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Poverty and self-regulation: Connecting psychosocial processes, neurobiology, and the risk for psychopathology☆

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ABSTRACT

In the United States, over 40% of youth under the age of 18 live at or near the federal poverty line. Several decades of research have established clear links between exposure to child poverty and the development of psychopathology, yet the mechanisms that convey this risk remain unclear. We review research in developmental science and other allied disciplines that identify self-regulation as a critical factor that may influence the development of psychopathology after exposure to poverty. We then connect this work with neurobiological research in an effort to further inform these associations. We propose a starting framework focused on the neural correlates of self-regulation, and discuss recent work relating poverty to alterations in brain regions related to self-regulation. We close this review by highlighting important considerations for future research on poverty/socioeconomic status, neurobiology, self-regulation, and the risks related to the development of negative mental health outcomes.

1. Introduction

Child poverty is a grievous public health problem, as over 13 million youth under the age of 18 are currently living in poverty in the U.S. [1]. Decades of research have underscored the damaging effects that child poverty has on multiple dimensions of mental health, including depression, anxiety, conduct problems, and substance abuse. Indeed, children and adolescents from low-income families are two to three times more likely to develop mental health problems compared to their more affluent counterparts [2–4]. Although these linkages have been well studied across different disciplines, the mechanisms that convey this risk are poorly understood. Here, we survey two distinct areas of research in an effort to make progress on this issue. First, we review self-regulation as a critical factor in the association between poverty and mental health challenges, as described by developmental science and other allied disciplines. Next, we outline neurobiologically-informed work on poverty and socioeconomic status (SES). While we believe this neuroscientific work to have profound potential to inform these links, the bridges between this nascent area and other related disciplines are still being formed. In service of uniting research subareas, we specifically review research connecting poverty and SES to variations in brain structure and function under a self-regulation framework. We believe connecting neurobiology and self-regulation may provide an effective means for linking poverty, neurodevelopment, and risk for psychopathology. Finally, we close this document by suggesting future research directions for projects using this framework.

2. Framing a focus on poverty and neurobiology

Poverty is a multifaceted concept marked by economic, social, and psychological challenges. In research, this construct is primarily operationalized through financial, educational, and occupational metrics. From a monetary perspective, the U.S. government's federal poverty line (FPL) is one of the most commonly-used markers of poverty. In 2018, the FPL classified a family of four with a household income of $25,100 as "poor." This means that approximately 20% of youth under the age of 18 in the U.S. are living in poverty. Moreover, an additional 20% of youth are "near poor," or from households with incomes of 100–199% of the FPL. Households that can be classified as either poor or near poor are often referred to as "low-income." Additionally, educational and occupational variations have been used to define poverty, describing those with limited educational histories (e.g., not completing high school) or less prestigious occupations (e.g., general laborer; taxi driver) as poor [5]. For those studying the effects of poverty on child development, the financial, educational, and occupational variables would be of the broader family (for income) or specific caregivers (for education and occupation). While there are many ways to conceptualize poverty, we focus this review on child poverty broadly, due to the strong connections with mental health problems.

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Regardless of the exact definition, exposure to poverty in either early (birth to age 5) or middle (ages 6 to 12) childhood is associated with significant increases in both externalizing (e.g., aggression, delinquency) and internalizing (e.g., depression, anxiety) symptomatology [6]. The effects of child poverty persist into adulthood, even after controlling for adult income [7]. In addition to correlational work, quasi-experimental studies find links between increases in family income and subsequent improvements in mental health [8]. For example, Costello and colleagues longitudinally tracked a sample of children (initially aged 9 to 13) living on a Native American reservation. Halfway through their study, a casino opening provided an income supplement to the Native Americans on the reservation. This allowed for the comparison of children in families who: were never poor, remained poor (a persistently-poor group), and moved out of poverty due to the supplemental income (an ex-poor group). After the income supplement, externalizing symptoms in the ex-poor group dropped to the same rate as the never-poor group. Critically, the symptoms in the persistently poor-group remained high [9–11]. Examined collectively, this large body of research suggests strong links between child poverty and negative mental health outcomes.

The linkage between child poverty and higher rates of psychopathology is perhaps not surprising when one considers the "true lived experience" of poverty. Researchers often measure poverty using household income and other imperfect proxies, but this does not capture the full gamut of challenges common to this type of experience. Numerous studies have shown that poverty is associated with a host of stressors and environmental disadvantages including: issues with household noise, structure, and organization; differences in cognitively stimulating experiences, such as home learning resources; higher rates of community violence; exposure to teratogens; and inferior perceptions of the self in comparison to others on the "social ladder" (for review, see [12,13]). These stressors and environmental challenges may be related to (and potentially explain) the connection between child poverty and psychopathology.

Given the high societal costs related to negative mental health [14], it is paramount to identify factors and mechanisms connected to how exposure to poverty and lower SES during childhood contribute to increases in psychopathology. Neuroscience has the potential to uniquely weigh into these investigations, as neurobiological methods may permit the targeted decomposition of outcomes of interest [15]. For example, nearly any form of psychopathology represents the integrated output of multiple underlying neural systems. Thus, neuroscience tools may allow a more elemental focus on specific behavioral, cognitive, and affective brain processes [16]. With information inaccessible at other levels of analysis, a focus on the brain may be prudent, given that the brain is particularly shaped by early in life experiences, and ultimately determines behavioral and physiological responses (whether adaptive or maladaptive) over the course of the lifespan [17].

2.1. Connecting neuroscience to developmental science and other allied disciplines

While likely to advance understanding of mental health disparities, neuroscientific investigations focused on poverty and lower SES should be considered preliminary, at present. Such research is still refining mechanistic theories, replicating key results, and working to apply knowledge about the brain to potentially reduce disparities (or at least minimize their impacts). Stated more succinctly by Farah, "the validity and usefulness of neuroscience for understanding SES is an empirical issue, and the proof will be in the pudding" [15].

Research in developmental science and other allied disciplines may, however, aid in this endeavor. Examining findings in these fields, self-regulation has emerged as an important factor, often mediating associations between child poverty and psychopathology. Self-regulation can be broadly defined as the ability to adaptively modulate one's own cognitions, emotions, and actions for the purposes of goal-directed behavior [18,19]. Underscoring the importance of self-regulation, a growing body of research has found that greater levels of this construct in early or middle childhood are related to more positive mental health across the lifespan. For example, longitudinal work in the Dunedin cohort has found that higher observational ratings of self-regulation between ages 3 and 5 significantly predicted better mental health outcomes in adolescence and adulthood, including significantly less instances of substance use and criminal offenses nearly three decades later [20]. Furthermore, adults from this cohort who were never diagnosed with a mental health disorder demonstrated significantly higher self-regulation between the ages of 3–11, compared to adults with documented psychopathology [21].

Self-regulation is developmentally important and may aid in integratively organizing neural circuits central to its components, such as attentional focus and emotion regulation. As a starting illustration, most developmental theorists argue for the separation of self-regulation into two categories: "bottom-up" and "top-down" processes [22,23]. Bottom-up mechanisms involve responses that are automatic and reactive. Top-down operations refer to deliberate and controlled thoughts or actions that are used to problem-solve, resolve conflict, or prepare for an expected objective. Nigg [19] further deconstructed top-down processes into separate executive functions including cognitive control and cognitive flexibility. Inspired by this idea, we can apply a similar heuristic to neurobiology, discussing top-down versus bottom-up processes and then drilling into differences for youth living in poverty compared to their more affluent peers. Such an approach may then allow for a more common framework for research on poverty and SES-related disparities.

