Review

Biological Psychiatry: GOS

Socioeconomic Disparities in Hypothalamic-Pituitary-Adrenal Axis Regulation and Prefrontal Cortical Structure

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ABSTRACT

Socioeconomic disadvantage during childhood predicts an increased risk for mental health problems across the life span. Socioeconomic disadvantage shapes multiple aspects of children's proximal environments and increases exposure to chronic stressors. Drawing from multiple literatures, we propose that childhood socioeconomic disadvantage may lead to adaptive changes in the regulation of stress response systems including the hypothalamic-pituitary-adrenal (HPA) axis. These changes, in turn, affect the development of prefrontal cortical (PFC) circuitry responsible for top-down control over cognitive and emotional processes. Translational findings indicate that chronic stress reduces dendritic complexity and spine density in the medial PFC and anterior cingulate cortex, in part through altered HPA axis regulation. Socioeconomic disadvantage has frequently been associated with reduced gray matter in the dorsolateral and ventrolateral PFC and anterior cingulate cortex and lower fractional anisotropy in the superior longitudinal fasciculus, cingulum bundle, and uncinate fasciculus during middle childhood and adolescence. Evidence of socioeconomic disparities in hair cortisol concentrations in children has accumulated, although null findings have been reported. Coupled with links between cortisol levels and reduced gray matter in the PFC and anterior cingulate cortex, these results support mechanistic roles for the HPA axis and these PFC circuits. Future longitudinal studies should simultaneously consider multiple dimensions of proximal factors, including cognitive stimulation, while focusing on epigenetic processes and genetic moderators to elucidate how socioeconomic context may influence the HPA axis and PFC circuitry involved in cognitive and emotional control. These findings, which point to modifiable factors, can be harnessed to inform policy and more effective prevention strategies.

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Socioeconomic disadvantage during childhood is prevalent and increases risk for mental health problems across the life span (1). Socioeconomic disadvantage refers to conditions of low economic resources and social and human capital. Common socioeconomic measures include family income and parental educational attainment, which tend to be highly correlated (2–4). Socioeconomic context affects mental health on a gradient, with the risk of psychopathology especially high among those with very low economic resources (e.g., poverty) and declining gradually as economic resources increase (5). Socioeconomic disadvantage is a distal factor that shapes multiple aspects of children's proximal environments (6) and often increases exposure to chronic stressors, such as neighborhood violence, family conflict, crowding/noise, and household unpredictability (3,4,7–10). In addition to affecting children directly, financial strain and these chronic stressors can affect children indirectly by increasing parental stress, which can lead to harsher, less responsive, and more unpredictable parenting (3,8,9). Frequently documented socioeconomic disparities in cognitive and emotional control among children and adolescents (8,10–12) have prompted research into the neurobiological mechanisms that underlie these associations.

Socioeconomic disadvantage has been repeatedly associated with altered functioning of the hypothalamic-pituitary-adrenal (HPA) axis stress response system (13,14) and the structure of prefrontal cortical (PFC) circuitry crucial to cognitive and emotional control in children and adolescents (5). These effects are theorized to occur as adaptations to the environment that may be beneficial in the short term but may impose higher risks for psychopathology in the long term (15,16). Drawing from multiple literatures, we propose that socioeconomic context during childhood may influence HPA axis function and PFC circuitry responsible for cognitive and emotional control, partially explaining socioeconomic disparities in mental health. Translational and human research bearing on this model is reviewed.

THE HPA AXIS

Stressors or threats influence multiple physiological systems (e.g., autonomic nervous system, neuroendocrine system, immune system) including the HPA axis. Stressors are

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detected and appraised in corticolimbic circuits that signal to increase HPA axis activation. Information is integrated by the paraventricular nucleus of the hypothalamus to control corticotropin-releasing hormone secretion, which stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone, leading the adrenal cortex to synthesize and release glucocorticoids (cortisol in humans, corticosterone in rodents) (17). Once secreted, cortisol binds to mineralocorticoid and glucocorticoid receptors (GRs) in multiple brain regions including the medial PFC (mPFC), hippocampus, and amygdala, which have high densities of GRs (18). As cortisol levels rise and reach moderate-to-high concentrations, low-affinity GRs become occupied, prompting energy-mobilizing effects that facilitate behavioral responses to the stressor. Circulation of glucocorticoids is controlled through a negative feedback loop involving GRs in regions including the hypothalamus, hippocampus, and mPFC (19–22). Elevated glucocorticoids typically suppress HPA axis activation by occupying GRs in these brain regions (17,20,23). Glucocorticoids are not only produced in response to stressors but are also released across the day to ensure basal levels sufficient for healthy functioning. The release of basal glucocorticoids follows a diurnal rhythm, with higher levels in the morning and then decreasing production throughout the day, reaching minimum levels at night.

PFC CIRCUITRY SUPPORTING COGNITIVE AND EMOTIONAL CONTROL

The PFC has a protracted developmental course that extends through adolescence, making it malleable to environmental influences for a lengthy period. Frontoparietal and frontolimbic networks support the interrelated processes of cognitive and emotional control, respectively (24–26). PFC regions that support cognitive control include lateral PFC regions, which are heavily implicated in inhibitory control and working memory, and the dorsal anterior cingulate cortex (ACC), which has been associated with error monitoring (24,27,28). Both the dorsolateral PFC (dlPFC) and mPFC (including ventral ACC) have been strongly associated with emotion regulation (29). In diffusion-weighted imaging studies, frontoparietal circuitry includes the superior longitudinal fasciculus (SLF), which connects the lateral prefrontal and parietal cortices. Frontolimbic circuitry includes the uncinate fasciculus, which connects the ventromedial PFC and amygdala (25,30). The cingulum bundle connects the anterior cingulate, prefrontal, parietal, and temporal cortices and the hippocampus and plays roles in both cognitive and emotional control (24,25,31,32). Frontolimbic connections allow PFC-mediated top-down control over amygdala reactivity, leading to modified emotional responses (25,33,34). Research suggests that the dlPFC signals to mPFC regions, including the ventral ACC, which connects with the amygdala, supporting emotion regulation (25,35).

CURRENT REVIEW

In our integrative model (Figure 1), socioeconomic disadvantage, via increases in exposure to chronic stressors during childhood, is proposed to lead to adaptive changes in the regulation of stress response systems including the HPA axis, which alter the development of PFC circuitry responsible for top-down control over cognitive and emotional processes. Our model builds on many earlier theories, including the allostatic load and adaptive calibration models (15,36). After an overview of translational work, we review the associations among socioeconomic factors, HPA axis functioning, and PFC structure and structural connectivity with parietal and limbic regions primarily in children and adolescents. Then, we discuss the role of multiple proximal factors, developmental timing, and racial/ethnic discrimination; encourage research on epigenetic processes; and consider possibilities for how socioeconomic context may influence PFC structural development. Finally, we cover future directions and implications for practice and policy. This review is comprehensive but not exhaustive; exemplar citations are provided in the main text, with additional information in the Supplement.

ANIMAL MODELS OF CHRONIC STRESS

It is well established in translational research that chronic stress impacts PFC morphology. Some of this research has employed the limited bedding and nesting (early-life scarcity) and chronic variable stress paradigms (juvenile stress), which increase exposure to environmental unpredictability (37–39). In rodent models, chronic stress causes structural remodeling of PFC neurons (36,40–44), including decreased dendritic length and branching and spine density in pyramidal neurons in the mPFC (e.g., Brodmann area 25) (38,40,42,43,45), indicating a potential reduction in excitatory synapses (46). The effects of chronic stress during early life or the peripubertal period on PFC morphology persist over time and are associated with reduced attentional control and working memory (37,47–56).

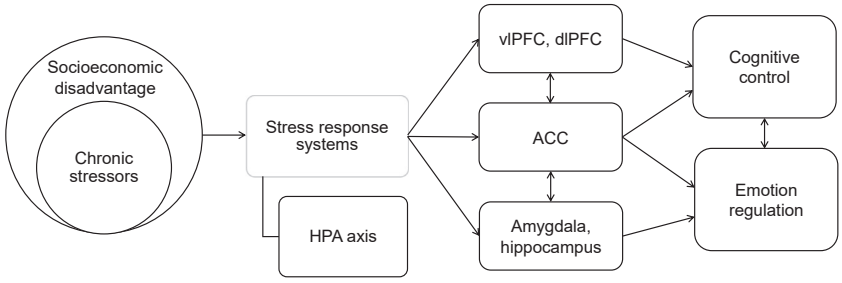

Figure 1. Integrative model highlighting the role of chronic stress and physiological stress mediators in the neurobiological mechanisms underlying socioeconomic disparities in cognitive and emotional control. Socioeconomic disadvantage leads to elevated exposure to chronic stressors, which may influence multiple stress response systems (e.g., autonomic nervous system, hypothalamic-pituitary-adrenal [HPA] axis, immune system) during childhood and adolescence, in turn impacting the development of prefrontal cortical (PFC) and frontolimbic circuitry underlying cognitive and emotional control. Although our review focuses on the HPA axis, multiple physiological systems are likely involved in these mechanisms. Socioeconomic disadvantage has been associated with altered HPA axis function and reduced PFC gray matter during childhood and adolescence. Beyond chronic stress, other proximal pathways, including differences in cognitive stimulation, are involved; these are not pictured here. ACC, anterior cingulate cortex; dlPFC, dorsolateral PFC; vlPFC, ventrolateral PFC.

