of hippocampal synapses and dendritic spines and branches which impairs learning (Baram et al., 2012; Davis et al., 2017). In parallel, threatening experiences impact behavior through physiological stress responses, including activation of the hypothalamic-pituitary-adrenocortical axis response, where glucocorticoids (cortisol in humans) are the primary output (Gunnar & Quevedo, 2007). Through interaction with glucocorticoid and mineralocorticoid receptors distributed throughout regions of corticolimbic system (De Kloet, Vreugdenhil, Oitzl, & Joels, 1998; McEwen et al., 2016), cortisol acts to stimulate neurobehavioral responses to environmental demands (e.g., a focus toward threat).

Thus, neuroscientists have articulated how a lack of resources and the presence stress are transmitted at the molecular and cellular level (Joëls & Baram, 2009; McEwen et al., 2016) and social scientists have established that poverty increases exposure to stress and fewer resources (Bradley & Corwyn, 2002; McLoyd, 1998). However, in focusing more specifically on the pathway from stress to the brain, what is it about poverty that is stressful? How is this stress transmitted to the developing brain, and how does this process unfold over time and in what specific contexts?

The Ecological Context

Decades of research in developmental science have emphasized that the ecology of child development unfolds through multiple mediating and interacting levels. For example, culture and government policies influence youth through more proximal, direct influences such as parents, schools, and peers (Bronfenbrenner, 1977). Key to these theories is that proximal experiences mediate the effects of more distal influences. In this case, family and community resources affect children via the more proximal social experiences (e.g., interactions with parents, peers, teachers, neighbors). Elucidating influences and mechanisms at multiple ecological levels can help to identify malleable systems to act on to promote positive child development and

thus this work can influence intervention, prevention, and policy.

A specific, multilevel model that explains the effects of poverty on children is the family stress model (FSM). In the FSM, economic hardships (e.g., low family income-to-needs ratio, parental job loss) lead to greater economic pressure on parents (e.g., material hardship). Economic pressure, in turn, gives rise to emotional distress in parents (e.g., depression). Greater parental distress leads to family conflict, including parenting that is harsher and lower in warmth, which can lead to youth psychopathology (Conger et al., 2000). Thus, parenting is a critical mechanism through which poverty impacts the child. Moreover, as disrupted parenting may provide an unpredictable and stressful environment, parenting may be a key initiator of stress processes in youth as their brain develops.

Though we focus much of this article on the "stress" pathway, prominent models and empirical studies also highlight a low resource pathway through which poverty influences brain development in regions supporting cognitive development (Johnson et al., 2016; McLaughlin, Sheridan, & Lambert, 2014). Rather than stressful, limited access to cognitive or social stimulation has been described as "a lack of species-expectant environmental inputs" (McLaughlin, Sheridan, & Nelson, 2017). In a similar mechanistic model that emphasizes how proximal experience mediate poverty effects on the child, the family investment model posits that high SES parents have greater access to financial, social, and human capital, leading to greater investment of resources (e.g., access to high quality education) that foster competent development (Conger & Donnellan, 2007; Mayer, 1998). Of course, stress and resource pathways are overlapping and interacting (e.g., Sheridan, Sarsour, Jutte, D'Esposito, & Boyce, 2012), as highlighted by the interactionist model of SES and human development (Conger & Donnellan, 2007).

Parenting and the Developing Brain

Recent empirical research supports the notion that extreme harsh caregiving environments including maltreatment and institutional rearing predict differences in brain structure and function, particularly within the corticolimbic system (McLaughlin, Weissman, & Bitrán, 2019). Motivated by an ecological neuroscience approach, we wondered whether a dimensional measure of parenting (from harsh to positive) during early childhood would predict amygdala reactivity to threat (fearful faces) in early adulthood. However, because much research had either focused on extreme experiences (e.g., orphanage care, abuse) or on distal predictors (e.g., retrospective SES), we wanted to explore *which* poverty-related adversities might best predict later amygdala activation. Thus, we examined sociodemographic factors (family income, maternal education), family processes (harsh parenting, maternal depression), and community factors (neighborhood poverty), when children were 2 years old, as longitudinal predictors of amygdala reactivity during socioemotional processing at age 20 (Gard et al., 2017). Harsh parenting, neighborhood disadvantage, maternal education, and maternal depression each predicted amygdala reactivity to emotional facial expressions, indicating that an array of risk factors may be linked to amygdala function nearly two decades later. However, in multiple regressions that examined the unique effects of these risk factors, only harsh parenting and neighborhood disadvantage remained as predictors of amygdala reactivity to fearful faces, which in turn predicted externalizing behavior. Thus, both parenting and neighborhood disadvantage served as unique poverty-related adversities signaling threat, and impacting stress and emotion neural systems, which in turn increased risk for negative outcomes.