Bridging neuroscientific investigations focused on poverty and lower SES to developmental science and other allied disciplines may abate limitations common to neuroimaging work. For example, a majority of the f/MRI studies focused on poverty are cross-sectional in nature, with small and homogenous samples. Neuroimaging work is also limited in its ability to examine bidirectional pathways and to generate clear developmentally-rich answers about mechanisms and precise pathways of risk. Moreover, a majority of neurobiologically informed studies are still working to control for the many confounds potentially present in samples that span the SES gradient. For instance, a great number of the studies do not control for psychopathology or account for proximal processes associated with poverty. Integrating constructs from developmental science and other allied disciplines may improve usefulness of neuroscience, as such fields have made important progress in understanding linkages between child poverty and negative mental health. Thus, we may be able to gain additional insights into understanding associations between poverty/lower SES and psychopathology by anchoring the neuroscientific studies of poverty with related research in developmental science and other allied disciplines.

2.2. Overview of commonly used neuroscience methods

Before reviewing the growing body of studies focused on neurobiology and poverty, it is important to provide some basic details of different methodologies for those unfamiliar or new to this research area. Each measure has different strengths and limitations that are important to note. In order to more precisely investigate the structure of the brain, researchers have employed magnetic resonance imaging (MRI). Structural MRI produces spatially rich images of brain anatomy where the volumes of regions are measured. Areas can be bigger or smaller in (gross) size, with alterations in cellular components of the brain (e.g., neural cell bodies, dendrites and synapses) likely generating these differences. Structural MRI, however, does not provide information about brain activity during a specific cognitive process. Regions may differ in volumes but be more or less active when a participant is making a decision, processing a reward, or engaging in other behaviors. Another type of MRI modality, diffusion-weighted imaging (DWI), probes structural connectivity in the brain through assessment of

water diffusion in brain white matter. One of the most commonly derived measures in DWI is fractional anisotropy (FA); FA describes the directionality of water diffusion and is modulated by microstructural properties of white matter, including fiber density, axonal diameter, and myelination. Lower FA may be indicative of white matter being more diffusely organized, connecting to portions of the brain in an equivalent fashion, or reflect reduced myelinated axons. More diffuse white matter connections, lower axonal density, or reduced myelination could impede brain functioning. However, like structural MRI, DWI does not measure brain functioning. Researchers using these MRI tools believe that brain volume and structural connectivity may be slow to change over time, speaking to trait-like differences in neurobiology.

To understand brain function, scholars can leverage different types of electroencephalography (EEG) and functional MRI (fMRI). EEG measures electrical activity on the head that is partially reflective of activity of neurons and the brain's other basic machinery. EEG often measures on-going electrical activity without a task, while event-related potentials (ERPs) specifically probe electrical activity in response to a particular stimulus. EEG and ERPs have incredibly precise temporal resolution (on a millisecond time scale), but poor spatial resolution. Electrical activity is measured on the surface of the skull but is generated locally in brain regions. The signal must then travel through other portions of the brain and the skull (blurring things further) before being measured at the scalp. This makes spatially locating signal more difficult. In fMRI, differences in blood oxygenation is measured and this is highly correlated with actual neuronal activity [24]. fMRI assays the brain's response to a specific set of stimuli or tasks (e.g., working memory; face processing); for example, if an experimenter were interested in brain activity related to the processing of anger, they might present different types of emotional faces and then subtract the brain activity in response to neutral faces from the brain activity in response to angry faces. This, in theory, isolates brain activity in response to anger. In addition, fMRI can be used to simply track the brain "at-rest." Spontaneous brain activity (assessed at rest) is highly correlated between multiple brain regions. This resting-state brain activity predicts task-response properties of neural circuits, and can identify subjects' aptitude for different cognitive tasks [25]. However, the brain may still respond differently with a specific task than while at-rest. With fMRI, the signal is not blurred during its acquisition, but the time-scale of fMRI is much coarser. With rich spatial resolution, one can examine questions regarding the underlying brain function but are less able to speak about the specific timing of the events occurring in the brain. For additional discussion of these methods and application to questions in developmental science, please see references [16, 26].

Each type of method (e.g., structural MRI, EEG, fMRI) detects alterations in neurobiology for a specific brain area or sets of brain areas. We can then link back to what is known about the brain areas from a basic neuroscience perspective. For example, if structural MRI differences in the hippocampus are detected, this may mean dendrites (the branchy extension of a neuron) are shrunken in size. With the hippocampus being involved with memory recall, this could then contribute to challenges in those processes. As an additional example, lower functional connectivity between brain regions in fMRI may mean that communication between brain areas is reduced, with information being exchanged less effectively between these regions. We recommend readers examine reviews by Hackman and Farah, for additional context on basic neuroscience and SES [27,28].

3. Neuroscientific frameworks of self-regulation

In regards to measuring self-regulation behaviorally, scholars across areas of study often rely on multidimensional, composite assessments grounded in multiple levels of analysis [29]. While challenging to conceptualize neuroscientifically, important theoretical frameworks by Beauchaine and colleagues [30], Casey [31], as well as Heatherton [32], provide starting articulations about how facets of self-regulation are instantiated in the brain. Though focused on different brain circuitry, these previous neurobiological frameworks divide this construct into top-down and bottom-up processes, much like developmental science accounts. Relating to these past models, as well as influential theories of social information processing [33] and emotion regulation [34], we posit two top-down elements of self-regulation: 1) executive attention and 2) response evaluation & emotion modulation. We also propose two bottom-up facets of self-regulation: 1) salience evaluation & interpretation, and 2) stimulus generalization. Central neural hubs connected to these processes are depicted in Fig. 1. We would urge consultation of Beauchaine and Zisner's recent review [35] for a "deeper dive" into related ideas. Similar to these investigators, we believe these processes to be hierarchically organized, with top-down and bottom-up components feeding into the larger construct of self-regulation.

Thinking about child poverty and its associated stressors, it is important to note that there are dramatic periods of neurodevelopment occurring during childhood and adolescence. A growing corpus of studies has delineated how the brain exhibits both linear and nonlinear changes during childhood, adolescence, and adulthood (for review, see [36,37]). There are ongoing debates about the specific "shape" of developmental trajectories and when functions may plateau or rise at different points in development [31,38]. This topic is beyond the scope of the current review, however, it is important to note that different regions associated with bottom-up influences of self-regulation appear to structurally mature earlier in development, while top-down elements, localized primarily in the prefrontal cortex (PFC), have a slower developmental trajectory. For example, the hippocampus and amygdala reach peak volume in infancy and early childhood (for review, see [39]). In contrast, PFC volumes increases early in development, peaks near puberty, and then decreases in adolescence and adulthood [36]. Furthermore, data suggests that reactivity in bottom-up regions (e.g., amygdala, ventral striatum) increases during adolescence and diminishes in adulthood (see [31] for review).

These windows of change mark heightened susceptibility to environmental input on the brain. Research in non-human animals, where the environment can be precisely manipulated, has found an animal's own experience plays a major role in brain development (see [40]). For example, subjecting non-human animals to high-levels of stress can impact cellular and molecular elements of brain circuitry (for review, see [41]). Likewise, youth living in poverty may be particularly vulnerable given the multiple hazards to development associated with lower SES conditions (such as lower cognitive stimulation and high levels of stress).

While multiple brain alterations have been noted after exposure to child poverty, few integrative theories connect these brain differences to a behavioral framework. By organizing the neural changes seen after poverty through a self-regulatory lens, we may be: 1) more able to connect neurobiological differences to longer-term behavioral development; and 2) to form a cogent roadmap for the study of SES-disparities. Here, we review relations between child poverty and neurobiological findings in our two top-down and two bottom-up facets of self-regulation (noted above). One can think that variations in self-regulation may come from differences in either top-down or bottom-up elements (e.g., youth with lower self-regulatory skills may have lower executive attention abilities or have excessive amounts of negative salience evaluation).

3.1. Poverty and executive attention

While there are multiple elements to attentional processes (see [42] for seminal review), "executive attention" is one form of attention that may be critical for controlling our thoughts, feelings, and behaviors. Executive attention can be thought of as the broad, top-down ability to hold and use stable representations to guide behavior, while also adapting to and incorporating important incoming information.