These effects are due in part to altered HPA axis functioning. Chronic stress during early life or childhood/adolescence has enduring effects on HPA axis functioning, often causing elevated corticosterone secretion (37,57) due in part to impaired glucocorticoid feedback control of the HPA axis. In rodents, early-life stress decreases GR expression (e.g., through increased GR gene methylation) (58) in regions including the hippocampus and mPFC that regulate feedback (18,59). Chronic corticosteroid administration is sufficient to cause reductions in dendritic complexity, including loss of dendritic spines, in the mPFC, thus weakening synaptic efficacy (38,60–63). Early-life stress also increases corticotropin-releasing hormone expression in the hypothalamus, amygdala, and hippocampus. In conjunction with glucocorticoid effects, high corticotropin-releasing hormone levels modulate synaptic structure and may disrupt dendritic arborization and pruning in limbic regions (37).

Chronic stress can also cause blunted HPA axis activity. Moderators such as sex and the timing and duration of exposure may influence whether chronic stress is found to be associated with heightened or blunted corticosterone. Prolonged chronic stress exposure has been associated with blunted HPA axis activity, including reduced corticosterone production (39,64,65), which may be related to elevated GR expression in the mPFC enhancing negative feedback (39).

Nonhuman primate models, such as those using variable foraging demand paradigms, have shown similar effects and suggested that chronic or uncontrollable stress weakens synaptic connectivity in the dlPFC (e.g., Brodmann area 46) (35,66). Thus, animal models, which through experimental designs have the benefit of allowing causal inferences to be drawn from study findings, have shown effects of chronic stress on PFC morphology and identified altered HPA axis function as a likely mediator of those effects.

SOCIOECONOMIC FACTORS AND PFC STRUCTURE

Consistent with these findings (36,48), in human magnetic resonance imaging research, socioeconomic disadvantage at both the household and neighborhood levels has been consistently associated with reduced gray matter in PFC regions, as measured by cortical volume, thickness, or surface area during early childhood (67–70), middle childhood (14,67,69–80), and adolescence (26,67,69,70,72,78–82) (Table 1). These associations have been found more frequently for cortical surface area than for cortical thickness (26,70,74). In large-scale studies using both whole-brain and region-of-interest approaches, these associations have been found in lateral PFC and mPFC regions (70,74–79). The most consistent findings have been for the dlPFC (rostral middle frontal gyrus), ventrolateral PFC (inferior frontal gyrus), and ACC. Although significant findings have been obtained during early childhood (68), including in neonates (83), as well as prenatally (84), more work is needed on these developmental periods.

SOCIOECONOMIC FACTORS AND FRONTOLIMBIC AND FRONTOPARIETAL STRUCTURAL CONNECTIVITY

Socioeconomic disadvantage in childhood has also been linked with reduced white matter organization in frontolimbic and frontoparietal tracts. In diffusion-weighted imaging studies, socioeconomic disadvantage has been repeatedly associated with lower fractional anisotropy (FA) in the uncinate fasciculus, cingulum bundle, and SLF during middle childhood (85–90) and adolescence (86,88,89,91–93) (Table 2). Some studies have yielded null findings for FA in these tracts (71,80) or inverse associations (94). Socioeconomic disadvantage has also been associated with indices beyond FA, such as reduced myelin growth during adolescence (95) and lower network efficiency in prefrontal and limbic regions (96). Lower FA in the SLF and cingulum bundle have been associated with reduced cognitive control, including working memory (31,88). In sum, these results provide insights into the neural networks that may underlie socioeconomic disparities in cognitive and emotional control. Socioeconomic disadvantage has often been associated with reduced gray matter and lower FA in PFC circuitry crucial to cognitive and emotional control in children and adolescents.

SOCIOECONOMIC FACTORS AND HPA AXIS FUNCTIONING

Socioeconomic disadvantage has been studied frequently in relation to salivary, urinary, and hair cortisol levels in children and adolescents. Given that socioeconomic disadvantage is associated with chronic stress, certain methods of measuring cortisol may be better matched to these investigations than others, such as measures that reflect long-term changes in cortisol secretion (e.g., hair, basal salivary, urinary cortisol) rather than acute cortisol reactivity (97). Although socioeconomic factors have frequently been associated with cortisol levels, a clear pattern of hyper- or hypocortisolism has not been established.

Basal salivary cortisol has been measured in multiple ways including basal levels at particular times of the day and area under the curve (AUC) measures of total cortisol output over the course of the day. Neighborhood and family socioeconomic disadvantage have been linked with elevated basal cortisol in some studies (98–104), lower basal cortisol in other studies (100,105,106), and no differences in basal cortisol in other studies (107) of children and adolescents (Table S1). Similarly, socioeconomic disadvantage has been associated with greater AUC in some studies (7,108) but with lower AUC in other studies (109,110) and no differences in AUC in others (103,111,112) (Table S2). Urinary cortisol provides an index of cumulative levels over the course of a day or night. In a longitudinal study, exposure to poverty during childhood was significantly associated with elevated urinary cortisol overnight in children and adolescents (113).

Hair cortisol concentrations provide an index of cumulative levels over the course of months. Measuring hair cortisol may be particularly well-suited to investigation of the effects of socioeconomic disadvantage on HPA axis function. Across multiple studies, socioeconomic disadvantage has been associated with higher hair cortisol concentrations in early childhood (103,111,114–116), middle childhood (14,117), and adolescence (118) (Table S3). However, other studies have failed to find significant associations (119–125).

Collectively, these findings suggest that socioeconomic disadvantage may lead to altered HPA axis activity in children

Table 1. Associations Between Socioeconomic Factors and Prefrontal Cortex Gray Matter Structure

Authors	*N*	Age (Range; Mean)	Study Design	SES Measure	Cortical Gray Matter Measure	ROI and/or Whole-Brain Analysis	Associations Between SES and PFC Structure	Direction of Association
Barch *et al.* (162)	167	AD (13–19 y; mean = 15.83 y)	L	Family income-to-needs ratio	CV	ROI	Family income-to-needs ratio was not significantly associated with dorsal ACC or rostral MFG volume.	NS
Brain Development Cooperative Group (156)	325	EC, MC, AD (4–18 y; mean = 11 y)	CS	Family income, parental education	CV	ROI	Socioeconomic factors were not significantly associated with frontal lobe volume.	NS
Dufford *et al.* (12)	34	MC (8–10 y; mean = 8.76 y)	CS	Family income-to-needs ratio	CV	ROI	Family income-to-needs ratio was not significantly associated with IFG volume.	NS
Gur *et al.* (80)	1395	MC, AD (8–21 y)	CS	Neighborhood SES	CV, gray-matter density	ROI	Lower SES was associated with smaller volume and gray matter density in the frontal lobe.	+
Hackman *et al.* (77)	8598	MC (9–10 y; mean = 9.9 y)	CS	Neighborhood disadvantage, family income, parental education	SA, CT	Whole-brain, ROI	Greater neighborhood disadvantage was associated with smaller SA in the rostral MFG and IFG (pars orbitalis).	+
Hair *et al.* (67)	389	EC, MC, AD (4–20 y; mean = 11 y)	L	Family income	CV	ROI	Children from low-income households had reduced volume in the frontal lobe.	+
Hanson *et al.* (68)	77	EC (0–5 y; mean at first scan = 13.5 mo)	L	Family income	CV	Whole-brain	Children from lower-income families had smaller volume in the frontal lobe.	+
Jednoróg *et al.* (71)	23	MC (8–10 y; mean = 9.58 y)	CS	Hollingshead 2-factor index (maternal education and occupational status)	CV, CT, SA	Whole-brain	Lower SES was associated with reduced volume in the left S/MFG.	+
Judd *et al.* (26)	551	AD (14–19 y; mean = 14.44 y at wave 1)	L	Combined sum of income-related variables[a], parental education, and neighborhood quality	SA, CT	Whole-brain	SES was not associated with regional SA while controlling for total SA. When not controlling for total SA, SES was associated with SA in medial PFC regions. SES was not associated with CT. SES was associated with change between 14 and 19 years of age in SA in the left caudal superior frontal sulcus.	NS/+
King *et al.* (72)	147 for CS sample, 109 for L sample	MC, AD (9–13 y at time 1; mean = 11 y)	L	Family income-to-needs ratio	CV	Whole-brain	Interactions between family income-to-needs ratio and sex were found. Cross-sectionally, lower family income-to-needs ratio was associated with reduced volume in the right SFG, right MFG, and left IFG in boys.	+
Lawson *et al.* (69)	283	EC, MC, AD (4–18 y; mean = 11.47 y)	CS	Family income, parental education	CT	ROI	Lower parental education was associated with reduced CT in the left SFG and right ACC.	+
Lu *et al.* (84)	144	Prenatal (24–39 gestational wk)	CS	Maternal and paternal SES composites (maternal and paternal education and occupation)	CV	ROI	Lower maternal and paternal SES were associated with decreased volume in the frontal lobe in fetuses.	+
Mackey *et al.* (81)	58	AD (mean = 14 y)	CS	Free or reduced-price lunch status; subset with family income data	SA, CT	Whole-brain	Lower SES was associated with reduced CT in the right rostral MFG and right IFG.	+