This study fits with an array of recent studies linking dimensions of parenting to the structure and function of the amygdala and broader cortiolimbic system (e.g., Farber et al., 2019; Kopala-Sibley et al., 2020; Whittle et al., 2016), as well as animal models linking adversity to disrupted maternal behavior, which in turn affects offspring brain development (Baram et al., 2012). Additionally, we have since found similar associations between parenting in early childhood and later amygdala reactivity to angry faces (Gard et al., in press) in a subsample of boys and girls from the Fragile Families and Child Wellbeing Study (FFCWS; Reichman, Teitler, Garfinkel, & McLanahan, 2001), a representative sample of families living in urban environments with substantial enrichment for families living in poverty. Thus, mounting evidence from our lab and others suggests that parenting is an important predictor of corticolimbic functioning longitudinally, particularly within families living in poverty. These studies highlight that, as in the FSM, parenting may be an important, proximal, social factor conveying poverty-related risk to child behavior via stress-related neural pathways.

In addition to this stress pathway, parental involvement in cognitive and social stimulation likely impacts language and executive function and related neural systems through a resource pathway (Farah, 2017; Johnson et al., 2016; Rosen et al., 2019). For example, SES-related disparities in exposure to aspects of parent–child speech (Whitehurst, 1997) predict children's vocabulary development (Hoff, 2003), via brain activation in language centers of the brain (Romeo et al., 2018). Similarly, cognitive stimulation in the home has been linked to cortical thickness in frontoparietal regions, and these factors mediate associations between SES and academic outcomes (Rosen, Sheridan, Sambrook, Meltzoff, & McLaughlin, 2018). Thus, poverty may undermine the proximal home environment via both stress and resource-related ways that play a key role in brain and behavioral development.

The Neighborhood and the Developing Brain

As children mature, they begin to spend more time in the neighborhood, which exposes them to a variety of social exposures, from peers and neighbors to schools. Adverse neighborhood conditions have been highlighted as a salient risk factor, predicting an array of negative outcomes for children living in poverty (Leventhal & Brooks-Gunn, 2000). Though familial poverty overlaps with neighborhood-level poverty, the neighborhood itself can confer unique effects beyond the family. Given that children spend more time in the neighborhood during times of peak neural development (i.e., adolescence), it is surprising that little research has examined the neighborhood as a predictor of neural structure and function.

Given the growing literature examining associations between family level SES and the brain, and the association between family SES and neighborhood SES, we wondered whether neighborhood poverty might be a critical and unmeasured poverty-related adversity. Thus, we examined whether family income, maternal education, or neighborhood poverty would predict response inhibition and inhibitory control-related neural activation in the prefrontal cortex in a sample of 215 twins sampled from birth records with oversampling for families living in above average levels of neighborhood poverty (i.e., >10% of neighbors with families live below the poverty line, the average in the State of Michigan at the time of recruitment; Burt & Klump, 2013; Tomlinson et al., 2020). Given the relatively robust association between family SES (income and education) and behavioral performance on self-regulation and cognitive control tasks (e.g., Hackman, Gallop, Evans, & Farah, 2015; Lengua et al., 2015) and prefrontal cortex structure (e.g., Hanson et al., 2013; Noble et al., 2015), we expected family SES to be the most important predictor of inhibition-related performance and neural activity. Surprisingly, although family income, maternal education, and neighborhood poverty were each related to *behavioral performance* on a go/no-go task, only neighborhood poverty was related to *neural activity* in the inferior frontal gyrus, a key prefrontal region supporting response inhibition. This association, which held even when controlling for the overlap of neighborhood poverty with family level SES, had behavioral relevance: Greater neighborhood poverty predicted worse response inhibition performance via reduced activity in the inferior frontal gyrus (Tomlinson et al., 2020). Thus, the neighborhood may be a critical unmeasured factor in SES-brain function associations, and *where children live* may be more important than their familial resources.