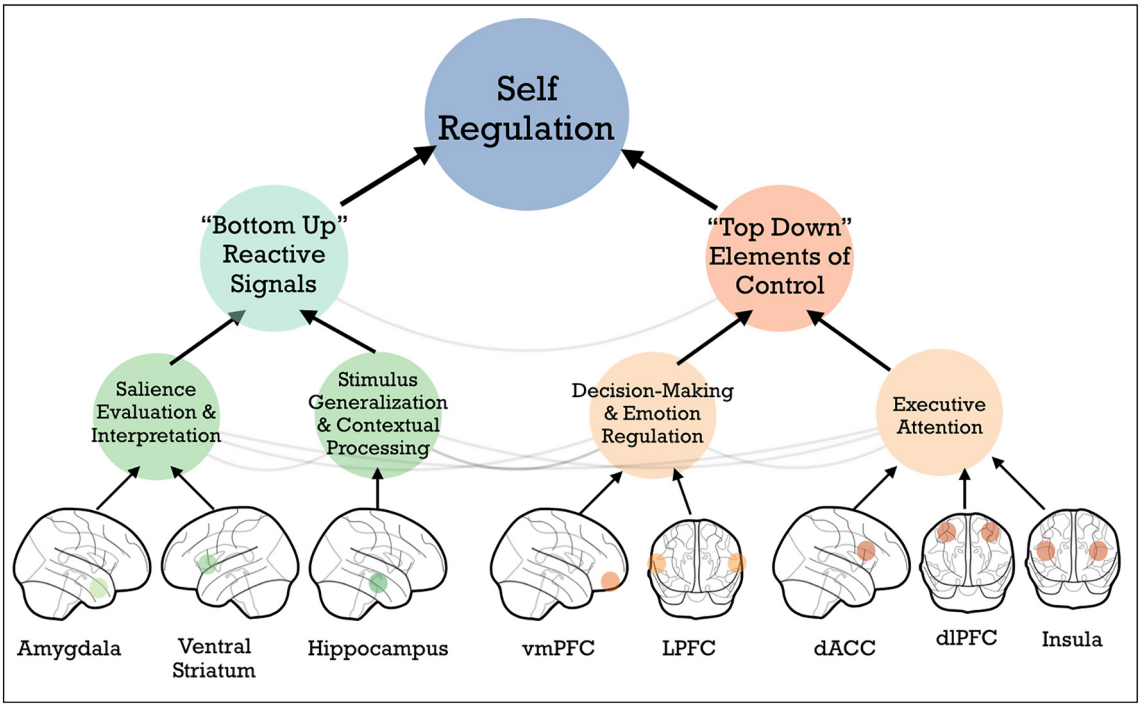

Fig. 1. Building off work by Beauchaine and colleagues [35,155], we suggest a hierarchical model of self-regulation. Two types of bottom-up reactivity, as well as two categories of top-down control, feed into a larger latent factor of self-regulation. We believe poverty impacts these processes, creating a general vulnerability that then gives way to higher rates of psychopathology. We organize past neuroscientific work on poverty based on these brain circuits and psychological processes.

Neurally, two brain networks act relatively independently to produce top-down control: 1) a fronto-parietal network and 2) a cingulo-opercular network [43] (as highlighted in portions of Fig. 1). As relevant cues start, stop, and change, the fronto-parietal control network initiates attentional control and then integrates feedback on an event-by-event (or trial-by-trial) basis. Integrity of this network is critical to rapid-adaptive control, and involves the dorsolateral prefrontal cortex (dlPFC), the precuneus, and portions of the inferior parietal lobe [42]. In contrast, the cingulo-opercular network may provide "stable set control." This network can be thought of as critical to the maintenance of task-relevant goals and includes the dorsal anterior cingulate cortex (dACC), the anterior insula/operculum (AI), and the thalamus.

In some of the earliest neuroscience work focused on poverty, researchers found lower SES was related to worse executive functioning, including differences in working memory, attention shifting, and inhibition [44]. Given that all of these behavioral processes are connected to executive attention neural networks, it is perhaps not surprising that Noble et al. [45] linked lower parental education to reduced right dlPFC volume in a sample of 1099 youth aged 3 to 20. Similar patterns have been noted in multiple independent reports [46–48]. Expanding out from brain structure, multimodal neuroimaging found that youth aged 6–19 from low SES households exhibited worse performance in a working memory task, lower functional activation in the dlPFC during the task, and lower FA for the white matter tracts that connect the dlPFC to portions of the parietal lobe [49]. These reports of lower functional brain activity also fit with research examining family income during childhood and brain activity in adulthood. For instance, lower family income at age 9 has been related to reduced dlPFC activation during different emotion regulation tasks completed in adulthood [50,51]. It is likely that these activation differences in the dlPFC reflect different executive attention processes occurring as individuals start, stop, and change their behavior. There is, however, complexity in the functional MRI results, as Finn and coworkers [52] found that youth from higher-income families showed greater activation in the dlPFC during high-load working memory conditions (2 and 3-back), while youth from low-income families exhibited greater activation at the lowest working memory load (0 back). Partially related, Sheridan and colleagues [53] found a negative association between family SES and activation of the dlPFC during a novel rule learning paradigm.

Looking at the other brain areas involved with executive attention, higher parental education has been linked with increased thickness of the right dACC in a sample of 283 children and adolescents [47]. In terms of function, early attention experiments examining ERP found that children from higher-SES families displayed higher electrical brain activity near the dACC for auditory tones that they selectively attended to, while displaying lower activity for ignored distractor tones. In contrast, children from lower-SES families displayed equal levels of electrical brain activity for the attended and unattended stimuli, suggesting challenges in filtering attention for youth living in poverty [54]. Similar patterns have been reported by this group in a more recent investigation [55], and by independent investigators working with slightly younger children [56,57]. Interestingly, and related to these results, one recent study of children aged 8–10 found that lower SES was associated with lower FA in the cingulum bundle [58]. This white matter tract connects the ACC with other brain regions, and lower FA may mark reduced efficacy in this circuit.

Developmentally, these reports are not perfectly consistent. In one fMRI study of adults aged 25–26, poor neighborhood quality measured at the age of 13 was related to enhanced dACC activation in response to social exclusion in adulthood [59]. However, current neighborhood quality was not controlled for. Longitudinal work following girls living in lower SES households has similarly demonstrated increased ACC activation during a behavioral inhibition task over a two-year period [60]. Increases in ACC activation over time predicted worse task performance. Furthermore, lower SES girls had major changes in coupling between attentional networks (ACC-dlPFC connectivity), with high levels at time 1 that decreased significantly at time 2 (compared to higher SES girls).

Given that these neural circuits are active during the maintenance of task-related information and also when shifting attention, additional work is needed to more finely probe these inter-related brain regions. Investigators could use mixed block/event-related fMRI designs to

measure both sustained and transient brain activity in this neural circuitry (e.g., [61]) while also probing task-based functional connectivity during these tasks. Often missing from this work is an integration of response timing, especially trial-by-trial fluctuations. This could be important, as individuals from low SES backgrounds may be recruiting different neural processes and/or completing different neural computations (as originally suggested by D'Angiulli et al. [54]). These youth may be deploying supplementary neural resources to attend to task-irrelevant information. Thus, youth from different SES backgrounds may be making speed/accuracy tradeoffs in different ways, and this could be interrogated by using designs that modulate (or match) on performance. One further challenge in synthesizing this work is the shifting definition of poverty, with past researchers using household income, income-to-needs ratios, parent education, and youth qualification for free or reduced-price lunch at school. Greater consistency in definitions of poverty/low-SES would aid in ruling out these inconsistencies as drivers of the complex brain and behavioral results.