Table 1. Continued

Authors	*N*	Age (Range; Mean)	Study Design	SES Measure	Cortical Gray Matter Measure	ROI and/or Whole-Brain Analysis	Associations Between SES and PFC Structure	Direction of Association
McDermott *et al.* (78)	623	MC, AD (5–25 y at first scan; mean = 12.0 y)	L	Hollingshead 2-factor index (parental education and occupation)	CV, SA, CT	Whole-brain	Lower SES was associated with reduced SA in the MFG, SFG, OFC, and ACC.	+
Merz *et al.* (14)	51	MC (5–9 y; mean = 7.03 y)	CS	Family income-to-needs ratio, parental education	CT, SA	ROI	Lower parental education was associated with reduced rostral and caudal ACC SA and greater rostral and caudal ACC CT.	+ and −
Noble *et al.* (82)	60	MC, AD (5–17 y; mean = 11.4 y)	CS	Family income-to-needs ratio, parental education	CV	ROI	No main effects of SES on left IFG or ACC volume. A parental education-by-age interaction was observed for left IFG volume, such that lower SES was associated with lower left IFG volume in adolescence.	+
Noble *et al.* (70)	1099	EC, MC, AD (3–20 y; mean = 11.9 y)	CS	Family income, parental education	SA, CT	Whole-brain	Lower family income and parental education were associated with reduced SA in the IFG, rostral MFG, SFG, medial OFC, and ACC.	+
Romeo *et al.* (73)	65	MC (6–9 y; mean = 7.75 y)	L	SES composite (maternal education, occupational prestige)	CT, CV	Whole-brain	Lower SES was associated with lower CT and volume in the left IFG (pars opercularis).	+
Rosen *et al.* (88)	49	MC, AD (6–19 y; mean = 13.7 y)	CS	Family income-to-needs ratio	CT	ROI	Family income-to-needs ratio was not significantly associated with CT in the MFG.	NS
Sanders *et al.* (79)	789	MC, AD (5–21 y; mean = 13.9 y)	CS	Family income-to-needs ratio, maternal education	CT	Networks of interest	Lower maternal education was associated with reduced CT in the CON. Significant age-by-maternal education interaction for CT in the DAN such that higher maternal education was associated with steeper age-related decreases in CT.	+
Spann *et al.* (83)	37	EC (1–6 wk; mean postmenstrual age = 42 wk)	CS	Hollingshead 2-factor index (parent education and occupation)	CV	Whole-brain	Lower SES was associated with greater volume in the right MFG and left IFG and ACC.	−
Taylor *et al.* (75)	11,875	MC (9–10 y)	CS	Neighborhood poverty, household income	CV	ROI	Lower household income and higher neighborhood poverty were associated with reduced volume in the MFG and SFG.	+
Tomasi and Volkow (74)	7784	MC (9–10 y; mean = 9.8 y)	CS	Family income, neighborhood deprivation, parental education	CT, SA, CV	ROI	Lower family income was associated with reduced volume and SA in the IFG, MFG, SFG, OFC, and ACC.	+
Vargas *et al.* (76)	10,205	MC (9–11 y; mean = 9.9 y)	CS	Neighborhood deprivation	CT, SA	ROI	Higher neighborhood deprivation was associated with greater overall PFC SA, right SFG SA, and right rostral MFG SA; lower SA in the bilateral frontal pole; and lower CT in the right rostral MFG, right SFG, right medial OFC, and left lateral OFC.	− and +

Table 1. Continued

Authors	*N*	Age (Range; Mean)	Study Design	SES Measure	Cortical Gray Matter Measure	ROI and/or Whole-Brain Analysis	Associations Between SES and PFC Structure	Direction of Association
Whittle *et al.* (153)	166	MC, AD (11–20 y; mean at time 1 = 12.79 y)	L	Parental income-to-needs ratio, occupation, and education level; neighborhood SES	CT	Whole-brain	No significant main effects or age-moderated or sex-moderated effects of parental education, occupation, income-to-needs ratio, or neighborhood disadvantage on CT in PFC regions.	NS

Where more than one study was conducted on subsamples within the same larger sample, an attempt was made to include only one publication unless methods were different. Studies that only used global measures of brain structure (e.g., total gray matter volume) are not included here. + indicates significant positive association, and − indicates significant negative association.

ACC, anterior cingulate cortex; AD, adolescence; CON, cingulo-opercular network; CS, cross-sectional; CT, cortical thickness; CV, cortical volume; DAN, dorsal attention network; EC, early childhood; IFG, inferior frontal gyrus; L, longitudinal; MC, middle childhood; MFG, middle frontal gyrus; NS, not significant; OFC, orbitofrontal cortex; PFC, prefrontal cortex; ROI, region of interest; SA, cortical surface area; SES, socioeconomic status; SFG, superior frontal gyrus.

[a]Financial difficulties, financial crisis, family stress, unemployment.

and adolescents, although null findings have been reported, and a consistent pattern of hyper- or hypocortisolism has not been shown. Socioeconomic disadvantage has been associated with HPA axis activity via increased exposure to chronic stressors (106), which may explain some of the variability in findings. Associations between socioeconomic disadvantage and HPA axis activity may also depend on the duration of (or time since) exposure to socioeconomic disadvantage. Childhood socioeconomic disadvantage may lead to increased HPA axis activity initially that is followed by eventual downregulation of HPA axis activity over time (13,15,126). Initial upregulation of the HPA axis may represent an adaptation to chronic stressors associated with socioeconomic disadvantage. Heightened HPA axis reactivity may facilitate early detection of potential stressors and adaptive behavioral responses in the short term (15). This biological adaptation to stressful conditions during childhood may lead to an increased risk for mental health problems in the long term. Differences in findings across studies may also result from variation in methods (e.g., saliva samples collected only once in some studies) and the severity of exposure (e.g., percentage of the sample in poverty). Null results in some studies could be due to range restriction in socioeconomic context or small sample size.

HPA AXIS FUNCTIONING AND PFC STRUCTURE AND STRUCTURAL CONNECTIVITY

In human neuroimaging studies, cortisol levels have often been associated with PFC structure in children and adults (Figure 2). Similar to findings from animal models (36,60,62,63), some studies have linked higher cortisol levels with reduced PFC volume and thickness, with most studies having been conducted in adults (Table S4). In adults, higher salivary, hair, and serum cortisol levels have been significantly associated with reduced lateral and medial PFC and ACC gray matter (127–132). This pattern of results has also been found in children and adolescents (133,134), but other studies have linked lower cortisol levels or reactivity to reduced PFC and ACC thickness in children (14,134). In addition, some studies have not detected links between cortisol and PFC morphology (135,136).

In studies of white matter microstructure in adults (131) and children or adolescents (137,138), higher basal cortisol has been associated with reduced FA in the cingulum bundle, uncinate fasciculus, and SLF (Table S5). In another study, lower hair cortisol was associated with reduced FA in the cingulum bundle in children (94). In young adults, sex differences in the associations between cortisol reactivity and cingulum FA were observed in one study, with positive associations in men and negative associations in women (139). Other studies have not found significant associations (128).