Similarly, following-up on our initial work linking harsh parenting and neighborhood poverty to amygdala reactivity, we wanted to examine whether neighborhood disadvantage might have a unique effect on amygdala reactivity to neutral faces. Neutral faces are ambiguous signals that can be interpreted as threatening (Marusak, Zundel, Brown, Rabinak, & Thomason, 2017). As the corticolimbic system detects and drives behavioral and physiological responses to perceived threats (LeDoux, 2000), the unpredictability of ambiguous facial expressions makes these stimuli potentially salient for youth living in disadvantaged neighborhoods, where threats may be ambiguous and unpredictable. Thus, we sought to replicate our previous finding linking low neighborhood SES to heightened amygdala response to neutral faces in an independent sample of low-income boys. To examine this question, we leveraged data collected from the neuroimaging subsample of the FFCWS called the Study of Adolescent Neural Development (SAND; Hein et al., 2018). Within the SAND, we replicated findings from our previous study and found that neighborhood disadvantage in early childhood (ages 1 to 5) was associated with greater amygdala reactivity to ambiguous neutral faces at age 15. To index neighborhood disadvantage, we constructed a composite score of several census-derived indicators geocoded to participant addresses at the tract- or block-group-level. Although these indicators (e.g., % families living below the poverty line, % single-headed households, % unemployed) are designed to jointly capture the greatest variation between neighborhoods (Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff, & Earls, 1999) and are highly correlated, we examined whether some indicators were more predictive of amygdala function than others. Ultimately, census indicators assessing the percentage of families living below the poverty line and median family income in the neighborhood were uniquely associated with greater amygdala reactivity to neutral faces. Thus, the income/poverty level of neighbors was critically important to the links between neighborhood poverty and later amygdala reactivity to ambiguity. Moreover, these neighborhood effects were above and beyond family level adversities, including family income, maternal education and depression, interparental conflict, and harsh parenting.

These studies highlight the inherent complexity of studying these ecological contexts. Whereas parenting may serve as a mechanism through which poverty impacts child development, neighborhood poverty likely has mechanistic, additive, and interactive impacts on child development in relation to family poverty: Though family resources influence which neighborhood children live in, and in turn the neighborhood-level adversities children are exposed to (e.g., schools, toxicants; a mechanism), neighborhood residence is determined by a myriad of factors including race (e.g., via housing discrimination) and intergenerational factors (e.g., where kin live; Harris, 1999; Sampson, 2012). Thus, neighborhood poverty may impact child development incrementally above family income (e.g., Chen & Paterson, 2006; Schulz et al., 2012) and may interact with family factors (Whittle, Vijayakumar, et al., 2017). Neighborhood

residence, in turn, affects opportunities across generations to influence family resources.

Overall, these studies suggest that *where children live*, not just their families' resources, may be critical in shaping brain development. Our findings align with other research that documents the unique effects of neighborhood poverty on youth brain development, including functional brain organization (Tooley et al., 2020) and corticolimbic structure (Whittle, Simmons, et al., 2017). Moreover, these findings have important policy implications as they may shift attention from a sole focus on family level mechanisms to community-level policies. A clear next step (outlined below) is to identify which aspects of the neighborhood may mediate neighborhood effects on the brain. For example, are neighborhood effects due to stress mechanisms (e.g., exposure to violence) or to lack of resources (e.g., low resourced schools) and will these pathways map specifically to cognitive versus affective neural circuitry?

Does Timing Matter?

