Neural Mechanisms Underlying the Impact of Low Socioeconomic Status on Self-Regulation

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Abstract

Self-regulation refers to the capacity of individuals to monitor and modulate their own cognition, emotion, and behavior to achieve goals and adapt to ever-changing environments. It functions across diverse domains including achievement, interpersonal relationships, and health, and is regarded as fundamental to human success and well-being. However, extensive research has demonstrated that lower socioeconomic status (SES) is associated with poorer self-regulation abilities. To enhance self-regulation among individuals of low socioeconomic status, it is imperative to thoroughly investigate the mechanisms underlying the impact of low SES on self-regulation. Neuroscience offers unique and crucial insights in this regard: low SES modifies the structure and function of the dorsolateral prefrontal cortex, cingulate gyrus, ventromedial prefrontal cortex, amygdala, hippocampus, and ventral striatum, consequently influencing various components of self-regulation (cognitive regulation, emotion regulation, and behavioral regulation). Future research should not only more rigorously examine each causal chain within these neural mechanisms, but also integrate neurobiology with developmental psychology to elucidate the distinct mechanisms through which low SES affects self-regulation across different developmental stages. Additionally, attention should be devoted to the adaptiveness of specific neural and behavioral responses exhibited by low-SES individuals, thereby providing a foundation for developing systematic, sustained, and effective intervention programs.

Full Text

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Abstract

Self-regulation is the capacity to monitor and modulate one' s cognition, emotion, and behavior to achieve goals and adapt to changing environments. It plays a crucial role across diverse domains including academic achievement, interpersonal relationships, and health, and is widely regarded as fundamental to human success and well-being. However, extensive research has demonstrated that lower socioeconomic status (SES) is associated with poorer self-regulation abilities. To effectively enhance self-regulation among individuals from low-SES backgrounds, it is essential to thoroughly investigate the mechanisms through which low SES influences self-regulation.

Neuroscience offers unique and critical insights into this question. Low SES alters the structure and function of several key brain regions that support self-regulation, including the dorsolateral prefrontal cortex, cingulate gyrus, ventromedial prefrontal cortex, amygdala, hippocampus, and ventral striatum. These neurobiological changes subsequently affect the various components of self-regulation—cognitive regulation, emotional regulation, and behavioral regulation. Future research should not only examine each causal link in these neural mechanisms more rigorously but also integrate neurobiology with developmental psychology to uncover stage-specific mechanisms of SES effects on self-regulation across different developmental periods. Additionally, research should attend to the adaptive nature of specific neural and behavioral responses observed in low-SES individuals, and develop systematic, sustained, and effective intervention programs based on these mechanistic insights.

Keywords: low socioeconomic status, cognitive regulation, emotional regulation, behavioral regulation, neural mechanism

In the historical stage of promoting common prosperity, low-income groups represent a key target population for support and assistance [**?**, **?**]. Enhancing the self-development capacity of these groups constitutes an important pathway to achieving common prosperity [**?**, **?**]. Self-regulation represents a core component of self-development capacity, exerting powerful and broad influences across achievement, interpersonal relations, and health domains. It promotes positive behaviors while preventing maladaptive ones, making it essential for human success and happiness [**?**, **?**]. Improving self-regulation among low-SES individuals could increase human capital, raise incomes, and reduce healthcare costs, thereby contributing to shared prosperity. However, numerous studies have shown that SES significantly affects self-regulation abilities [**?**, **?**, **?**, **?**, **?**]. For example, children who moved out of impoverished neighborhoods showed significantly improved teacher-reported self-regulation and performance on computerized tasks measuring working memory, inhibitory control, and attention shifting by upper elementary school, compared to those who remained in poverty.