In sum, human neuroimaging studies have revealed associations of HPA axis activity with PFC and ACC gray matter and FA in frontolimbic and frontoparietal tracts. Given that most studies have been cross-sectional and correlational, conclusions cannot be drawn about the directionality or causality of these associations. Based on findings from animal research (36), these associations may reflect the effects of elevated cortisol levels on PFC structure. They could also reflect reductions in the capacity of the PFC to adequately

Table 2. Associations Between Socioeconomic Factors and FA in Frontolimbic and Frontoparietal White Matter Tracts

Authors	*N*	Age (Range; Mean)	Study Design	SES Measure	Tract-of-Interest and/or Whole-Brain Analysis	Associations Between SES and FA	Direction of Association		
							UNC	CB	SLF
Bell *et al.* (91)	303	AD (SES measured at mean = 11.20 y, MRI at mean = 20.25 y)	L	Neighborhood disadvantage, family income	Tracts-of-interest	Greater neighborhood disadvantage was associated with reduced quantitative anisotropy in the CB and UNC. Lower family income was associated with lower quantitative anisotropy in the CB.	+	+	NA
Dufford and Kim (85)	27	MC (8–10 y; mean = 8.66 y)	CS	Family income-to-needs ratio, maternal education	Whole-brain	Lower family income was associated with lower FA in the left UNC, CB, and SLF.	+	+	+
Dufford *et al.* (92)	43	MC, AD (SES measured at 9, 13, 17, and 24 y; 20–27 y at scan)	L	Family income-to-needs ratio	Whole-brain	Lower childhood income-to-needs ratio was associated with reduced FA in the bilateral UNC, CB, and SLF.	+	+	+
Gullick *et al.* (86)	42	MC, AD (7–13 y; mean = 10.4 y)	CS	Parental education	Whole-brain	Lower parental education was associated with lower FA in the right anterior inferior fronto-occipital fasciculus and left SLF.	NS	NS	+
Gur *et al.* (80)	1395	MC, AD (8–21 y)	CS	Neighborhood SES	Tracts-of-interest	Neighborhood SES was not significantly associated with FA.	NS	NS	NS
Jednoróg *et al.* (71)	23	MC (8–10 y; mean = 9 y)	CS	Hollingshead 2-factor index (maternal education and occupational status)	Whole-brain	No significant correlations were found between SES and white matter properties.	NS	NS	NS
Li *et al.* (90)	8842	MC (9–11 y; mean = 9.9 y)	CS	Neighborhood disadvantage, household income, parental education	Tracts-of-interest	Higher neighborhood disadvantage and lower parental education were associated with lower FA in the SLF.	NS	NS	+
Ozernov-Palchik *et al.* (87)	125	MC (5–7 y; mean = 5.58 y)	CS	Maternal and paternal education	Tracts-of-interest	Maternal and paternal education were not significantly associated with FA in the SLF or CB. Lower parental education was correlated with lower FA in the UNC after controlling for gender.	+	NS	NS
Rosen *et al.* (88)	43	MC, AD (6–19 y; mean = 13.7 y)	CS	Family income-to-needs ratio	Tract-of-interest	Lower family income-to-needs ratio was associated with lower FA in the right and left SLF.	NA	NA	+
Simon *et al.* (94)	51	MC (5–9 y; mean = 7 y)	CS	Family income-to-needs ratio; parental education	Tract-of-interest	Lower family income-to-needs ratio and parental education were both associated with higher FA in the dorsal cingulum.	NA	−	NA
Ursache *et al.* (89)	1082	EC, MC, AD (3–21 y; mean = 12.21 y)	CS	Family income, parental education	Whole-brain, tract-of-interest	Lower family income was related to lower FA in the right parahippocampal cingulum.	NS	+	NS
Vanderauwera *et al.* (93)	35	AD (13–14 y; mean = 13.7 y)	CS	Parental education	Tracts-of-interest	Lower paternal education was associated with lower FA in the left UNC.	+	NA	NA

+ indicates significant positive association, and − indicates significant negative association.

AD, adolescence; CB, cingulum bundle; CS, cross-sectional; EC, early childhood; FA, fractional anisotropy (the degree of directionality of water diffusion); L, longitudinal; MC, middle childhood; MRI, magnetic resonance imaging; NA, not applicable because not measured; NS, not significant; SES, socioeconomic status; SLF, superior longitudinal fasciculus; UNC, uncinate fasciculus.

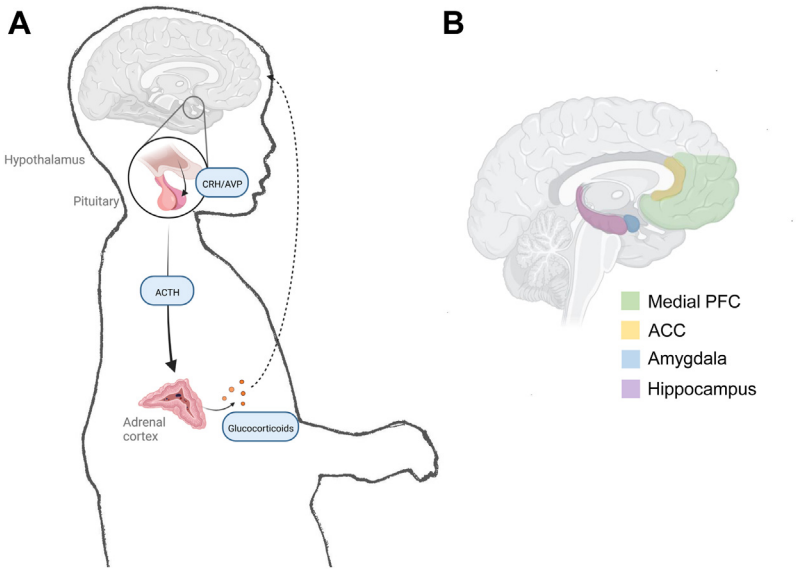

Figure 2. **(A)** Stress-induced increases in hypothalamic-pituitary-adrenal axis activation culminate in the release of glucocorticoids, which bind to receptors in multiple regions of the developing brain, including the medial prefrontal cortex (PFC), anterior cingulate cortex (ACC), amygdala, and hippocampus. **(B)** Similar to findings from nonhuman animal models, associations between altered cortisol levels and PFC and ACC structure have been found in studies of humans. Findings across multiple literatures suggest that frontolimbic circuits may be especially vulnerable. Figure created using BioRender.com. ACTH, adrenocorticotropic hormone; AVP, arginine vasopressin; CRH, corticotropin-releasing hormone.

regulate HPA axis activity, especially in studies of adults. Although more work is needed, the findings from this literature complement results showing that cortisol levels mediate associations between socioeconomic disadvantage and PFC function in children (109).

OVERALL SUMMARY AND DISCUSSION

Socioeconomic disadvantage has often been associated with reduced gray matter in the rostral middle frontal gyrus, inferior frontal gyrus, and ACC and lower FA in frontolimbic and frontoparietal tracts in children and adolescents (70,74–76,85). These circuits are heavily involved in cognitive and emotional control, which are transdiagnostic processes found to underlie multiple psychiatric disorders (140). Animal research points to altered HPA axis regulation as part of the mechanistic pathway potentially underlying these associations, and human research spanning various lines of inquiry is consistent with this possibility.

Socioeconomic disadvantage has been associated with altered cortisol secretion (either higher or lower levels) in childhood and adolescence (14,100,106,109,141). Initial increases in cortisol secretion and reactivity are thought to be adaptive changes that enhance detection of stressors in the short term and facilitate behavioral responses (15). Chronic stress may initially cause higher cortisol secretion, which eventually leads to lower cortisol secretion (13,126). Altered cortisol secretion has been associated with reduced gray matter in the PFC and ACC regions (14). These stress-related pathways may be especially salient for socioeconomic differences in the structure of the ventromedial PFC, ventral ACC, and limbic system (25,66). Reduced gray matter partially reflects reduced dendritic complexity, including spine density (36,61,62,142), which can lower synaptic connectivity (42).

Although fewer studies have been conducted on white matter microstructure, altered cortisol secretion has also been associated with FA in frontoparietal and frontolimbic tracts. FA as measured using diffusion-weighted imaging may reflect multiple processes (e.g., myelination, axonal packing) and cannot be attributed to any specific cellular mechanism (48,143,144). Chronic stress and elevated glucocorticoid levels lead to structural changes in glial cells (oligodendrocytes, astrocytes, microglia) that can reduce myelination in the PFC (48,145).

Research is needed that investigates the epigenetic processes that mediate the effects of childhood socioeconomic context on PFC structure, which may involve altered GR gene methylation (58), including research that employs epigenome-wide approaches. Childhood socioeconomic disadvantage may alter the expression of genes involved in glucocorticoid signaling, inflammation, and neuronal development, for example, impacting the development of PFC circuitry and leading to persistent reductions in cognitive and emotional control. In addition, associations between childhood socioeconomic disadvantage and HPA axis function are likely moderated by genetic factors, such as polymorphisms in genes involved in the HPA axis stress response (e.g., *FKBP5* genotype) (146). Research is needed to examine interactions between childhood socioeconomic disadvantage and genetic factors in the prediction of PFC structure and function, including studies that use genome-wide methods.