Though studies now suggest that harsh parenting and neighborhood poverty predict brain structure and function, a key question is whether the timing of these experiences matter. Developmentally, infants and young children spend most of their time in the home with caregivers (Hofferth & Sandberg, 2001), highlighting the salience of the parent–child relationship during the first decade of life. As children pass into adolescence, peer and neighborhood influences become more salient, which dampens the relative impact of family processes on youth outcomes (Smetana, Campione-Barr, & Metzger, 2006). In parallel, different regions of the brain develop at different rates. Volumetric growth in the amygdala is the largest during the early postnatal years, with >100% increase during the first year of life (Gilmore et al., 2012). In contrast, prefrontal gray matter density peaks during the early puberty (i.e., 10–12 years), followed by synaptic pruning and dendritic arborization (Casey, Jones, & Hare, 2008). During adolescence, as projections from prefrontal regions to other brain regions become more well-defined, medial prefrontal cortex activation to emotional facial expressions increases (Blakemore, 2008) and PFC-amygdala connectivity shifts from positive to negative (Gee et al., 2013). Thus, based on the timing of neural development, early experiences could be more important for subcortical structures like the amygdala, whereas adolescent experiences could be more important for PFC development.

Although recent reviews emphasize the potential importance of developmental timing for adversity effects on corticolimbic function (Lupien et al., 2009; Tottenham, 2015), few studies have tested this hypothesis in humans. Thus, we examined the extent to which the timing of exposure to adversity predicted activity in specific neural regions. An ecological context hypothesis would posit that parenting during early childhood would be important for amygdala development, whereas neighborhood poverty during early adolescence would most strongly relate to prefrontal development, because those are the most salient ecological contexts in those developmental stages. Alternatively, a neural developmental timing hypothesis would suggest that both parenting and neighborhoods would be associated with amygdala development in early childhood versus prefrontal development during adolescence. To test these competing hypotheses, we examined the associations between trajectories of parenting across childhood and amygdala and prefrontal function during adolescence. Using growth curve modeling, we estimated an intercept and slope of harsh parenting using data from ages 3, 5, and 9 in the FFCW-SAND. Results indicated that harsh parenting in early childhood (i.e., the intercept) was associated with blunted amygdala activation during face processing at age 15, whereas increases in harsh parenting from ages 3 to 9 (i.e., the slope) were associated with less activation in the PFC (dorsal ACC) at age 15 (Gard et al., in press).

Similarly, we examined whether neighborhood poverty would show similar developmentally specific effects. We found that neighborhood poverty experienced during early childhood was uniquely associated with greater amygdala reactivity to neutral faces at age 15 and at age 20 across two separate low-income samples. By contrast, neighborhood disadvantage experienced during adolescence was uniquely associated with blunted mPFC activation to neutral faces (Gard, Maxwell, et al., 2020). Thus, in both studies, results supported a neural developmental timing hypothesis, rather than an ecological context hypothesis: adversity experienced during early childhood predicted amygdala function, whereas adversity experienced during late childhood and adolescence predicted prefrontal cortex function.

These fMRI studies fit with several structural MRI studies of maltreatment (Andersen et al., 2008; Pechtel, Lyons-Ruth, Anderson, & Teicher, 2014) that support a developmental timing hypothesis. For example, Andersen et al. (2008) found that sexual abuse in early childhood was more strongly associated with subcortical volumes, whereas sexual abuse that occurred in late adolescence was more strongly associated with prefrontal volume. At the same time, there is also important evidence that early experiences have lasting impacts on cortical development (Avants et al., 2015; McLaughlin, Sheridan, Winter, et al., 2014), potentially undermining the universality of this developmental timing theory. Thus, to test these hypotheses more strongly, we need repeated measures of brain and social context across developmental periods, to examine whether developmental timing effects are unique to structure versus function and to what types of experiences (e.g., Do some experiences have timing-dependent effects vs. others that have timing independent effects?).