Conversely, children who moved into high-poverty neighborhoods during early or middle childhood exhibited significantly reduced self-regulation by upper elementary school [?, ?]. Longitudinal research further indicates that lower family SES predicts slower development of self-regulation in children [?, ?]. The detrimental effects of low SES extend beyond childhood to affect self-regulation in adolescents [?, ?, ?, ?, ?], adults [?, ?, ?, ?], and middle-aged and older adults [?, ?, ?]. To improve self-regulation in low-SES populations, we must thoroughly investigate the mechanisms underlying these effects. Since the brain serves as the primary pathway through which environmental influences affect psychology and behavior, neuroscientific methods that observe specific behavioral, cognitive, and affective brain processes provide unique and irreplaceable insights [?, ?]. This paper therefore adopts a cognitive neuroscience perspective to address the question of how low SES influences self-regulation.

1. Self-Regulation

Self-regulation is defined as the ability to monitor and adjust one' s cognition, emotion, and behavior to achieve goals and adapt to changing environments [?, ?, ?]. This definition emphasizes that self-regulation is a goal-directed capacity that extends beyond simple impulse control, demonstrating flexibility and adaptability to situational demands and social norms [?, ?]. The definition identifies three components: cognitive regulation, emotional regulation, and behavioral regulation. Cognitive regulation refers to the adjustment of goal-directed cognitive processes independent of behavioral or emotional regulation [?, ?], typically encompassing three core executive functions: inhibitory control, working memory, and cognitive flexibility [?, ?]. Emotional regulation involves capacities to influence the occurrence and characteristics of emotional responses, including actively managing intense and unpleasant affect and maintaining adaptive functioning during emotional arousal. Common strategies include cognitive reappraisal, expressive suppression, and attentional distraction [?, ?]. Behavioral regulation involves optimizing goal-directed overt physical actions—the seamless coordination of attention, working memory, and inhibitory control with motor or verbal functions to produce observable behaviors, including rule compliance, delay of gratification, persistence, impulse control, conflict resolution, and proactive coping strategy formulation [?, ?, ?].

These three components represent distinct yet interrelated dimensions of self-regulation [?, ?, ?, ?]. They are relatively independent [?, ?, ?] but also mutually influential [?, ?, ?, ?, ?, ?]. Behavioral regulation emerges from the integrated balance of cognitive and emotional regulation [?, ?]. Specifically, individuals regulate emotional valence and arousal to facilitate executive function engagement during goal-directed action [?, ?]. For example, children who learn to regulate emotions and stress while acquiring classroom rules become better at managing boredom or frustration, which promotes executive function activation and use. In turn, effective executive function facilitates emotion and stress regulation, creating a positive cycle that improves task completion and inhibition

of inappropriate behaviors [?, ?].

2. Low Socioeconomic Status Affects Self-Regulation

Large-scale cross-sectional surveys, longitudinal studies, field experiments, and laboratory experiments have consistently demonstrated that socioeconomic status significantly influences various aspects of self-regulation.

Numerous correlational studies reveal that lower SES is associated with poorer self-regulation. In a study examining the relationship between low family income and executive control (a core component of executive function), Ruberry et al. [?, ?] tested 118 preschoolers from diverse income backgrounds, measuring income-related stressors, cumulative risk, and financial security. Using a neurophysiological battery of six tasks (e.g., Day-Night task, Head-Toes-Knees-Shoulders task) and two computerized tasks (Frog/Fish task and Flanker task) to assess executive control, they found positive correlations between family income and executive control performance, with low-income preschoolers showing poorer performance. Similar patterns emerge in emotional regulation research. Parent-report data indicate that compared to high-SES children, low-SES children struggle with sadness, worry, and withdrawal from early to middle childhood. In emotion regulation processes—including attentional bias to threat, facial emotion evaluation, and negative affect—low-SES children show significantly reduced ability to orient and sustain attention to neutral and threatening stimuli and to accurately evaluate facial information compared to their high-SES peers [?, ?]. In behavioral regulation research, individuals raised in poverty tend to focus on the present and exhibit higher temporal discounting rates compared to those from more affluent backgrounds [?, ?]. Increased economic stress significantly correlates with reduced delay of gratification [?, ?]. For instance, a large-scale study of 42,863 UK adults examined the relationship between social class and intertemporal decision-making using a single-item choice paradigm (e.g., "£45 in 3 days vs. £70 in 3 weeks"). Results showed that low-SES individuals more strongly preferred the immediate option [?, ?].