Socioeconomic disadvantage is a distal factor associated with multiple proximal factors that may play roles in these associations (6). Unpredictability as a specific dimension of stress exposure often characterizes socioeconomically disadvantaged environments (8,15) (e.g., household instability, residential changes, changes in household composition, parental unemployment, changing parental work schedules, less frequent family routines). Reduced cognitive and linguistic stimulation (e.g., deprivation) is another major pathway through which socioeconomic disadvantage has been proposed to affect the development of cognitive control (2–4,9,10).

Socioeconomic disadvantage has been consistently associated with reduced cognitive and linguistic stimulation including lower levels of linguistic input at home, limited access to material resources such as books and toys, and fewer opportunities to visit libraries and museums (2,3,9). Animal models have shown that lower cognitive stimulation reduces dendritic complexity and spine density in PFC and hippocampal neurons and decreases myelination (147,148). Similar results have been reported in human studies. For example, lower cognitive stimulation has been associated with reduced dlPFC thickness in children and adolescents (88). Exposure to both frequent chronic stressors and fewer cognitively enriching activities likely has stronger and differential effects on the HPA axis and PFC than exposure to one or the other. In animal models, for example, environmental enrichment reverses the effects of chronic stress on HPA axis regulation and PFC function (65,148,149). Future studies should examine multiple dimensions of proximal exposures, along with the severity of those exposures, when aiming to explain socioeconomic disparities in PFC structure and function.

The factors that mediate socioeconomic differences in brain structure fall along a dimension of more proximal (e.g., parent-child interactions) to more distal (e.g., neighborhood qualities), and whether these exposures are more proximal or more distal, along with their duration and severity, likely affects children's outcomes. At the most proximal level, parent-child interactions and relationship quality have powerful effects on the HPA axis during early childhood (150). While financial strain may increase parental stress, which in turn leads to lower parental warmth and responsiveness, many parents are able to provide supportive parenting to their children, even in circumstances of economic hardship. High levels of parental warmth and responsiveness have been found to reduce the effects of socioeconomic disadvantage on the developing HPA axis (151) and PFC structure and function (95,152,153).

Race/Ethnicity

Socioeconomic status is closely intertwined with race/ethnicity in the United States (2,4). Children from socioeconomically disadvantaged families are also often from racial/ethnic minority backgrounds and additionally exposed to chronic stress stemming from systemic racism, prejudice, and discrimination. Exposure to systemic racism and discrimination and socioeconomic disadvantage may have distinct effects on HPA axis regulation and PFC structure. For example, racial/ethnic disparities in hair cortisol concentrations persisted after controlling for socioeconomic factors in children (114), and exposure to racial discrimination was associated with lower FA in the cingulum and SLF in Black women (154). In a study using machine learning, when a wide range of predictors were included in the model, race (Black vs. non-Black) was not an important predictor of total brain volume in infants (155). Further research is needed to disentangle the effects of racial/ethnic discrimination and socioeconomic disadvantage on HPA axis regulation and PFC development in children.

Developmental Trajectories

During typical development, cortical volume and surface area increase into middle childhood and early adolescence and then decrease during subsequent phases of adolescence (143,156). Cortical thickness increases during the first 2 years of life and then decreases during early and middle childhood and adolescence (143). These trajectories are likely due in part to synaptogenesis followed by synaptic pruning. In diffusion-weighted imaging studies of typical development, FA increases steeply during the first few years of life and then more slowly throughout childhood and adolescence, due in part to myelination (31,143,144).

Some evidence suggests that socioeconomic disadvantage may accelerate cortical thinning throughout childhood and adolescence (144,157), including in frontoparietal circuitry (158). Chronic stress and reduced cognitive stimulation may accelerate cortical thinning due in part to increased synaptic pruning. Early-life stress may prompt accelerated frontolimbic development as an adaptation to the environment that confers benefits in the short term (16). Longitudinal studies are needed to investigate these possibilities. Most studies conducted to date have used cross-sectional designs, which are limited in their ability to support inferences about development (5,143).

Developmental Timing

Early childhood is the most frequently identified postnatal sensitive period of enhanced malleability of the HPA axis to environmental experiences (150). Animal models highlight the peripubertal period as another possible sensitive period (49,55,56). During puberty, the effects of chronic stressors on the HPA axis may relate to reduced gray matter in the PFC. These effects likely depend on early experiences and genetic factors. For example, elevated exposure to chronic stressors during puberty may exacerbate the effects of early-life adversity on HPA axis regulation and PFC development (13,150). Conversely, puberty also offers an opportunity for positive environments to counteract effects of early-life socioeconomic disadvantage.

Sex Hormones

Translational models indicate that sex may moderate the effects of chronic stress on HPA axis function (57,65) and frontolimbic morphology (41,159,160), and sex differences have been found in some human studies (96,105,123,161). The hypothalamic-pituitary-gonadal axis, which is responsible for pubertal elevations in testosterone and estrogen, interacts with the HPA axis (13,162). The way in which socioeconomic disadvantage impacts both the HPA and hypothalamic-pituitary-gonadal axes (e.g., through pubertal timing) may have implications for the development of PFC circuitry involved in cognitive and emotional control (79). For example, early poverty has been found to reduce increases in testosterone levels from school age to adolescence, which was associated with smaller hippocampal volume and more difficulty with emotion regulation (162). Cortisol reactivity may also interact with increases in gonadal hormones during puberty, leading to changes in PFC structure and sex differences in risk for affective psychopathology. Future studies are needed to elucidate the role of the hypothalamic-pituitary-gonadal axis in these mechanisms.

IMPLICATIONS FOR PRACTICE AND POLICY

The biological embedding of early socioeconomic context reinforces the need for policies that support families with low levels of socioeconomic resources. Addressing the structural

determinants that lead to childhood socioeconomic disadvantage is necessary to ensure equitable opportunities for mental health across the life span. Social safety net programs, including cash transfers, health insurance, and nutritional assistance for low-income families, vary in their generosity (2). Evidence suggests that policies that reduce economic hardship could bolster children's brain development and mental health (2,163). In a randomized controlled trial, providing low-income families with unconditional cash transfers was found to support infant brain function (163).

CONCLUSIONS

Converging evidence points to the key roles of the HPA axis and PFC in the mechanisms underlying socioeconomic disparities in cognitive and emotional control. Socioeconomic disadvantage is associated with elevated exposure to chronic stress, which in animal models when experienced early in life exerts pronounced and lasting effects on PFC structure and function in part through effects on the HPA axis. Socioeconomic disadvantage has frequently been associated with altered cortisol levels and reduced PFC gray matter in children, with these adaptive changes thought to confer short-term benefits in a stressful environment. Cultivating supportive environments for children by reducing economic hardship is critical to promoting brain development and long-term mental health. Public policies that assist families with low socioeconomic resources should be a priority to ensure that all children have strong foundations for health and well-being.