Summary—Parents, Neighborhoods, Development, and the Brain

Across several longitudinal studies, parenting behaviors and living in a disadvantaged neighborhood appear to sculpt brain function in regions that underlie emotion processing (i.e., amygdala, mPFC), and executive function (i.e., inferior frontal gyrus). Importantly, these findings highlight two poverty-related contexts, parenting and neighborhood poverty, that may influence neurobiological development. Although the experience of childhood poverty includes exposure to a host of risk factors (e.g., low family income, parental psychopathology, harsh parenting, neighborhood poverty), it is critical to identify *which* poverty-related adversities sculpt brain development and whether any of these experiences mediate poverty effects on the brain (and/or which are independent of family SES effects). Moreover, the timing of adversity may matter, paralleling the developmental trajectories of different brain areas. This work also demonstrates that it is not only extreme experiences (e.g., abuse) that shape brain development, but also dimensions of more common adversities, such as having a relatively harsh parent or living in a neighborhood with neighbors who have fewer resources. Importantly, some of these studies have used well-sampled cohorts with substantial numbers of families living in poverty, which improves generalization to families who face the most risk, but have otherwise been underrepresented in neuroimaging research (Falk et al., 2013).

Next (Baby) Steps

The Broader Ecology of Poverty

Though we have focused on two relatively common candidate poverty-related contexts, they are not the only factors. First, though it is important to focus on factors like harsh parenting as more common and normative adversities that may not be considered "adverse childhood experiences," research on more extreme experiences like abuse and neglect is also critical. These adverse childhood experiences are unfortunately common (rates as high as 13% for sexual abuse, 23% for physical abuse, 16% for neglect: Stoltenborgh, Bakermans-Kranenburg, Alink, & van Ijzendoorn, 2015) and these extreme experiences likely exert larger effects on brain development. Thus, we need a balance of research on extreme and dimensionally common or "normative" harsh experiences, because each can inform intervention. For example, just as child abuse is a robust indicator of delinquency and has thus been targeted by large-public health interventions (Dodge, 2019), studies linking more normative experiences of harsh parenting have also been key to informing parent-focused interventions (Webster-Stratton & Reid, 2003).

Second, family income is only one contributing component to socioeconomic "disadvantage." Family income, parental education, occupational status, and accumulated wealth are differentially stable across time, capture diverse social and economic aspects of (dis)advantage, and have varying effects on child outcomes (Bradley & Corwyn, 2002). Each of these factors may affect child development in different ways and thus "poverty" should be characterized in multiple ways. Although some research has documented shared and unique effects of different components of SES on youth brain development (Farah, 2017), most of the work in this area has focused only on family income or education to index disadvantage. Third, there are a myriad of other social and physical risk factors associated with poverty and negative child outcomes beyond parenting and neighborhoods. For example, poverty heightens the risk of exposure to deviant peers, lower resourced schools, and features of the built environment (e.g., proximity to roadways) that increase exposure to noise pollution, overcrowding, and risk for toxicant exposure (Bradley & Corwyn, 2002; Evans, 2004; McLoyd, 1998). These experiences may be "active ingredients" through which poverty impacts brain development. For example, formal schooling is known to improve children's executive functioning and associated brain activation (Brod, Bunge, & Shing, 2017), and recent work has found that better school climate is associated with increased global cortical thickness and better executive functioning (Piccolo, Merz, & Noble, 2019). Noise pollution, family conflict, and parent work schedules may undermine sleep, a critical factor in brain development, particularly during adolescence (Carskadon, 2011; El-Sheikh, 2011; Telzer, Goldenberg, Fuligni, Lieberman, & Gálvan, 2015). Thus, though we have highlighted parenting and neighborhoods as key poverty-related risk factors, poverty exposes children to a myriad of cascading and interacting exposures that may affect brain development. Importantly, sociologists and public health scholars have been studying features of poverty and disadvantaged neighborhoods (e.g., crime, social cohesion, informal social control) for decades (e.g., Jencks & Mayer, 1990; Sampson, 2012), and this work will be important in articulating how and when specific experiences associated with disadvantage impact brain development.

Fourth, it is important to understand whether poverty-related adversities are mediators through which poverty influences brain development or whether these are independent or interacting risk factors. By understanding the complex ecology of poverty and brain development, we can better inform targets for prevention. For example, our understanding the complex social ecology of antisocial behavior (e.g., SES, parenting, peers), has informed personalized preventative interventions (Dishion et al., 2008), and targeted them in age-related ways (e.g., targeting parents/families during early childhood, peers and teachers during adolescence). Thus, the hope of ecological neuroscience is to delineate the complex influences on brain development

by specifying when and for which brain regions risk is transferred from experience to brain.