Longitudinal evidence further illuminates the impact of low SES on self-regulation. In a four-year longitudinal study investigating the relationship between low SES and children' s executive function, researchers tracked 1,259 children from low-income families from birth through 7, 15, 24, 36, and 48 months. Using income-to-needs ratio, economic pressure, and housing quality to measure SES and related risks, and span working memory, spatial conflict inhibitory control, and item selection attention flexibility tasks to assess executive function, they found that children experiencing low SES for more years showed poorer executive function performance [?, ?]. A six-year longitudinal study replicated these findings with 602 low-SES children, examining how original and post-move neighborhood SES levels affected fifth-graders' self-regulation. After controlling for baseline self-regulation measured during preschool, children who had moved showed poorer performance on computerized executive function tasks and lower teacher-rated self-regulation skills in

fifth grade, particularly those who moved to lower-SES neighborhoods [?, ?]. Additionally, a longitudinal study of preschoolers (n=306) found that low income predicted poorer executive control [?, ?].

Experimental research further establishes causal relationships between low SES and self-regulation. Field experiments demonstrate that SES significantly affects executive function. Mani et al. [?, ?] published findings in *Science* showing that Indian sugarcane farmers' performance on the Stroop task (an indicator of executive function) differed significantly between pre-harvest (impoverished) and post-harvest (relatively affluent) states. Since these farmers rely on annual harvest income, they experience relative poverty before harvest and relative affluence afterward. Randomly selected farmers completed Stroop tasks before and after harvest, revealing longer reaction times and higher error rates in the pre-harvest impoverished state, indicating that poverty significantly impairs executive function. Shah et al. [?, ?] replicated these findings experimentally by assigning different budget amounts to create "poor" and "rich" participants in a game. "Poor" participants received fewer default game rounds than "rich" participants but could borrow from future rounds. When borrowing was permitted, "poor" participants borrowed extensively from future rounds and showed poorer game performance. In another study, participants assigned smaller budgets made a series of purchasing decisions, after which those with smaller budgets performed worse on subsequent handgrip and Stroop tasks measuring behavioral control [?, ?].

3. Neural Mechanisms of Low SES Effects on Self-Regulation

Low SES shapes self-regulation development by altering neural systems that support self-regulation and physiological stress-related processes [?, ?]. Research shows that children from materially deprived families have smaller gray matter volumes in frontal and temporal cortices and the hippocampus compared to children from affluent families [?, ?]. Prefrontal cortex atrophy leads to reduced executive function, while hippocampal reduction affects emotional regulation [?, ?]. Furthermore, compared to high-SES individuals, low-SES individuals show abundant glucocorticoid receptors in the amygdala, hippocampus, and prefrontal cortex. Excessive glucocorticoid exposure may affect neuroplasticity, altering the size and neuronal structure of these regions and thereby influencing subsequent cognitive, emotional, and behavioral regulation [?, ?]. The following sections elaborate on the neural mechanisms through which SES influences the three components of self-regulation.

3.1 Neural Mechanisms of Low SES Effects on Cognitive Regulation

Current research indicates that two relatively independent brain networks operate in the relationship between SES and cognitive regulation [?, ?]. First, the frontoparietal network initiates attentional control as relevant cues begin,

change, or cease in the environment, integrating feedback on an event-by-event basis. The integrity of this network is crucial for rapid adaptive control and involves the dorsolateral prefrontal cortex (dlPFC), precuneus, and portions of the inferior parietal lobule [**?**, **?**]. Second, the cingulo-opercular network provides "stable set control" and is considered critical for maintaining task-relevant goals, including the dorsal anterior cingulate cortex (dACC), anterior insula/operculum (AI), and thalamus. Currently, dlPFC in the frontoparietal network and dACC in the cingulo-opercular network are the most extensively documented neural mechanisms through which SES influences cognitive regulation.