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REFERENCES

1. Wadsworth ME, Evans GW, Grant K, Carter JS, Duffy S (2016): Poverty and the development of psychopathology. In: Cicchetti D, editor. Developmental psychopathology: Risk, resilience, and intervention, vol. 4. Hoboken, NJ: John Wiley & Sons, Inc., 136–179.
2. Duncan GJ, Magnuson K, Votruba-Drzal E (2017): Moving beyond correlations in assessing the consequences of poverty. Annu Rev Psychol 68:413–434.
3. Bradley RH, Corwyn RF (2002): Socioeconomic status and child development. Annu Rev Psychol 53:371–399.
4. McLoyd VC (1998): Socioeconomic disadvantage and child development. Am Psychol 53:185–204.
5. Farah MJ (2017): The neuroscience of socioeconomic status: Correlates, causes, and consequences. Neuron 96:56–71.
6. Bronfenbrenner U, Morris PA (1998): The ecology of developmental processes. In: Damon W, Lerner RM, editors: Handbook of Child Psychology: Theoretical Models of Human Development, 5th ed, vol. 1. Hoboken, NJ: John Wiley & Sons Inc., 993–1028
7. Chen E, Cohen S, Miller GE (2010): How low socioeconomic status affects 2-year hormonal trajectories in children. Psychol Sci 21:31–37.
8. Evans GW, Kim P (2013): Childhood poverty, chronic stress, self-regulation, and coping. Child Dev Perspect 7:43–48.
9. Conger RD, Donnellan MB (2007): An interactionist perspective on the socioeconomic context of human development. Annu Rev Psychol 58:175–199.
10. Gershoff ET, Aber JL, Raver CC, Lennon MC (2007): Income is not enough: Incorporating material hardship into models of income associations with parenting and child development. Child Dev 78:70–95.
11. McCoy DC, Roy AL, Raver CC (2016): Neighborhood crime as a predictor of individual differences in emotional processing and regulation. Dev Sci 19:164–174.
12. Dufford AJ, Bianco H, Kim P (2019): Socioeconomic disadvantage, brain morphometry, and attentional bias to threat in middle childhood. Cogn Affect Behav Neurosci 19:309–326.
13. Koss KJ, Gunnar MR (2018): Annual Research Review: Early adversity, the hypothalamic-pituitary-adrenocortical axis, and child psychopathology. J Child Psychol Psychiatry 59:327–346.
14. Merz EC, Desai PM, Maskus EA, Melvin SA, Rehman R, Torres SD, *et al.* (2019): Socioeconomic disparities in chronic physiologic stress are associated with brain structure in children. Biol Psychiatry 86:921–929.
15. Ellis BJ, Del Giudice M (2019): Developmental adaptation to stress: An evolutionary perspective. Annu Rev Psychol 70:111–139.
16. Callaghan BL, Tottenham N (2016): The Stress Acceleration Hypothesis: Effects of early-life adversity on emotion circuits and behavior. Curr Opin Behav Sci 7:76–81.
17. Herman JP, McKlveen JM, Ghosal S, Kopp B, Wulsin A, Makinson R, *et al.* (2016): Regulation of the hypothalamic-pituitary-adrenocortical stress response. Compr Physiol 6:603–621.
18. de Kloet ER, Joëls M, Holsboer F (2005): Stress and the brain: From adaptation to disease. Nat Rev Neurosci 6:463–475.
19. Diorio D, Viau V, Meaney MJ (1993): The role of the medial prefrontal cortex (cingulate gyrus) in the regulation of hypothalamic-pituitary-adrenal responses to stress. J Neurosci 13:3839–3847.
20. McKlveen JM, Myers B, Flak JN, Bundzikova J, Solomon MB, Seroogy KB, Herman JP (2013): Role of prefrontal cortex glucocorticoid receptors in stress and emotion. Biol Psychiatry 74:672–679.
21. Myers B, McKlveen JM, Herman JP (2014): Glucocorticoid actions on synapses, circuits, and behavior: Implications for the energetics of stress. Front Neuroendocrinol 35:180–196.
22. Oitzl MS, Champagne DL, van der Veen R, de Kloet ER (2010): Brain development under stress: Hypotheses of glucocorticoid actions revisited. Neurosci Biobehav Rev 34:853–866.
23. Herman JP, McKlveen JM, Solomon MB, Carvalho-Netto E, Myers B (2012): Neural regulation of the stress response: Glucocorticoid feedback mechanisms. Braz J Med Biol Res 45:292–298.
24. Crone EA, Steinbeis N (2017): Neural perspectives on cognitive control development during childhood and adolescence. Trends Cogn Sci 21:205–215.
25. Etkin A, Büchel C, Gross JJ (2015): The neural bases of emotion regulation. Nat Rev Neurosci 16:693–700.
26. Judd N, Sauce B, Wiedenhoeft J, Tromp J, Chaarani B, Schliep A, *et al.* (2020): Cognitive and brain development is independently influenced by socioeconomic status and polygenic scores for educational attainment. Proc Natl Acad Sci USA 117:12411–12418.
27. Niendam TA, Laird AR, Ray KL, Dean YM, Glahn DC, Carter CS (2012): Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. Cogn Affect Behav Neurosci 12:241–268.
28. Gratton G, Cooper P, Fabiani M, Carter CS, Karayanidis F (2018): Dynamics of cognitive control: Theoretical bases, paradigms, and a view for the future. Psychophysiology 55:e13016.
29. Ochsner KN, Gross JJ (2005): The cognitive control of emotion. Trends Cogn Sci 9:242–249.

30. Lichtin RD, Merz EC, He X, Desai PM, Simon KR, Melvin SA, *et al.* (2021): Material hardship, prefrontal cortex-amygdala structure, and internalizing symptoms in children. Dev Psychobiol 63:364–377.
31. Peters BD, Ikuta T, DeRosse P, John M, Burdick KE, Gruner P, *et al.* (2014): Age-related differences in white matter tract microstructure are associated with cognitive performance from childhood to adulthood. Biol Psychiatry 75:248–256.
32. Etkin A, Egner T, Peraza DM, Kandel ER, Hirsch J (2006): Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. Neuron 51:871–882.
33. Buhle JT, Silvers JA, Wager TD, Lopez R, Onyemekwu C, Kober H, *et al.* (2014): Cognitive reappraisal of emotion: A meta-analysis of human neuroimaging studies. Cereb Cortex 24:2981–2990.
34. Milad MR, Quirk GJ (2012): Fear extinction as a model for translational neuroscience: Ten years of progress. Annu Rev Psychol 63:129–151.
35. Arnsten AFT (2015): Stress weakens prefrontal networks: Molecular insults to higher cognition. Nat Neurosci 18:1376–1385.
36. McEwen BS, Morrison JH (2013): The brain on stress: Vulnerability and plasticity of the prefrontal cortex over the life course. Neuron 79:16–29.
37. Chen Y, Baram TZ (2016): Toward understanding how early-life stress reprograms cognitive and emotional brain networks. Neuropsychopharmacology 41:197–206.
38. Anderson RM, Johnson SB, Lingg RT, Hinz DC, Romig-Martin SA, Radley JJ (2020): Evidence for similar prefrontal structural and functional alterations in male and female rats following chronic stress or glucocorticoid exposure. Cereb Cortex 30:353–370.
39. Perry RE, Rincón-Cortés M, Braren SH, Brandes-Aitken AN, Opendak M, Pollonini G, *et al.* (2019): Corticosterone administration targeting a hypo-reactive HPA axis rescues a socially-avoidant phenotype in scarcity-adversity reared rats. Dev Cogn Neurosci 40: 100716.
40. Cook SC, Wellman CL (2004): Chronic stress alters dendritic morphology in rat medial prefrontal cortex. J Neurobiol 60:236–248.
41. Garrett JE, Wellman CL (2009): Chronic stress effects on dendritic morphology in medial prefrontal cortex: Sex differences and estrogen dependence. Neuroscience 162:195–207.
42. Liston C, Miller MM, Goldwater DS, Radley JJ, Rocher AB, Hof PR, *et al.* (2006): Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. J Neurosci 26:7870–7874.
43. Radley JJ, Sisti HM, Hao J, Rocher AB, McCall T, Hof PR, *et al.* (2004): Chronic behavioral stress induces apical dendritic reorganization in pyramidal neurons of the medial prefrontal cortex. Neuroscience 125:1–6.
44. Radley JJ, Rocher AB, Janssen WGM, Hof PR, McEwen BS, Morrison JH (2005): Reversibility of apical dendritic retraction in the rat medial prefrontal cortex following repeated stress. Exp Neurol 196:199–203.
45. Radley JJ, Rocher AB, Miller M, Janssen WGM, Liston C, Hof PR, *et al.* (2006): Repeated stress induces dendritic spine loss in the rat medial prefrontal cortex. Cereb Cortex 16:313–320.
46. Liu RJ, Aghajanian GK (2008): Stress blunts serotonin- and hypocretin-evoked EPSCs in prefrontal cortex: Role of corticosterone-mediated apical dendritic atrophy. Proc Natl Acad Sci USA 105:359–364.
47. Albrecht A, Müller I, Ardi Z, Çalışkan G, Gruber D, Ivens S, *et al.* (2017): Neurobiological consequences of juvenile stress: A GABAergic perspective on risk and resilience. Neurosci Biobehav Rev 74:21–43.
48. Kaul D, Schwab SG, Mechawar N, Matosin N (2021): How stress physically re-shapes the brain: Impact on brain cell shapes, numbers and connections in psychiatric disorders. Neurosci Biobehav Rev 124:193–215.
49. Romeo RD (2017): The impact of stress on the structure of the adolescent brain: Implications for adolescent mental health. Brain Res 1654:185–191.
50. Bock J, Gruss M, Becker S, Braun K (2005): Experience-induced changes of dendritic spine densities in the prefrontal and sensory cortex: Correlation with developmental time windows. Cereb Cortex 15:802–808.
51. Chocyk A, Bobula B, Dudys D, Przyborowska A, Majcher-Maślanka I, Hess G, Wędzony K (2013): Early-life stress affects the structural and functional plasticity of the medial prefrontal cortex in adolescent rats. Eur J Neurosci 38:2089–2107.
52. Monroy E, Hernández-Torres E, Flores G (2010): Maternal separation disrupts dendritic morphology of neurons in prefrontal cortex, hippocampus, and nucleus accumbens in male rat offspring. J Chem Neuroanat 40:93–101.
53. Yang XD, Liao XM, Uribe-Mariño A, Liu R, Xie XM, Jia J, *et al.* (2015): Stress during a critical postnatal period induces region-specific structural abnormalities and dysfunction of the prefrontal cortex via CRF1. Neuropsychopharmacology 40:1203–1215.
54. Romano-López A, Méndez-Díaz M, García FG, Regalado-Santiago C, Ruiz-Contreras AE, Prospéro-García O (2016): Maternal separation and early stress cause long-lasting effects on dopaminergic and endocannabinergic systems and alters dendritic morphology in the nucleus accumbens and frontal cortex in rats. Dev Neurobiol 76:819–831.
55. Eiland L, Ramroop J, Hill MN, Manley J, McEwen BS (2012): Chronic juvenile stress produces corticolimbic dendritic architectural remodeling and modulates emotional behavior in male and female rats. Psychoneuroendocrinology 37:39–47.
56. Pinzón-Parra C, Vidal-Jiménez B, Camacho-Abrego I, Flores-Gómez AA, Rodríguez-Moreno A, Flores G (2019): Juvenile stress causes reduced locomotor behavior and dendritic spine density in the prefrontal cortex and basolateral amygdala in Sprague–Dawley rats. Synapse 73:e22066.
57. Barha CK, Brummelte S, Lieblich SE, Galea LAM (2011): Chronic restraint stress in adolescence differentially influences hypothalamic-pituitary-adrenal axis function and adult hippocampal neurogenesis in male and female rats. Hippocampus 21:1216–1227.
58. Turecki G, Meaney MJ (2016): Effects of the social environment and stress on glucocorticoid receptor gene methylation: A systematic review. Biol Psychiatry 79:87–96.
59. Herman JP, Ostrander MM, Mueller NK, Figueiredo H (2005): Limbic system mechanisms of stress regulation: Hypothalamo-pituitary-adrenocortical axis. Prog Neuropsychopharmacol Biol Psychiatry 29:1201–1213.
60. Cerqueira JJ, Pêgo JM, Taipa R, Bessa JM, Almeida OFX, Sousa N (2005): Morphological correlates of corticosteroid-induced changes in prefrontal cortex-dependent behaviors. J Neurosci 25:7792–7800.
61. Gourley SL, Swanson AM, Koleske AJ (2013): Corticosteroid-induced neural remodeling predicts behavioral vulnerability and resilience. J Neurosci 33:3107–3112.
62. Wellman CL (2001): Dendritic reorganization in pyramidal neurons in medial prefrontal cortex after chronic corticosterone administration. J Neurobiol 49:245–253.
63. Anderson RM, Glanz RM, Johnson SB, Miller MM, Romig-Martin SA, Radley JJ (2016): Prolonged corticosterone exposure induces dendritic spine remodeling and attrition in the rat medial prefrontal cortex. J Comp Neurol 524:3729–3746.
64. Wulsin AC, Wick-Carlson D, Packard BA, Morano R, Herman JP (2016): Adolescent chronic stress causes hypothalamo–pituitary–adrenocortical hypo-responsiveness and depression-like behavior in adult female rats. Psychoneuroendocrinology 65:109–117.
65. Smith BL, Morano RL, Ulrich-Lai YM, Myers B, Solomon MB, Herman JP (2018): Adolescent environmental enrichment prevents behavioral and physiological sequelae of adolescent chronic stress in female (but not male) rats. Stress 21:464–473.
66. Arnsten AFT, Joyce MKP, Roberts AC (2023): The Aversive Lens: Stress effects on the prefrontal-cingulate cortical pathways that regulate emotion. Neurosci Biobehav Rev 145:105000.
67. Hair NL, Hanson JL, Wolfe BL, Pollak SD (2015): Association of child poverty, brain development, and academic achievement. JAMA Pediatr 169:822–829.