Lastly, the ecology of poverty and its effects on neurobehavioral development must also be contextualized in light of broader social and cultural norms, and policies at the exo- and macrosystem levels of influence (Bronfenbrenner, 1977). Cultural neuroscience (Kim & Sasaki, 2014) has demonstrated ways that cultural beliefs, family obligation values (Telzer, Masten, Berkman, Lieberman, & Fuligni, 2010), and repeated engagement in culturally embedded behaviors practices (e.g., meditation; Creswell & Lindsay, 2014) alters neural structure and function (Hyde, Tompson, Creswell, & Falk, 2015). However, as most of this work has been restricted to adult samples, developmental neuroscience could better investigate cultural influences (Chiao, 2018). Beyond culture, little research has evaluated the influence of policy changes on youth brain development. National policy changes such as the Medicare expansion (Currie & Gruber, 1996) and the Clean Air Act of 1970 (Chay & Greenstone, 2003) exert causal effects on infant mortality and health, but research is needed to examine policy effects on neural development.

Do Brain Phenotypes Matter for Outcomes?

Although documenting associations between social ecologies and brain development is important for understanding how environmental experience becomes biologically embedded, an important step is to contextualize what these brain phenotypes mean for outcomes. Much of the research described here implies that neural outcomes are important because they have been associated with negative outcomes (e.g., greater amygdala reactivity is predicted by poverty and predicts psychopathology). However, few studies have examined these mediating pathways in a single study (e.g., Whittle, Vijayakumar, et al., 2017).

Moreover, beyond rare conditions, brain phenotypes should not be considered unequivocally "bad." Emerging research suggests that the same neural phenotype may lead to different outcomes depending on context. For example, in a recent study, in high-resourced contexts, low amygdala reactivity was associated with greater future earnings and lower antisocial behavior, but in low-resourced contexts, it was associated with lower income and greater antisocial behavior (Gard, Shaw, Forbes, & Hyde, 2018).

Care in Interpretation

Relatedly, research into the social ecology of brain development generally, and the identification of mechanisms linking poverty to differences in brain development specifically, has great scientific, intervention, and policy implications. For example, research emphasizing brain explanations has been particularly compelling in Supreme Court cases (Steinberg, 2005), and may provide further motivation to politicians and the public to focus on childhood adversity, especially broader community-level and structural characteristics (Johnson, Blum, & Giedd, 2009). However, this work is fraught with ethical and practical issues (Tolwinski, 2019). First, the compelling nature of brain science is often based on misunderstandings about what these brain images mean (e.g., that differences in groups are static and unchangeable; Johnson et al., 2009). Additionally, these findings can be communicated poorly to imply that youth living in poverty have "holes in their head," and are not as competent, talented, or do not have the same potential as their more well-resourced peers. The goal of this work is not to document that youth living in poverty will be "ruined" by the experience and have "bad brains," but rather to understand how and why the adversity they face becomes biologically embedded. Through the study of adversity, we can identify how to best support positive development, even in the context of adversity. More broadly, the brain is plastic and adapts to the environment. As low SES may be defined by experiences and culture promoting attention to threat (to avoid danger) and greater attunement to others (to promote more collectivistic attitudes to cope with adversity), these neural changes are likely adaptive in unpredictable, uncontrollable, and potentially threatening environments (Varnum & Kitayama, 2017).

Resilience

Beyond studying how adversity leads to negative outcomes via disrupted brain development, we need to understand why some youth do well in adverse environments. Resilience is common (Masten, 2001) and may hold even more promise in terms of informing policy and interventions. An important question is the role the brain plays in resilience (van der Werff, van den Berg, Pannekoek, Elzinga, & van der Wee, 2013), with an increase in studies beginning to find neural markers of resilience (e.g., Dennison et al., 2016), though more work needed. Moreover, focus is needed on aspects of the environment that may help promote positive outcomes, even in the face of adversity. For example, a rich behavioral literature has shown that factors such as social cohesion and collective efficacy protect youth in disadvantaged neighborhoods from developing psychopathology (Leventhal & Brooks-Gunn, 2000), but research is needed that examines whether these factors buffer the impact of poverty on brain development.