In sum, youth living in poverty demonstrate structural differences in the fronto-parietal and cingulo-opercular attention networks. Functionally, reports are not perfectly uniform, as increases in brain activity have also been noted during low attentional load conditions. Nevertheless, there remains consistent suggestions that child poverty alters executive attention networks that may be central to self-regulation. This may mean a lessened ability, for example, to direct attention away from rewards when needing to delay gratification or to shift focus away from negative stimuli related to anger and aggression (see [62], as detailed in [63]).

3.2. Decision-making & emotion regulation neural circuitry after poverty

In addition to executive attention, other forms of top-down control are likely to be important for self-regulation. Thinking about emotion regulation, affective states can change quickly. When there is a mismatch between our emotions and a given situation, we may try to alter our emotions to increase the likelihood that we can reach a targeted goal [34]. For example, explicit emotion regulation might involve reappraising the meaning of negative pictures to lessen or to stop the negative affect elicited (e.g., seeing a crying woman outside of a church, as attending a wedding rather than a funeral). During this type of effortful regulation, subjects have reported less negative affect and have shown increased activity in the lateral PFC (see recent meta-analysis, [64]). Connected to value-based decision-making, if a person decides to pursue a goal, they may consider both the perceived value (e.g., enjoyment) and costs (e.g., effort required) of the goal [65]. These subjective elements then critically determine the behaviors pursued. A growing body of research suggests that portions of the ventromedial prefrontal cortex (vmPFC) are involved in making choices between stimuli with different types or amounts of value, including making decisions about monetary, social, and food rewards [66].

In research focused on child poverty, multiple studies have noted differences in the vmPFC and to a lesser extent, the lateral PFC. For example, investigators found that exposure to poverty at 3-months of age, but not at age 25, was associated with smaller vmPFC volumes in adulthood ($n = 362$) [67]. Functionally, several studies have observed that child poverty alters activation in the default mode network (DMN; a network of brain regions, including the vmPFC and other PFC regions that interact during wakeful rest). Sripada et al. [68] found that exposure to poverty at age 9 was associated with reduced DMN connectivity in a sample of adults, when controlling for current income, race, perceived social status, and depression/anxiety symptoms. Family income and maternal education have similarly been associated with DMN maturation early in development, in a sample of infants [69].

SES-related measures have also been linked to differences in vmPFC activation during fMRI tasks. In a sample of adults aged 31–54, lower parental education (by age 18; retrospectively reported) predicted reduced dACC-vmPFC and dlPFC-vmPFC connectivity during positive feedback in a reward task [70]. This finding remained significant after controlling for each participant's current age, sex, education, income, SES, alcohol use, and depressive symptoms. Another study found that, independent of adult income, exposure to poverty at age 9 was associated with increased vmPFC and amygdala activation in response to threat faces compared to happy faces [71]. However, child poverty was associated with decreased functional connectivity between the vmPFC and left amygdala. Such patterns would fit with Dufford et al. [58] who found reduced FA in white matter tracts connecting the vmPFC and amygdala. Again, lower FA may mean lower integrity and efficacy of this brain circuitry.

Comparably, studies have noted poverty-related differences in the lateral PFC. Kim and colleagues [50] found that adults exposed to poverty at age 9 demonstrated reduced lateral PFC activity during an explicit emotion regulation task. These findings fit well with work by Gianaros et al. [70], who found associations between higher retrospectively-reported parental education and greater lateral PFC activity during the processing of positive feedback in a reward task. Similar patterns have been noted in ERP work focused on attentional processing in children aged 7–12. In this work, Kishiyama et al. [72] found frontal ERP components were lower in children from low SES backgrounds compared to those from high SES backgrounds, and this was localized to more lateral PFC. One cautious note however, is that race was notably mismatched between the two groups and not controlled for.

Considered together, these findings link child poverty with smaller vmPFC volumes, and lower structural and functional connectivity between the vmPFC and other regions of the brain. While some studies find relations between child poverty and higher functional activation in the vmPFC, many consistently report reduced functional activity of the vmPFC and lateral PFC during different experimental tasks. This impaired functionality may mean that disadvantaged youth may be less effective when explicitly regulating emotion or making value-based decisions. While these patterns are promising, it is important to note that no work to date has examined task-based fMRI activity related to the vmPFC and lateral PFC in youth samples specifically. Groups have used ERPs and resting state fMRI in children, but all the other reviewed work relate brain function in adulthood to measures of childhood poverty. This is a major shortcoming, as differences in vmPFC and lateral PFC could be emerging later in life and are potentially more “consequence” than “cause” (in terms of impaired self-regulation).

3.3. Salience evaluation & interpretation neural hubs in low SES samples

After someone deploys attention, individuals must next work to understand stimuli and experiences as positive or negative, rewarding or threatening. These bottom-up elements may contribute to hyper/hypo-vigilance, or changes in motivation. The approach or avoidant behavior that accompany these changes in motivation feed into self-regulatory abilities. A central neural hub for vigilance is the amygdala. This subcortical brain region mediates the learning of associations between an unconditioned stimulus (like a shock) and a conditioned one (like a light or tone that predicts the shock [73,74]; as reviewed in [75]). fMRI studies in humans find that the amygdala is activated by a number of negative emotions, including the processing of anger and fear [76,77]. Another subcortical structure, the ventral striatum (VS), is critical to incentive motivation. A vast array of research indicates that this region supports reward responsiveness and learning [78]. The VS displays higher activity depending on different dimensions of reward including magnitude, probability, effort, and delay [79]. Thinking about classic work by Mischel (and the “marshmallow test”), multiple studies have now found an association between higher levels of VS activity during different reward paradigms and a preference for smaller immediate rewards (over larger delayed amounts of money) [80]. We believe this reward and vigilance processing can be broadly referred to as “salience evaluation & interpretation.”

Relating to child poverty, conflicting findings have been reported regarding exposure to poverty and amygdala structure. There have been reports of smaller left amygdalae for impoverished youth [81,82], larger amygdala volumes in adults who grew up in low SES households [83], and multiple reports of no relation between poverty and structure in this region [45,84,85]. As we suggest in [86], it is likely that there are non-linear effects of exposure to poverty on amygdala volumes, dependent on the levels of stress and the timing of such exposures. In terms of amygdala function, there is more consistent evidence showing an association between child poverty and increased adult amygdala activation. This is true for lower perceived social standing [87], as well as lower family income at age 9 [50].

In regards to reward-related neural hubs, one cross-sectional study of 1082 youth aged 3–21, found that lower family income was associated with lower FA in the right superior corticostriatal tract, a white matter bundle connecting portions of the VS to sub-regions of the PFC [88]. These patterns fit with recent work showing that food insecurity, one form of deprivation associated with poverty, relates to worse performance in a reward processing task [89]. In this same sample of youth aged 6–19, greater food insecurity correlated with reduced FA in the left anterior limb of the internal capsule (a white matter tract that includes the VS). Lower integrity of this tract may mean less effective processing of reward information, which may impair the adaptive guidance of behavior. Of note, no reports to date have examined impacts of poverty on task-based functional activity of the VS. This is surprising given that structural connectivity would suggest aberrant organization of the broader circuit that the VS is nested in.

Abnormal functional connectivity between the VS and other brain areas further motivates a future focus on the VS. Work by Romens et al. [90] found that the total number of years that a child's household was a recipient of public assistance was linked to heightened dmPFC activation during reward anticipation in a sample of girls aged 5–16 [90]. Relatedly, resting state work by Marshall et al. [91] found that youth aged 6–17 from low SES conditions (as indexed by higher community distress) had reductions in medial PFC-VS functional connectivity, when controlling for interpersonal adversity and internalizing symptomatology.