In the frontoparietal network, low SES affects dlPFC structure and function, thereby influencing cognitive regulation. Low SES is associated with impaired prefrontal cortical maturation, and the protracted development of this region makes it particularly vulnerable to chronic stress environments. Chronic activation of the HPA axis affects prefrontal tissue volume and function through glucocorticoid receptors. Structurally, lower SES is associated with smaller surface area in executive function-related brain regions and smaller prefrontal volume [**?**, **?**, **?**]. Neurophysiological research using executive function as an indicator of cognitive regulation has found that poorer executive function in low-SES individuals is closely related to smaller dlPFC volume [**?**, **?**], a pattern replicated across multiple studies [**?**, **?**, **?**]. Some research directly demonstrates that dlPFC volume mediates the relationship between low SES and cognitive regulation (executive function and working memory) [**?**, **?**, **?**]. The consequences of low SES leading to smaller dlPFC volume and subsequent cognitive regulation failure include increased externalizing behaviors and alcohol use during adolescence [**?**, **?**] and higher rates of ADHD or conduct disorder [**?**, **?**]. Functionally, low SES reduces dlPFC activation during cognitive regulation tasks, potentially reflecting impaired executive function processes involved in initiating, stopping, and changing behavior [**?**, **?**]. For example, fMRI studies examining neural mechanisms during scarcity states found that compared to affluent states, individuals in scarcity states showed significantly reduced dlPFC activation during working memory and task-switching tasks [**?**, **?**]. Additionally, multimodal neuroimaging revealed that low-SES youth aged 6–19 showed poorer working memory performance, lower functional dlPFC activation during tasks, and reduced fractional anisotropy (FA) in white matter fiber bundles connecting dlPFC and parietal regions. Lower FA values indicate compromised structural integrity and connectivity efficiency between dlPFC and parietal areas [**?**, **?**]. These findings demonstrate that low SES affects cognitive regulation by altering dlPFC activation levels and connectivity with other brain regions.

In the cingulo-opercular network, low SES also influences cognitive regulation by affecting dACC structure and function. In a sample of 283 children and adolescents, low SES was associated with reduced dACC thickness [**?**, **?**]. A cross-sectional study of 11,875 9- and 10-year-olds found that prefrontal volume, including dACC, mediated the relationship between low SES and cognitive regulation (measured by Flanker task performance) [**?**, **?**]. A 17-year longitu-

dinal study of children aged 3–5 years revealed that the developmental slope of prefrontal volume, including dACC, mediated the relationship between low SES and cognitive regulation (measured by Flanker and working memory tasks) [?, ?]. These findings indicate that low SES affects cognitive regulation through dACC structural changes. Functionally, early ERP studies of selective attention found that children from high-SES families showed greater brain activity near dACC to target stimuli and reduced activity to distractors, whereas low-SES children showed equivalent activity levels to both stimuli types, suggesting inhibitory control deficits [?, ?]. Subsequent fMRI research clarified that low SES is associated with reduced dACC activation and impaired inhibitory control [?, ?, ?, ?]. For example, an fMRI study of spontaneous low-frequency amplitude alterations in 655 children aged 6–14 found that lower family SES predicted lower dACC activation [?, ?]. Another resting-state fMRI (rsfMRI) longitudinal study of 167 preschoolers found that low-SES adolescents showed declining dACC activation from ages 13–19 [?, ?]. Additionally, lower SES is associated with reduced connectivity between dACC and other regions, such as the right amygdala and right hippocampus [?, ?]. However, developmental findings are not entirely consistent. A two-year longitudinal study of low-SES adolescents (ages 11–13) found that low-SES girls showed increased dACC activation over time and poor performance on inhibitory control tasks (Go/NoGo) [?, ?]. This difficulty may stem from significantly reduced dACC-dlPFC connectivity, requiring compensatory dACC activation during cognitive regulation tasks [?, ?, ?]. This suggests adolescence represents a vulnerable critical period for low-SES girls, with unique neurodevelopmental impacts. Despite varying dACC activation patterns, the overall evidence indicates that low SES affects cognitive regulation by altering the structure and function of dlPFC in the frontoparietal network and dACC in the cingulo-opercular network.