Methodological Needs

Causal inference. Additional care is needed in interpreting this research, because most studies are cross-sectional, observational/correlational studies (Duncan, Magnuson, & Votruba-Drzal, 2017). As several examples

above illustrate, there are often multiple confounding (and potentially unmeasured) factors present that may be the true causal mechanism (e.g., studies of family poverty may be tapping neighborhood poverty; Tomlinson et al., 2020). Minimally, researchers should account for this confounding by controlling for correlated social contexts (e.g., neighborhood SES when examining family SES). Another obvious need is for longitudinal brain data that can leverage cross-lagged designs to examine the direction of effects. These models are also better-positioned to examine potential sensitive periods.

Beyond correlational designs, several other approaches are needed: First, one major limitation of much of the work is that many of these associations may not be causal, but the product of shared genes between parents and children (genotype-environment correlation). As one example, parental drug use may increase the probability that the family lives in poverty, while also indexing an inherited neural hyperresponsivity to reward. Gene-environment correlations undermine causal inference in studies and translation to interventions. Behavior genetic approaches, such as twin (Waller, Hyde, Klump, & Burt, 2018) and adoption designs (Hyde et al., 2016), can address these potential genetic confounds directly.

Second, natural and quasi-experiments provide stronger causal inference and may address questions behavior genetic designs cannot. For example, neighborhood effects can be difficult to study in twin designs because twins are nested within neighborhoods and adoption studies may show restriction of range in neighborhood advantage. Potential natural experiments such as measuring the effect of stressful natural disasters on brain structure and function can address these limitations. For example, closer proximity to the World Trade Center on 9/11 was correlated with reduced gray matter volume in the amygdala, hippocampus, insula, ACC, and mPFC, suggesting a causal effect of this major stressor on brain volume in corticolimbic regions (Ganzel, Kim, Glover, & Temple, 2008). Additionally, natural experiments that change economic conditions can provide direct documentation of the effect of changes in SES on brain function. For example, a natural experiment that leveraged variation in casino profits for families where zero to two parents were of American Indian descent, found large effects of exogenous household income on decreases in youth externalizing behaviors that operated via increases in parenting quality (Akee, Copeland, Keeler, Angold, & Costello, 2010).

Third, direct intervention studies provide strong inference and clear policy implications. For example, the Moving to Opportunity study, which randomized families into private housing in near-poor or nonpoor neighborhoods, found that children who moved to nonpoor neighborhoods had fewer symptoms of psychopathology as children, and greater earnings and educational attainment as adults (Chetty, Hendren, & Katz, 2016; Ludwig et al., 2013). Similarly, intervention studies that target caregiving can test this mechanism directly. For example, the Strong African American Families program randomized families into a parenting prevention program and found that the associations between childhood poverty and hippocampal and amygdala volumes was only significant for families who did NOT receive the intervention (Brody et al., 2017). In parallel, the Bucharest Early Intervention Project has shown that children randomized to high quality foster care (and never-institutionalized children) exhibit larger total cortical white matter than children raised in orphanages (Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012).

Samples. One major need for the field of neuroscience is to increase focus on who is studied and to whom results may generalize. That is, we need a population neuroscience approach that focuses on sampling and inclusion of relevant populations (see Falk et al., 2013). Most neuroimaging studies have focused on middle-class participants of European origin, often with few participants facing substantial adversity (or clinical settings that only compare those with a disorder vs. ultrahealthy "control" participants). Though there is certainly a need for examining extreme groups (e.g., families with documented child protective case histories to examine the effects of abuse), more studies are needed that take a dimensional and population approach with representative sampling of the population of interest. Recent empirical research has shown that the application of sampling weights, designed to recapitulate the demographics of a target population, can have a dramatic effect on "well-established" findings in developmental neuroscience (LeWinn, Sheridan, Keyes, Hamilton, & McLaughlin, 2017). Though we have highlighted some of the few studies in this area that have focused sampling (e.g., work using hospital and birth record sampling frames and enrichment for families facing adversity: Gard et al., 2017; Tomlinson et al., 2020), a greater focus on sample composition and design will be important if developmental neuroscience findings are to replicate and generalize to populations of interest. Moreover, though a population approach is important, it is not as simple as sampling approaches. Many families facing the most adversity can be the most difficult to engage in research, may not trust researchers, and may approach neuroimaging research differently (Rowley & Camacho, 2015). Additionally, developmental neuroscientists need to address thorny issues such as whether fMRI tasks and the experience of an MRI scan are equivalent across participants from different SES, race, and cultural contexts (Hyde et al., 2015).