68. Hanson JL, Hair N, Shen DG, Shi F, Gilmore JH, Wolfe BL, Pollak SD (2013): Family poverty affects the rate of human infant brain growth. PLoS One 8:e80954.
69. Lawson GM, Duda JT, Avants BB, Wu J, Farah MJ (2013): Associations between children's socioeconomic status and prefrontal cortical thickness. Dev Sci 16:641–652.
70. Noble KG, Houston SM, Brito NH, Bartsch H, Kan E, Kuperman JM, *et al.* (2015): Family income, parental education and brain structure in children and adolescents. Nat Neurosci 18:773–778.
71. Jednoróg K, Altarelli I, Monzalvo K, Fluss J, Dubois J, Billard C, *et al.* (2012): The influence of socioeconomic status on children's brain structure. PLoS One 7:e42486.
72. King LS, Dennis EL, Humphreys KL, Thompson PM, Gotlib IH (2020): Cross-sectional and longitudinal associations of family income-to-needs ratio with cortical and subcortical brain volume in adolescent boys and girls. Dev Cogn Neurosci 44:100796.
73. Romeo RR, Christodoulou JA, Halverson KK, Murtagh J, Cyr AB, Schimmel C, *et al.* (2018): Socioeconomic status and reading disability: Neuroanatomy and plasticity in response to intervention. Cereb Cortex 28:2297–2312.
74. Tomasi D, Volkow ND (2021): Associations of family income with cognition and brain structure in USA children: Prevention implications. Mol Psychiatry 26:6619–6629.
75. Taylor RL, Cooper SR, Jackson JJ, Barch DM (2020): Assessment of neighborhood poverty, cognitive function, and prefrontal and hippocampal volumes in children. JAMA Netw Open 3:e2023774.
76. Vargas T, Damme KSF, Mittal VA (2020): Neighborhood deprivation, prefrontal morphology and neurocognition in late childhood to early adolescence. NeuroImage 220:117086.
77. Hackman DA, Cserbik D, Chen JC, Berhane K, Minaravesh B, McConnell R, Herting MM (2021): Association of local variation in neighborhood disadvantage in metropolitan areas with youth neurocognition and brain structure. JAMA Pediatr 175:e210426.
78. McDermott CL, Seidlitz J, Nadig A, Liu S, Clasen LS, Blumenthal JD, *et al.* (2019): Longitudinally mapping childhood socioeconomic status associations with cortical and subcortical morphology. J Neurosci 39:1365–1373.
79. Sanders AFP, Baum GL, Harms MP, Kandala S, Bookheimer SY, Dapretto M, *et al.* (2022): Developmental trajectories of cortical thickness by functional brain network: The roles of pubertal timing and socioeconomic status. Dev Cogn Neurosci 57: 101145.
80. Gur RE, Moore TM, Rosen AFG, Barzilay R, Roalf DR, Calkins ME, *et al.* (2019): Burden of environmental adversity associated with psychopathology, maturation, and brain behavior parameters in youths. JAMA Psychiatry 76:966–975.
81. Mackey AP, Finn AS, Leonard JA, Jacoby-Senghor DS, West MR, Gabrieli CFO, Gabrieli JDE (2015): Neuroanatomical correlates of the income-achievement gap. Psychol Sci 26:925–933.
82. Noble KG, Houston SM, Kan E, Sowell ER (2012): Neural correlates of socioeconomic status in the developing human brain. Dev Sci 15:516–527.
83. Spann MN, Bansal R, Hao X, Rosen TS, Peterson BS (2020): Prenatal Socioeconomic Status and Social Support are associated with Neonatal Brain Morphology, Toddler Language and Psychiatric Symptoms. Child Neuropsychol 26:170–188.
84. Lu YC, Kapse K, Andersen N, Quistorff J, Lopez C, Fry A, *et al.* (2021): Association between socioeconomic status and in utero fetal brain development. JAMA Netw Open 4:e213526.
85. Dufford AJ, Kim P (2017): Family income, cumulative risk exposure, and white matter structure in middle childhood. Front Hum Neurosci 11:547.
86. Gullick MM, Demir-Lira Ö.E, Booth JR (2016): Reading skill–fractional anisotropy relationships in visuospatial tracts diverge depending on socioeconomic status. Dev Sci 19:673–685.
87. Ozernov-Palchik O, Norton ES, Wang Y, Beach SD, Zuk J, Wolf M, *et al.* (2019): The relationship between socioeconomic status and white matter microstructure in pre-reading children: A longitudinal investigation. Hum Brain Mapp 40:741–754.
88. Rosen ML, Sheridan MA, Sambrook KA, Meltzoff AN, McLaughlin KA (2018): Socioeconomic disparities in academic achievement: A multimodal investigation of neural mechanisms in children and adolescents. NeuroImage 173:298–310.
89. Ursache A, Noble KG, Pediatric Imaging, Neurocognition and Genetics Study (2016): Socioeconomic status, white matter, and executive function in children. Brain Behav 6:e00531.
90. Li ZA, Cai Y, Taylor RL, Eisenstein SA, Barch DM, Marek S, Hershey T (2023): Associations between socioeconomic status, obesity, cognition, and white matter microstructure in children. JAMA Netw Open 6:e2320276.
91. Bell KL, Purcell JB, Harnett NG, Goodman AM, Mrug S, Schuster MA, *et al.* (2021): White matter microstructure in the young adult brain varies with neighborhood disadvantage in adolescence. Neuroscience 466:162–172.
92. Dufford AJ, Evans GW, Dmitrieva J, Swain JE, Liberzon I, Kim P (2020): Prospective associations, longitudinal patterns of childhood socioeconomic status, and white matter organization in adulthood. Hum Brain Mapp 41:3580–3593.
93. Vanderauwera J, van Setten ERH, Maurits NM, Maassen BAM (2019): The interplay of socio-economic status represented by paternal educational level, white matter structure and reading. PLoS One 14: e0215560.
94. Simon KR, Merz EC, He X, Desai PM, Meyer JS, Noble KG (2021): Socioeconomic factors, stress, hair cortisol, and white matter microstructure in children. Dev Psychobiol 63:e22147.
95. Ziegler G, Moutoussis M, Hauser TU, Fearon P, Bullmore ET, Goodyer IM, *et al.* (2020): Childhood socio-economic disadvantage predicts reduced myelin growth across adolescence and young adulthood. Hum Brain Mapp 41:3392–3402.
96. Kim DJ, Davis EP, Sandman CA, Glynn L, Sporns O, O'Donnell BF, Hetrick WP (2019): Childhood poverty and the organization of structural brain connectome. NeuroImage 184:409–416.
97. Short SJ, Stalder T, Marceau K, Entringer S, Moog NK, Shirtcliff EA, *et al.* (2016): Correspondence between hair cortisol concentrations and 30-day integrated daily salivary and weekly urinary cortisol measures. Psychoneuroendocrinology 71:12–18.
98. Gustafsson PE, Gustafsson PA, Nelson N (2006): Cortisol levels and psychosocial factors in preadolescent children. Stress Health 22:3–9.
99. Lupien SJ, King S, Meaney MJ, McEwen BS (2001): Can poverty get under your skin? basal cortisol levels and cognitive function in children from low and high socioeconomic status. Dev Psychopathol 13:653–676.
100. Desantis AS, Kuzawa CW, Adam EK (2015): Developmental origins of flatter cortisol rhythms: Socioeconomic status and adult cortisol activity. Am J Hum Biol 27:458–467.
101. Zhu Y, Chen X, Zhao H, Chen M, Tian Y, Liu C, *et al.* (2019): Socioeconomic status disparities affect children's anxiety and stress-sensitive cortisol awakening response through parental anxiety. Psychoneuroendocrinology 103:96–103.
102. Clearfield MW, Carter-Rodriguez A, Merali AR, Shober R (2014): The effects of SES on infant and maternal diurnal salivary cortisol output. Infant Behav Dev 37:298–304.
103. Tarullo AR, Tuladhar CT, Kao K, Drury EB, Meyer J (2020): Cortisol and socioeconomic status in early childhood: A multidimensional assessment. Dev Psychopathol 32:1876–1887.
104. Fernald LCH, Gunnar MR (2009): Poverty-alleviation program participation and salivary cortisol in very low-income children. Soc Sci Med 68:2180–2189.
105. McFarland MJ, Hayward MD (2014): Poverty and awakening cortisol in adolescence: The importance of timing in early life. Soc Ment Health 4:21–37.
106. Zalewski M, Lengua LJ, Kiff CJ, Fisher PA (2012): Understanding the relation of low income to HPA-axis functioning in preschool children: Cumulative family risk and parenting as pathways to disruptions in cortisol. Child Psychiatry Hum Dev 43:924–942.
107. West P, Sweeting H, Young R, Kelly S (2010): The relative importance of family socioeconomic status and school-based peer hierarchies