In sum, research examining the effects of child poverty on the structure of the amygdala appear to be inconsistent at first glance. There is, however, reasonable evidence to show that poverty relates to heightened amygdala activity. Such patterns may mean greater negative affect or more rapid learning during some emotional situations. For the VS, there is limited work specifically on poverty and brain activity in this region. However, there is evidence of alterations in functional connectivity in the broader circuit that the VS is nested in; this might connect to aberrant use of reward signals to guide behavior. Additional work is needed to understand if poverty or low SES impacts these brain areas. Similar to shortcomings noted above, much of the work reviewed here associates brain function in adulthood to measures of childhood poverty, which limits our understanding of neurodevelopment in low-SES youth. Clarification of these patterns would be useful and would potentially connect to programmatic lines of research in developmental psychology. For example, classic work by Dodge and colleagues [33,92,93] on hostile attributional biases (connected to the amygdala), and delay of gratification studies by Mischel and colleagues [94–96] (associated with the VS), relate to the development of self-regulatory processes (or the lack thereof).

3.4. The Hippocampus, generalization, context, and exposure to poverty

After salience is determined, past experiences are drawn upon to guide behavior in service of goals. These different associations may be activated due to memory representations, and this bottom-up process may then impact other elements of self-regulation. Imagine a child excluded from a social group (e.g., from a sports team); that child may store the event either as an act of willful rejection by the peer group or as an inadvertent slip-up. These memories, however, can then bias future interactions with peers and influence future decisions (as noted in Crick and Dodge [33]). Multiple neuroimaging studies have found that the hippocampus is central to these processes and other elements of memory [97,98]. Furthermore, successful generalization of memories and learned experiences to new stimuli is associated with changes in learning-phase activity in the hippocampus [99]; all of which may impact self-regulation.

One of the most replicated findings in the neuroscientific work focused on child poverty is smaller hippocampal volumes for low-SES youth. In one of the first reports of this association, Hanson, Chandra, Wolfe, & Pollak [84] found that low family income significantly related to smaller hippocampal volumes in a sample of 317 youth aged 4–18. Similar findings were reported in a large cohort of 1099 youth aged 5–17, with lower income-to-needs ratio relating to reduced hippocampal volume [100]. These patterns have been replicated by a number of groups [46,81,82,101,102]. Sadly, these effects appear to emerge as early as infancy, as Betancourt and colleagues [103] found that 5 week old infants from low SES households had smaller volumes in the hippocampi. Underscoring the developmental sequalae of poverty, Hair and colleagues [104] leveraged longitudinal MRI to construct models of structural growth in a sample ranging from 4 to 22 years old. These investigators found lower SES youth had hippocampal volumes that were 3 to 4 percentage points below the developmental norm of the sample, with the lowest SES children presenting with volumes that were 8 to 10 percentage points below the norm.

Child poverty has also been linked to deviations in the function of the hippocampus at all stages of development. One longitudinal sample found that lower income-to-needs ratios at preschool age was associated with reduced resting state functional connectivity between the hippocampus and amygdala at school age [105]. Interestingly, low income-to-needs ratio at preschool age was associated with greater negative mood and depression severity at school age, and this relation was mediated by left hippocampus-right superior frontal cortex resting connectivity. Task-related fMRI studies similarly note associations between SES-related factors and hippocampal activation. One study of children aged 8–12 found that lower mother-reported perceived social status was related to lower hippocampal activation during the recognition phase of the paired associate learning task [106]. Another study found that adults with lower income-to-needs ratio at age 9 had reduced hippocampal activation overall during an emotion regulation task [51].

Surveying this body of studies, one sees a clear picture of structural and functional alterations in the hippocampi of youth exposed to poverty. Such differences may be due to the Hypothalamic-Pituitary-Adrenal (HPA; "stress") axis and cortisol (as reviewed in [107]). Sustained elevated HPA axis activity may cause dendritic remodeling and neuronal death in the hippocampus and other brain regions [108]. Theories about what these neural differences may mean behaviorally however, remain lamentably incomplete. Many would say the hippocampus is clearly involved with long-term memory, so the differences should/could be related to that, but this has not been commonly reported. We believe future work focused on child poverty and the hippocampus would be well-served by translating the exciting ongoing work and emerging theories of hippocampal functioning, arising from studies in basic cognitive neuroscience (e.g., [109]). Use of functional paradigms focused on different forms of learning, contextual processing, and decision-making could break new ground in understanding the implications of hippocampal alterations after poverty.

3.5. Summarizing neurodevelopmental alterations after poverty

The different studies reviewed are slowly beginning to answer the question of "where" (in the brain), which can be important and informative. Pulling out common patterns across these studies, we believe there is suggestive evidence that child poverty alters the various

neurobiological hubs involved with attention, emotion, reward, and memory. We also believe these neurobiological variations index aspects of self-regulation (bottom-up reactivity/top-down control) and overtime, these differences give way to challenges in mental health. These different brain areas have extensive postnatal developmental trajectories, making them particularly susceptible to environmental influences. Indeed, in terms of normative brain development and self-regulation, research supports the idea that brain regions relating to bottom-up processes, including the hippocampus and amygdala, mature early in development while regions associated with top-down control, such as portions of the PFC, continue to develop into adulthood. Child poverty and its related stressors may impact these brain regions as youth are developing.

Looking at these different facets of self-regulation, consistent reductions have been noted in the structure and function of the dlPFC, as well as multiple reports of differences in the dACC for samples living in or exposed to child poverty. However, further work is needed to deeply probe these systems, as open questions exists about the maintenance versus the switching of attentional processes over time. In terms of emotion-regulation and decision-making, child poverty is linked to smaller vmPFC volumes, decreases in functional activation of the lateral PFC, and reduced connectivity between the vmPFC and other regions of the brain. This may mean value-based decision-making and explicit, effortful emotion regulation is less effective or less sustained in low SES groups. Child poverty is also consistently linked with increased amygdala activation, and decreased VS connectivity with portions of the PFC. Most consistently, child poverty relates to compromised hippocampal structure, activation, and connectivity with other neural systems. If one conceptualizes self-regulation (or failures in self-regulation) as emerging from these various neural hubs, the multitude of neural differences reviewed above may have important developmental implications for mental health outcomes. Changes in these brain areas could represent a "neural equifinality," as different profiles of neural alterations may all lead to the same developmental endpoint—challenges in self-regulation and ultimately, worse mental health.

However, this work also creates more questions, most notably–"when" and "how" these differences are occurring. Pertaining to developmental timing (and "when"), very few studies have been completed in youth samples. A large proportion of research reviewed here has connected adult brain functioning to exposure to child poverty or low childhood SES. While these studies could begin to speak to developmental timing, there may still be issues in determining what is cause and what is consequence. Given that exposure to poverty in either early and middle childhood is associated with significant increases in psychopathology, there may be potential confounds in such an approach. The majority of studies measure brain functioning after this developmental period and often psychopathology is not controlled for in these reports. Regarding the "how" and questions of mechanisms, this limited body of work uses shifting definitions of poverty, creating additional challenges for interpreting results and for building a collective knowledge base with such variability. While much more work is needed (and we provide recommendations in section 6), there is however interesting work on the hippocampus and poverty. Luby and colleagues [81] found that poverty was associated with hippocampal volumes, and that this effect was mediated by caregiving support/hostility, as well as stressful life events. It will be important to mirror studies like this moving forward, sharpening a focus on potential intervening (or indirect) effects related to poverty/low-SES.

4. Connecting exposure to poverty to behavioral differences in self-regulation

While neuroscientific investigations suggest brain differences related to different self-regulatory processes, longitudinal behavioral work focused on self-regulation and poverty may answer many questions that neuroscience cannot truly speak to. Indeed, challenges common to poverty (e.g., lower cognitive stimulation; higher rates of community violence) may cause alterations in top-down and/or bottom-up self-regulation behavioral processes.

From a top-down perspective, beautiful work by Roy, McCoy, and Raver [110] examined how facets of self-regulation changed when children experienced a move into or out of poverty during early or middle childhood. These investigators found that children who experienced a moved out of poverty had higher teacher-reported self-regulation and better executive functioning (on a computerized task assessing working memory, inhibitory control, and attention set shifting) in 5th grade compared to children who stayed in poverty. The reverse was also significant, such that children who experienced a move into poverty during early or middle childhood had lower teacher-reported self-regulation and worse executive functioning. We can think about this as indexing top-down facets of self-regulation. Similar patterns have been noted in behavioral work completed in a large sample ($n = 2402$) of 2–4 year old children. In this study, each additional exposure to sociodemographic adversity was associated with subsequent drops in performance on effortful control tasks, an element of top-down self-regulation, 16 months later [111].