Furthermore, poverty is not experienced uniformly across demographic groups. Within an ecological neuroscience approach, this means attending to issues of racial and ethnic identity (Rivas-Drake et al., 2014), institutional racism (Kendi, 2017; Rothstein, 2017), and generational disparities

in wealth for families of color (Oliver, Shapiro, & Shapiro, 2006). Moreover, for families of color who face a myriad of additional stressors (e.g., microaggressions, instability and threats about immigration), associations between poverty and brain development need to be interpreted carefully within the context of the systems of oppression families are living in. This point highlights an urgent need for the field in terms of representation—few studies in this area even include marginalized groups such as low-income families and families of color (for notable exceptions see examples from the Strong African American Families Project: Brody et al., 2017; SAND-FFCWS: Gard et al., in press; and the California Families Project/Proyecto de las Familias de California: Weissman et al., 2015). Though sampling representation in these studies is important, even more important is a true developmental cultural neuroscience approach to appreciate brain development within the context of various identities (e.g., race, gender) and systems of oppression.

Assessment. Assessment is also key to ecological neuroscience. Developmental scientists have emphasized the use of multiple methods of assessment including observations of participant interaction (e.g., parent–child interactions), leveraging public/official records (census data, court records), geospatial coding, and multiple reporters (e.g., parents, youth, teachers). Studies that utilize these methods may better articulate how and why risk factors affect the brain. For example, comparing the effects of official reports of gun shots versus neighbors' perceptions of safety may highlight whether "objective" versus "subjective" experiences are more important to brain development, just as objective versus subjective reports of social support have differential effects on health (Solomon, Mikulincer, & Hobfoll, 1987).

Similarly, neuroimaging methods are still developing. There is now some concern about the reliability of task-based fMRI data (e.g., Elliott et al., 2020). However, to the extent that we can measure a reliable signal, task-based fMRI has major ecological validity advantages in assessing constructs such as the neural response to threat. Additionally, it is unlikely that activity in a single region of the brain is as important as the coordinated activity of neural networks (Sporns, Chialvo, Kaiser, & Hilgetag, 2004), necessitating the need for network approaches. Thus, basic work identifying which networks are reliable predictors of outcomes and how to identify them is critical to testing developmental precursors.

Summary

Integrating research and methods from cellular, molecular, and human developmental neuroscience with the rich literature outlining a multilevel, transactional ecological approach to understanding development can help to inform our understanding of brain development. This ecological neuroscience approach can build a nuanced, mechanistic, and interactive account of how experience shapes brain development across time. As one potent example, poverty likely impacts behavior via the structure and function of the developing brain. Poverty-related adversities such as harsh parenting and neighborhood poverty are important in shaping the developing brain to pay attention to threat and interpersonal emotion, particularly under conditions of ambiguity. Moreover, the impact of these experiences appears to be stronger during periods in which specific brain regions are developing (the amygdala during early childhood, the PFC during adolescence). Emerging research in this area lays a groundwork, but also highlights the complexity of the science needed to identify mechanisms through which poverty impacts the brain (via stress or resources) and the ways in which associated adversities (e.g., neighborhood poverty) affect family resources, interact with family resources, and mediate the effect of family resources. Moreover, this work highlights the needed advances in the field including genetically informed and experimental designs, greater attention to samples, sampling, and culture, advances in measurement of the brain and context, and a greater focus on resilience. By articulating a more complex and nuanced view of the effects of poverty on brain and behavior, this work can better inform prevention, intervention, and policy, and identify the ways in which these early exposures shape the developing brain.

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