3.2 Neural Mechanisms of Low SES Effects on Emotional Regulation

Substantial evidence identifies the amygdala, ventromedial prefrontal cortex (vmPFC), and hippocampus as three key brain regions in the pathway from low SES to emotional regulation [?, ?, ?, ?, ?].

The amygdala, located in the anterior temporal lobe, serves as an information processing hub involved in physiological and behavioral responses to environmental and social challenges [?, ?, ?]. Low SES affects amygdala structure and function, thereby influencing emotional regulation. Low SES significantly negatively predicts amygdala volume, with low-SES children and adolescents showing smaller amygdala volumes [?, ?, ?]. Functionally, low SES affects amygdala activation during emotional regulation tasks. Individuals with low childhood SES show greater amygdala activation to fearful emotional cues in adulthood, whereas those with high childhood SES show greater activation to happy cues [?, ?]. Additionally, low neighborhood income correlates with heightened amygdala responses to negative emotional faces, suggesting that low-SES individuals may develop enhanced sensitivity and vigilance to threat cues due to

exposure to adverse social environments during childhood, resulting in elevated amygdala activation [?, ?, ?]. Smaller amygdala volume combined with heightened functional reactivity contributes to aggressive behavior, as hypervigilance and increased amygdala activity may lead to greater negative affect and hostile attributions, promoting aggressive responses [?, ?, ?].

The ventromedial prefrontal cortex (vmPFC) is preferentially involved in monitoring ongoing emotional states, semantically encoding stimuli, and implementing regulation strategies according to context [?, ?]. Low SES affects vmPFC structure and function, thereby influencing emotional regulation. Research shows that low SES induces structural changes in vmPFC. For example, income positively correlates with vmPFC volume [?, ?], and individuals with low childhood SES show reduced orbitofrontal cortex (OFC) volume, which is part of vmPFC [?, ?]. Relatedly, exposure to low SES during middle childhood reduces FA values in white matter tracts connecting vmPFC and amygdala, indicating compromised structural integrity of this pathway [?, ?]. Functionally, childhood low SES alters default mode network (DMN) connectivity—a brain network including vmPFC and other PFC regions that interact during rest. After controlling for current income, race, perceived social status, and depression/anxiety symptoms, low SES at age 9 predicted reduced DMN connectivity in adulthood [?, ?]. This finding has been replicated in infant samples, where low family SES reduced early DMN connectivity [?, ?]. Furthermore, childhood low SES correlates with lower resting-state coupling between amygdala and vmPFC at age 15 [?, ?]. Low SES is also associated with reduced dACC-vmPFC and dlPFC-vmPFC connectivity during positive feedback in reward tasks [?, ?].

Moreover, low SES causes structural and functional alterations in the hippocampus, likely mediated by the hypothalamic-pituitary-adrenal (HPA) axis and cortisol [?, ?]. Sustained HPA axis activation may lead to dendritic remodeling and neuronal death in the hippocampus and other brain regions [?, ?]. Hanson et al. [?, ?] found that among 317 individuals aged 4–18, lower SES predicted smaller hippocampal volume. This difference has been replicated across numerous samples and appears as early as 5 weeks of age [?, ?]. Using longitudinal MRI across ages 4–22, Hair et al. [?, ?] found that low-SES adolescents had hippocampal volumes 3–4 percentage points below developmental norms, while low-SES children showed volumes 8–10 percentage points below norms. Low SES also correlates with functional hippocampal deviations across all developmental stages. One longitudinal study found that lower income-to-needs ratio during preschool predicted reduced resting-state functional connectivity between hippocampus and amygdala during school age. This relationship between preschool low income-to-needs ratio and later negative affect and depression severity was mediated by left hippocampus-right superior frontal cortex resting-state connectivity [?, ?]. Task-based fMRI studies show that adults with lower income-to-needs ratio at age 9 exhibit reduced overall hippocampal activation during emotional regulation tasks [?, ?]. These results demonstrate that low SES affects neural circuits underlying emotional regulation, revealing specific brain mechanisms through which SES influences self-regulation [?, ?].