Regarding bottom-up facets of self-regulation, developmental science work leveraging measures of the parasympathetic nervous system and the HPA axis may connect to the functional integrity of the amygdala and hippocampus. Interestingly, aspects of poverty have been associated with blunted sympathetic and HPA-axis reactivity [112]. This has also been reported for other aspects of physiological reactivity, with elevated systolic and diastolic blood pressure at rest in low-SES populations [113]. Such effects have sadly been found as early as kindergarten [114]. Across development, dysregulation in the parasympathetic and HPA-axis systems may be indexing hyper-vigilance and increased amygdala activity. Behaviorally, this may lead to greater negative affect and inference of hostility, edging an individual closer to a maladaptive behavioral response. Furthermore, excessive HPA-axis output may relate to impaired hippocampal structure and function. Collectively, these results drawn from developmental science suggest poverty-related impacts on top-down and bottom-up facets of self-regulation. Importantly, and complementing the neuroscience work, these data begin to suggest longitudinal effects of poverty on behavioral components of self-regulation, particularly during important developmental periods. Moving forward, it will be important to attempt to integrate behavioral and neural markers of self-regulation in order to understand associations between poverty and poor mental health (as depicted in Fig. 2).

5. Associations between self-regulation, neurobiology and psychopathology

As alluded to previously, different forms of psychopathology are related to challenges in self-regulation. Conceptually, high self-regulation may mean better attention to relevant information in the environment and more positive regulation and reappraisal of emotion. Furthermore, as Heatherton [32] and Strauman [115] both underscored, deficits in self-regulation could lead to an unawareness of one's behavior and its consequences, an inability to detect and anticipate negative outcomes, and difficulties resolving differences between one's actual behavior and outcomes. For example, one could conceptualize depression as the loss of motivation to respond effectively to cues for reward [115]. More broadly, all of these challenges may transdiagnostically contribute to the development and maintenance of multiple forms of psychopathology, in keeping with ideas from the National Institute of Mental Health's Research Domain Criteria initiative [116]. Indeed, theorists have applied these ideas to multiple forms of internalizing and externalizing psychopathology [30,115,117], even connecting to disorders such as schizophrenia and autism [118,119].

In line with these ideas, neurobiological models of self-regulation can be applied to multiple mental health outcomes. Different forms of

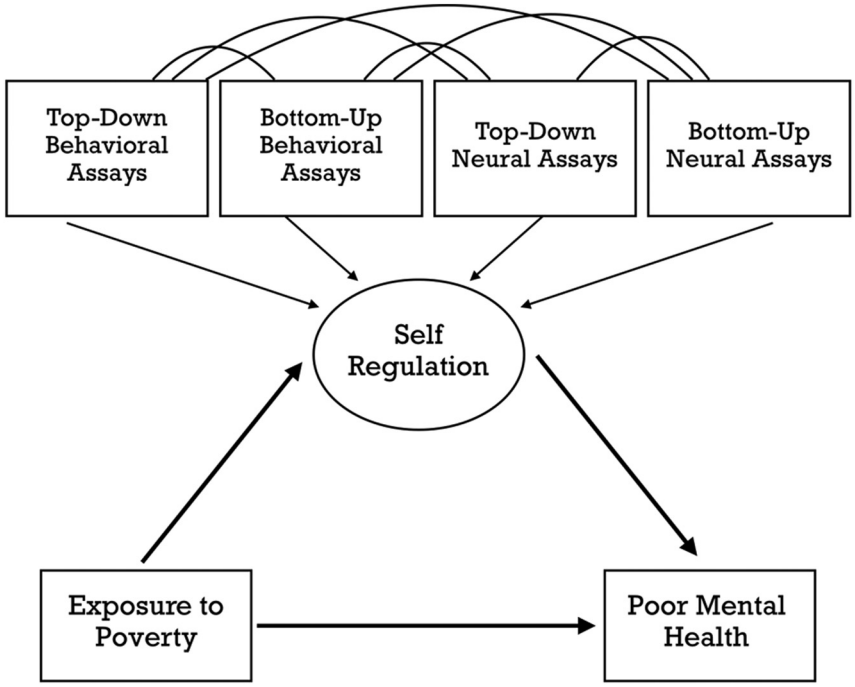

Fig. 2. A conceptual diagram showing links between exposure to poverty and poor mental health, with self-regulation shown as a potential mediator. Moving forward (and drawing from methods common to developmental psychology and developmental science), we may be able to think about "latent factors" of self-regulation that combine behavioral and neural assays of top-down or bottom-up elements of this construct.

psychopathology have been connected to alterations in the brain regions implicated in these self-regulatory skills. Thinking about top-down neurobiological circuitry (i.e., PFC subdivisions), the fronto-parietal and cingulo-opercular networks have been related to multiple forms of negative mental health outcomes [120]. For example, dlPFC volumes prospectively predict both externalizing behaviors and binge drinking in adolescence [121], while individuals with attention-deficit/hyperactivity disorder or conduct disorder show maturational lags in prefrontal development [122–124]. This may relate to impulsivity or compromised emotion regulation through dorsolateral, ventromedial, and lateral PFC, as well as the ACC [125,126]. From a bottom-up (neural) perspective, a large body of research suggests connections between aberrant development in subcortical regions and psychopathology. Alterations in the amygdala have been linked to major depression, anxiety, post-traumatic stress disorder, and aggression, with higher functional reactivity and smaller volumes often relating to psychopathology [127–130]. The ventral striatum has also been connected to major depression, addiction, and disruptive behavioral disorders [131–133]. Finally, alterations in the hippocampus have been connected to problems with memory and mood (for theoretical discussion, see [41]). These associations between neurobiology and psychopathology are particularly important given that these same brain regions are impacted by poverty (as detailed in Sections 3.1–3.5).

In toto, alterations in top-down (prefrontal) and bottom-up (subcortical) neural processes connect to emotional and behavioral dysregulation, culminating in externalizing and/or internalizing psychopathology. It is likely these neural alterations may confer transdiagnostic vulnerability to psychopathology. These research findings fit quite squarely with our neurobiological framework of self-regulation noted above.

6. Future directions for neuroscientific investigations of poverty

Looking holistically, there is evidence to suggest that child poverty may influence brain regions involved with top-down and bottom-up elements of self-regulation. However, additional work is critically needed at each step in the potential "causal chain" from poverty, to alterations in the brain, to reduced self-regulation, to later psychopathology.

First, while researchers have often used family income or caregiver education/occupation in relation to brain variables, these are all imperfect proxies for the multiple developmental hazards commonly noted in samples living in poverty. For example, parental education may relate to the level of cognitive stimulation a child is exposed to at home, while household income may loosely capture housing quality and home learning resources, as well as many other diverse hazards to child development. Furthermore, there may be bidirectional (direct and indirect) genetic effects impacting associations between poverty, self-regulation, and neurobiology. Recent work indicates genetic influences on educational attainment; to be concrete, if a parent has lower educational attainment, this may then relate to greater exposure to the stressors associated with poverty for their child. Recent work, in part, supports this idea as polygenetic predictors of educational attainment also related to social mobility [134].

Elements of this shortcoming could be overcome through quantitative and conceptual means. Quantitatively, one could look at tools such as "Language ENvironment Analysis" (LENA) that estimates the amount of adult words spoken to the child, conversational turns, and child vocalizations [135]. One can also think about the measurement of other environmental features common to poverty including household chaos [136], parental unpredictability [137], neighborhood violence [138] and other salient features of low SES environments. Similarly, polygenetic risk scores for educational obtainment could be integrated (when available) for studies investigating neurobiology, self-regulation, and poverty. Conceptually, two orthogonal strategies have been used in relation to this limitation—1) cumulative risk approaches and 2) dimensional approaches to adversity. Cumulative risk approaches, most notably detailed by Evans and colleagues [139], have argued that, given the multiple dimensions of poverty, no one singular measure adequately captures the full experience of this construct. Rather than attempt to disentangle very collinear elements, these theorists urge investigators to instead aggregate across the multiple developmental challenges common to poverty. This would provide high explanatory power, but less information about specific mechanisms.

Interestingly, there is suggestive evidence connecting cumulative adversity, poverty, and self-regulation (for review, see [140]). In contrast to cumulative exposure approaches, dimensional models of adversity argue that separate types of developmental hazards occur for impoverish youth and focusing on one specific form of adversity may improve mechanistic understanding of risk. A starting framework [141] argues for the difference between deprivation (absence of expected cognitive and social input) and threat (presence of a threat to one's physical integrity) in adverse contexts, like child poverty. Promising data has emerged in support of differential pathways from adversity to psychopathology (e.g., [142]), but such a framework has not been robustly connected to self-regulation to our knowledge. The field must strike a balance between more mechanistic approaches and the reality of the high co-occurrence of different stressors for those living in poverty.

Second, and related, neurobiological work, especially investigations interested in self-regulation, will need to think more deeply about the developmental timing and "depth" of poverty. Thinking about the normative development of self-regulation, it is likely that self-regulation develops hierarchically, with basic, lower-level components (e.g. working memory, attention, response inhibition) building into more complex, higher-level components (e.g. cognitive flexibility, shifting, reasoning) [19,143]. Exposure to poverty at specific developmental epochs might uniquely influence different facets of self-regulation. These impacts could be on top-down versus bottom-up facets, or on the specific elements we note for different sub-aspects of self-regulation (e.g., executive attention; salience evaluation & interpretation). Independent of this, the brain may be impacted due to continuous variations in SES or through a step-function with effects only emerging at a "break-point" (e.g., at 100% of the FPL; at 50% of the FPL). Neurobiologically, researchers have compared extreme groups ("poor" versus "not poor") and also examined continuous connections with SES. Greater clarity is needed, as certain brain circuits may show "step-functions" while others may be continuously related to SES.

Third, neurobiological investigations on the effects of poverty will need to move beyond pure "neural" description, disconnected from behavior. A large number of past studies compare low-income to more-affluent youth in terms of brain structure, function, or connectivity, report the differences in the brain, and then discuss those findings in relation to poverty. Very few studies link brain differences associated with poverty to behavioral measures, particularly those outside of the lab. For example, Hanson et al. published one of the first reports of associations between SES and the hippocampus [84]. The work reported a few brain-SES correlations but did not unpack or analyze behavior in relation to the hippocampus or other brain areas. Obviously, many fMRI studies focused on poverty consider behavior *in* the MRI scanner, but it will be important for this work to also connect to "life outside the lab." Considering different top-down versus bottom-up elements of self-regulation, it will be important to think about inclusion of self-report and task-based behavioral measures to assay more facets of behavior. Executive functioning batteries, such as the NIH Toolbox, could be used to probe aspects of top-down control involved with self-regulation. Similarly, emotion reactivity and psychophysiological measures (e.g., startle response; heart rate variability) could shed light on bottom-up influences on self-regulation. It would be particularly interesting to think about latent factors that can potentially combine behavioral and neural indices of related self-regulatory processes (as conceptually depicted in Fig. 2).

Fourth, neuroscientific work will need to probe neural circuitry in a form commensurate with the interactive, dynamic, true nature of the brain. This could involve deploying advanced analytic approaches, such as graph theory, with resting state fMRI and diffusion-weighted imaging. Approaches such as these reduce down complex systems and more precisely study system-level organization and interactions. Future work could also leverage multiple task-based fMRI measures to more robustly predict developmental challenges. For instance, investigators are now mapping brain activity during reward and threat processing using separate fMRI tasks to meaningfully probe the VS and amygdala. Interestingly, for problematic alcohol use, it was patterns of neural activation across these two tasks that most robustly predicted problem drinking, a potential exemplar of low self-regulation [144].

6.1. Overcoming past limitations by bridging to developmental science

Research examining the effects of poverty on brain differences is often motivated by greater rates of psychopathology in impoverished youth. Therefore, this corpus of work is actually, in part, confounded by psychopathology. For the most part, symptoms of psychopathology have not typically been dealt with in research, either through adding simple covariates (related to mental health) or by matching groups on rates of psychopathology. This introduces potential bias into the findings, as poverty may be increasing internalizing and externalizing symptomatology, and current mental health issues could be related to differences in the brain.

While longitudinal neuroimaging is clearly needed, bolstering connections between developmental science and neurobiological studies of poverty may lessen the impact of this limitation. For example, multiple studies suggest differences in executive attention brain circuits including the dlPFC and dACC, but these results ignore the "third factor" of participant psychopathology. However, multiple longitudinal studies in developmental science that track behavioral development have found that children from impoverished households perform worse on working memory and attentional shifting tasks [145–147]. Many of these studies have large sample sizes ($n > 1000$) and track children for multiple years of development. These longitudinal designs are particularly important, as research has noted that exposure to sociodemographic adversity leads to drops in performance on different behavioral tasks related to self-regulation [111]. By piecing together neuroscience and developmental science: 1) we can often "descriptively" identify neural circuitry impacted by poverty and relate them to important behavioral processes; 2) track corresponding behavioral processes across critical developmental epochs in larger cohorts with adequate controls for multiple potential confounds. Collectively, this approach suggests that early childhood exposure to sociodemographic risk impacts the development of self-regulation, and this has been revealed through studies at multiple levels of analysis.

6.2. Creating a "developmental science" of the neurobiological impact of poverty

Overall, neurobiologically-informed work on SES needs a deeper grounding in developmental science and allied disciplines. The future directions outlined above begin to articulate this, but only illustrate a small portion of ways to expand research to better understand how neurobiology fits into the complex "developmental matrix" of an individual [148]. In keeping with theoretical perspectives articulated by Cairns, Elder & Costello [149], it will be important to consider how variations in important neural circuitry fit into the dynamic interplay of developmental processes across timeframes, levels of analysis, and contexts. For the most part, rich work connecting the brain to contextual factors, an individual's developmental history and current experiences, etc. has not been completed with samples living in poverty. For example, no work to our knowledge has examined how child poverty may change the brain, and then how those brain changes may bidirectionally interact with current psychosocial demands to create risk for an individual. Adoption of such a framework could be useful to iron out past research inconsistencies. One could think about the conflicting patterns of dACC findings after poverty. There may be developmental adaptations created by poverty, psychosocial processes, or neurobiology that may clarify why hypo- and hyper-active patterns of function have been noted. It is possible that while individuals who develop in poverty have high dACC activity, they may also display psychosocial markers of

resiliency (or unmeasured positive neurobiology). These are open questions in need of deeper exploration.

This idea of bidirectional effects will be important in research connecting self-regulation and neurobiology. Transactions between "other-" and "self-" regulation occur throughout development, as children learn to balance feedback from external sources with their internal goals [63,150]. For example, the self-regulation skills needed for a child to learn how to read may be influenced by the other-regulation of a parent encouraging and supporting their child in this endeavor. In adolescence, other-regulation from parents, friends, and peers could bidirectionally strengthen or weaken self-regulatory abilities [151]. High self-regulation is associated with high quality, supportive relationships, which in turn further promotes the development of self-regulation. In contrast, low self-regulation is associated with low quality of life, unsupportive relationships, and this in turn further reduces self-regulatory abilities [151]. Given that self-regulation unfolds through the interaction of multiple influences, it will be important to think about bolstering biopsychosocial models and deeply considering the "person-in-context" in neurobiological research. Motivated by ideas in developmental science, it will be important to think about connecting neurobiology to behavioral measures in multiple contexts (e.g., school; home; with peers). Few, if any, investigations have reported consistent patterns of brain-behavior correlations across contexts. Regardless of whether there would be continuity in the results, this information would be informative to understanding developmental processes.