3.3 Neural Mechanisms of Low SES Effects on Behavioral Regulation

Current research suggests that the ventral striatum (VS) may serve as the physiological basis for low SES effects on behavioral regulation. Structurally, low SES causes tissue abnormalities in broader brain circuits involving VS. A cross-sectional study of 1,082 individuals aged 3–21 found that lower family income correlated with reduced FA values in the right superior corticostriatal tract, a white matter bundle connecting portions of VS with PFC subregions [**?**, **?**]. These patterns align with recent findings showing that low SES correlates with reduced FA in white matter bundles containing VS among 6–19-year-olds, suggesting that low-SES individuals process reward information less efficiently, which may impair adaptive behavioral guidance [**?**, **?**]. Functionally, VS is critical for reward motivation, supporting reward sensitivity and learning by showing activity across reward dimensions (magnitude, probability, effort, and delay). Numerous studies find that heightened VS activation in low-SES individuals leads to impulsive, addictive, and disruptive behaviors [**?**, **?**, **?**, **?**, **?**]. Additionally, low SES creates abnormal functional connectivity between VS and other brain regions. For example, Romens et al. [**?**, **?**] found that among girls aged 5–16, total years of family public assistance correlated with increased mPFC activation during reward anticipation. Relatedly, after controlling for interpersonal problems and internalizing symptoms, lower SES correlated with weaker resting-state dlPFC-VS connectivity, which in turn predicted more impulsive decision-making among low-SES individuals [**?**, **?**, **?**].

Although behavioral, cognitive, and emotional regulation represent distinct domains of self-regulation, they are interrelated [**?**, **?**, **?**]. Behavioral regulation emerges from the integrated balance of cognitive and emotional regulation, and the neural mechanisms through which low SES affects behavioral regulation likely involve brain regions associated with cognitive and emotional regulation. For instance, Oshri et al. [**?**, **?**] investigated cognitive and affective mechanisms linking chronic socioeconomic adversity to impulsive behavior, finding that reduced activation in brain regions engaged during working memory tasks (cognitive control network) mediated the relationship between low SES and impulsive behavior, but only among adults with high emotional reactivity. High emotional reactivity suggests unsuccessful emotion regulation and impaired executive function, conditions under which low SES promotes impulsive behavior by reducing cognitive control network activation. This evidence indicates that low SES can affect behavioral regulation through its impact on brain regions involved in cognitive and emotional regulation.

In summary, researchers have proposed a self-regulation neural circuit in which the prefrontal cortex, amygdala, and ventral striatum form a neural network responsible for self-regulatory activity, with the hippocampus and ventral tegmental area providing additional modulatory influences [**?**, **?**].

4. Summary and Outlook

Research on the relationship between low SES and self-regulation has made substantial progress over the past two decades, with investigators from different disciplines beginning to answer "where" these effects occur—in the brain. These studies reveal that low SES alters various neurobiological centers related to attention, emotion, reward, and memory, specifically changing the structure and function of the dorsolateral prefrontal cortex (dlPFC), dorsal anterior cingulate cortex (dACC), ventromedial prefrontal cortex (vmPFC), amygdala, hippocampus, and ventral striatum (VS). These neurobiological variations affect all components of self-regulation (cognitive, emotional, and behavioral regulation). To translate these findings into practical and policy value, future research should pursue three key directions.