In the strictest sense of developmental science, neuroimaging would be integrated into longitudinal investigations. Ideally, there would be multiple measures of brain functioning. Realistically, this may be challenging given the high costs associated with MRI scanning. With or without longitudinal data, those employing neuroimaging and focused on SES could start to think about an evolution of analytic perspectives. As illustrated in Fig. 3, the preponderance of neurobiologically-informed work (if it links to behavioral measures at all) does so in a (somewhat) disconnected fashion. There will be correlations between child poverty and the brain, as well as separate correlations between the brain and some outcome behavior (Fig. 3; Panel A). Given that all data is typically cross-sectional, this raises questions of directionality and causality as noted above. With larger sample sizes, even with cross-sectional measures of the brain, it will be important for investigators to expand current approaches, investigating the "brain as a moderator" (Fig. 3; Panel B). Rather than attempt to speak to causal mechanisms, which is difficult with cross-sectional data, thinking of the brain as a moderator might be a more reasonable starting point. Such interactions would begin to answer questions of "for whom" and identify subgroups of individuals based on psychosocial and neurobiological variables. For example, perhaps heightened amygdala activity in the context of exposure to poverty significantly predicts externalizing psychopathology; rather than simply correlating poverty with brain functioning, we can think about exploring the interaction of brain variables and poverty exposure to understand mental health disparities. As more longitudinal data becomes available, neuroimaging may pivot to examining the "brain as mediator" (Fig. 3; Panel C) and truly speak to mechanisms, deepening our understanding of "how" poverty leads to psychopathology.

Looking to developmental science research, different conceptual approaches have treated self-regulation as a moderator or a mediator. Some research groups have noted a moderating effect of self-regulation, such that children living in poverty who had high levels of self-regulation experienced lower mood and behavioral problems compared to those who had low levels of this skill [152,153]. In contrast, low-SES youth who participated in a family-centered intervention aimed at reducing problem behaviors exhibited greater growth in

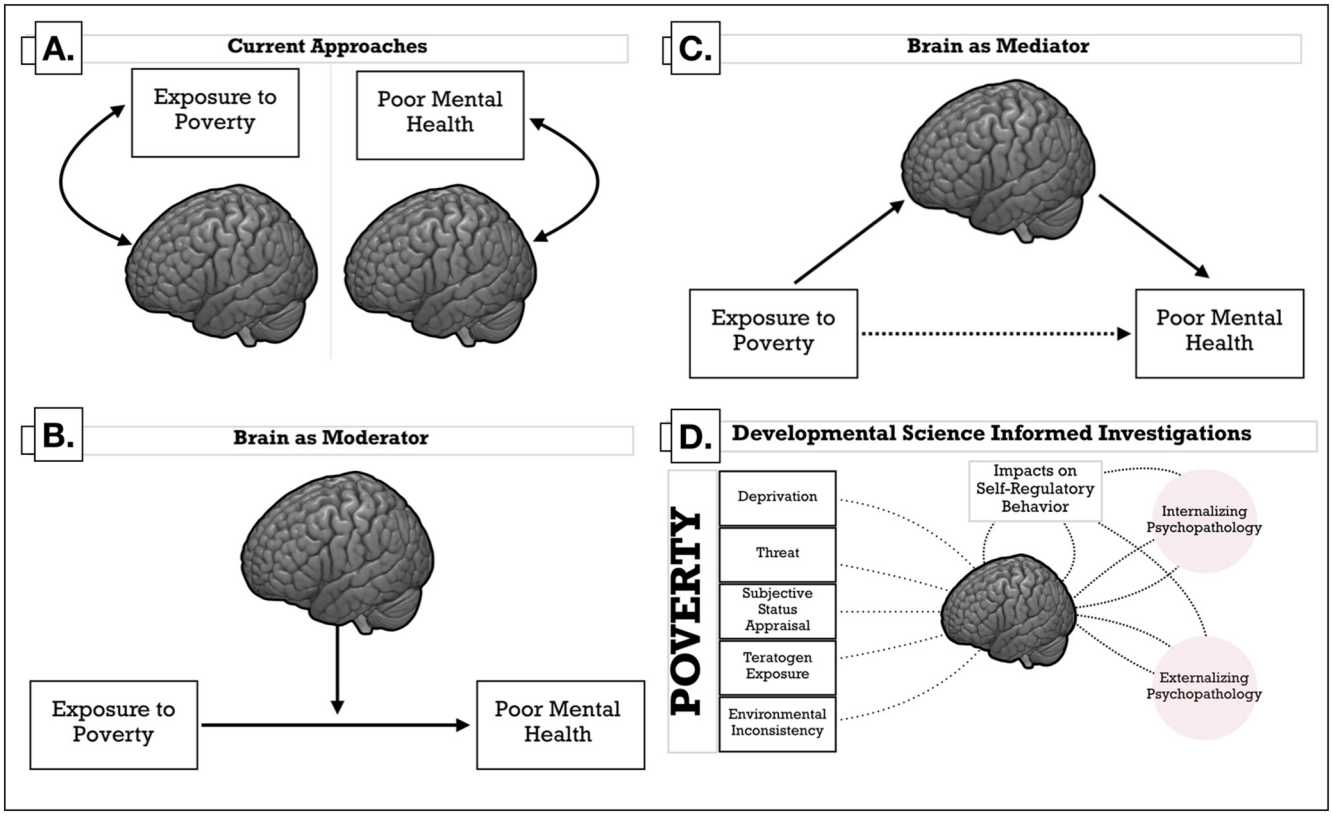

Fig. 3. The potential evolution of analytic perspectives for neuroscientific work focused on poverty. As a starting point, much of the work focused on poverty and the brain examines associations in a disconnected way (top left, panel a). However, neurobiologically informed work can strive to leverage more complex analytic techniques (bottom left, panel b: brain as moderator; or top right, panel c: brain as mediator). As the discipline matures, we hope to reach what is illustrated in panel d (bottom right)— complex analytic models that consider the multiple facets of poverty, bidirectional associations between current behavior and the brain, and longitudinal associations with multiple forms of psychopathology.

parent-rated inhibitory control, which was found to indirectly effect teacher-reported oppositional defiant behavior [154]. This would suggest a mediating role for self-regulation, partially explaining connections between poverty and forms of psychopathology.

In Fig. 3, Panel D, we depict what neurobiologically-informed research might look like if it truly embodied developmental science principles. There would be a push to understand how the multiple risk factors associated with poverty independently and interactively impact the brain and self-regulation, a focus on how the development of brain and self-regulation may bidirectionally influence one another, and how these neural and self-regulatory processes then give rise to psychopathology. Given that many brain regions are connected to multiple mental health outcomes, it will be useful to also connect these developmental principles of equi- and multi-finality.

7. Conclusions

Exposure to child poverty has been consistently linked to negative mental health outcomes. However, the mechanisms that convey this risk remain unclear. Here, we present self-regulation as a critical factor in this deleterious association, as poverty negatively impacts self-regulatory systems, which in turn contribute to the development and maintenance of psychopathology. We provide a starting framework for conceptualizing self-regulation neurobiologically. We detail research on the effects of poverty on the brain within this framework. We believe that the use of this model can greatly aid in better understanding the mechanisms leading to SES disparities in the brain, self-regulatory behavioral processes, and psychopathology. This interdisciplinary approach can serve as a junction point between developmental psychology and neuroscience, in order to identify more specific pathways between child poverty and gaps in cognitive/socioemotional domains. Ultimately, we hope to foster greater psychological well-being for all youth in our society, regardless of social class, which is a goal we believe that all should be striving towards.

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