First, each step in the potential causal chain from "low SES → brain structure and function → self-regulation → adverse outcomes" requires deeper investigation. Current research primarily compares brain structural, functional, or connectivity differences between low- and high-SES individuals and discusses potential consequences of these differences. However, specific response patterns in low-SES individuals (brain structural and functional differences) do not uniformly lead to adverse outcomes. Some evidence shows that although low- and high-SES individuals exhibit different neural responses to identical stimuli, their behavioral performance (task accuracy and reaction time) does not differ [**?**, **?**, **?**, **?**]. Moreover, some studies find that disadvantaged children actually score higher on inhibitory control and problem-solving than non-disadvantaged peers [**?**, **?**]. Additionally, low-SES individuals are not necessarily more impulsive or shortsighted; when threats are removed, they demonstrate greater patience [**?**, **?**, **?**, **?**, **?**]. Furthermore, brain structural and functional differences resulting from low SES may represent adaptive responses. For example, right lateral prefrontal cortex (RLPFC) thickness positively correlates with reasoning ability in low-SES children and adolescents, but this relationship is absent in high-SES youth [**?**, **?**]. In low-SES young adults, reduced resting-state functional connectivity between basolateral amygdala and vmPFC correlates with lower anxiety, whereas the opposite pattern appears in high-SES youth [**?**, **?**]. Taken together, these findings suggest that some so-called "deficits" in low-SES individuals may simply reflect differences arising from environmental adaptation, representing human diversity rather than a dichotomy of "defective" versus "normal" [**?**, **?**]. Moreover, the pathways linking low SES to self-regulation and its neural mechanisms are complex. Prenatal factors, parent-child interactions, and cognitive stimulation in the home environment [**?**, **?**], as well as stress [**?**, **?**, **?**, **?**], mediate these relationships, while parental attachment moderates them, with higher attachment levels buffering the effects of early low SES on vmPFC volume reduction and behavioral regulation [**?**, **?**]. However, existing studies have examined only single factors in isolation. Future research must clarify each pathway and construct comprehensive mechanistic models.

Second, neuroscience must be more deeply integrated with developmental psy-

chology to reveal stage-specific mechanisms of low SES effects on self-regulation. Self-regulation likely 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) [**?**, **?**]. Experiencing low SES at specific developmental stages may uniquely affect different aspects of self-regulation. Evidence shows that low-SES children perform worse than peers on selective attention tasks at age 6, but this difference often disappears by adolescence [**?**, **?**]. Additionally, social class may influence the brain through continuous changes or step functions at specific points. Neurobiological research has compared extreme groups ("poor" vs. "non-poor") and examined continuous SES relationships with self-regulation, but it remains unclear which self-regulation circuits show step-function relationships with low SES and which show continuous associations. Future research must strengthen connections between developmental psychology and the neurobiology of low SES to address these questions. Examining how changes in critical neural circuits dynamically interact across timeframes, levels of analysis, and environmental contexts is essential for revealing core mechanisms in the psychosocial and neurobiological processes linking low SES to self-regulation.

Third, promoting self-regulation in low-SES individuals is key to enhancing human capital and achieving common prosperity. Future research should develop systematic, sustained, and effective interventions based on mechanistic insights. Over the past two decades, researchers have developed interventions leveraging recent cognitive neuroscience concepts and methods to improve self-regulation in low-SES children. At the laboratory level, computer games targeting specific cognitive control processes (e.g., customized Go/NoGo tasks) have been developed to enhance cognitive regulation [**?**, **?**, **?**, **?**]. School-based interventions combining cognitive training (Tower of London for planning, chunking tasks for working memory, Stroop tasks for inhibitory control) with nutritional supplementation (iron and folic acid) significantly improved attention, working memory, and planning in low-SES children aged 4–6 [**?**, **?**]. Additionally, positive academic support from teachers significantly enhanced executive function in low-SES children and adolescents [**?**, **?**]. Family-based interventions such as the Family Check-Up, which involves parents meeting with professional consultants to address concerns and family issues critical for child development, effectively improved children' s self-regulation [**?**, **?**]. Community-level interventions that enhance community trust and buffer financial needs reduced impulsive behavior in low-SES individuals [**?**, **?**]. However, recent perspectives argue that interventions should abandon deficit-based models that aim to repair damage and instead adopt an adaptation-based approach that identifies and leverages adaptive and advantageous responses in low-SES individuals [**?**, **?**]. For example, low-SES individuals show enhanced attentional shifting under economic uncertainty [**?**, **?**], and low-SES children demonstrate enhanced problem-solving in reward-oriented contexts [**?**, **?**]. Future intervention research should focus on these "hidden talents"of low-SES individuals, designing classroom environments, teaching strategies, and job training that better accommodate their needs and

potential, thereby maximizing their capabilities in policy and practice.

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