for morning cortisol in youth: An exporatory study. Soc Sci Med 70:1246–1253.
108. Saridjan NS, Huizink AC, Koetsier JA, Jaddoe VW, Mackenbach JP, Hofman A, *et al.* (2010): Do social disadvantage and early family adversity affect the diurnal cortisol rhythm in infants? The generation R Study. Horm Behav 57:247–254.
109. Tian T, Young CB, Zhu Y, Xu J, He Y, Chen M, *et al.* (2021): Socioeconomic disparities affect children's amygdala-prefrontal circuitry via stress hormone response. Biol Psychiatry 90:173–181.
110. Marsman R, Nederhof E, Rosmalen JGM, Oldehinkel AJ, Ormel J, Buitelaar JK (2012): Family environment is associated with HPA-axis activity in adolescents. The TRAILS study. Biol Psychol 89:460–466.
111. Bhopal S, Verma D, Roy R, Soremekun S, Kumar D, Bristow M, *et al.* (2019): The contribution of childhood adversity to cortisol measures of early life stress amongst infants in rural India: Findings from the early life stress sub-study of the SPRING cluster randomised controlled trial (Spring-ELS). Psychoneuroendocrinology 107:241–250.
112. Deer LK, Shields GS, Alen NV, Hostinar CE (2021): Curvilinear associations between family income in early childhood and the cortisol awakening response in adolescence. Psychoneuroendocrinology 129:105237.
113. Evans GW, Kim P (2007): Childhood poverty and health: Cumulative risk exposure and stress dysregulation. Psychol Sci 18:953–957.
114. Anand KJS, Rovnaghi CR, Rigdon J, Qin F, Tembulkar S, Murphy LE, *et al.* (2020): Demographic and psychosocial factors associated with hair cortisol concentrations in preschool children. Pediatr Res 87:1119–1127.
115. Schloß S, Müller V, Becker K, Skoluda N, Nater UM, Pauli-Pott U (2019): Hair cortisol concentration in mothers and their children: Roles of maternal sensitivity and child symptoms of attention-deficit/hyperactivity disorder. J Neural Transm (Vienna) 126:1135–1144.
116. Vaghri Z, Guhn M, Weinberg J, Grunau RE, Yu W, Hertzman C (2013): Hair cortisol reflects socio-economic factors and hair zinc in preschoolers. Psychoneuroendocrinology 38:331–340.
117. Rippe RCA, Noppe G, Windhorst DA, Tiemeier H, van Rossum EFC, Jaddoe VWV, *et al.* (2016): Splitting hair for cortisol? Associations of socio-economic status, ethnicity, hair color, gender and other child characteristics with hair cortisol and cortisone. Psychoneuroendocrinology 66:56–64.
118. Vliegenthart J, Noppe G, van Rossum EFC, Koper JW, Raat H, van den Akker ELT (2016): Socioeconomic status in children is associated with hair cortisol levels as a biological measure of chronic stress. Psychoneuroendocrinology 65:9–14.
119. Gerber M, Endes K, Brand S, Herrmann C, Colledge F, Donath L, *et al.* (2017): In 6- to 8-year-old children, hair cortisol is associated with body mass index and somatic complaints, but not with stress, health-related quality of life, blood pressure, retinal vessel diameters, and cardiorespiratory fitness. Psychoneuroendocrinology 76:1–10.
120. Groeneveld MG, Vermeer HJ, Linting M, Noppe G, van Rossum EFC, van IJzendoorn MH (2013): Children's hair cortisol as a biomarker of stress at school entry. Stress 16:711–715.
121. Hoffman MC, D'Anna-Hernandez K, Benitez P, Ross RG, Laudenslager ML (2017): Cortisol during human fetal life: Characterization of a method for processing small quantities of newborn hair from 26 to 42 weeks gestation. Dev Psychobiol 59:123–127.
122. Karlén J, Frostell A, Theodorsson E, Faresjö T, Ludvigsson J (2013): Maternal influence on child HPA axis: A prospective study of cortisol levels in hair. Pediatrics 132:e1333–e1340.
123. Ouellet-Morin I, Cantave C, Lupien S, Geoffroy MC, Brendgen M, Vitaro F, *et al.* (2021): Cumulative exposure to socioeconomic and psychosocial adversity and hair cortisol concentration: A longitudinal study from 5 months to 17 years of age. Psychoneuroendocrinology 126:105153.
124. Simmons JG, Azpitarte F, Roost FD, Dommers E, Allen NB, Havighurst S, Haslam N (2019): Correlates of hair cortisol concentrations in disadvantaged young children. Stress Health 35:104–111.
125. Malanchini M, Engelhardt LE, Raffington LA, Sabhlok A, Grotzinger AD, Briley DA, *et al.* (2021): Weak and uneven associations of home, neighborhood, and school environments with stress hormone output across multiple timescales. Mol Psychiatry 26:4823–4838.
126. Miller GE, Chen E, Zhou ES (2007): If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. Psychol Bull 133:25–45.
127. Kremen WS, O'Brien RC, Panizzon MS, Prom-Wormley E, Eaves LJ, Eisen SA, *et al.* (2010): Salivary cortisol and prefrontal cortical thickness in middle-aged men: A twin study. NeuroImage 53:1093–1102.
128. Green C, Stolicyn A, Harris MA, Shen X, Romaniuk L, Barbu MC, *et al.* (2021): Hair glucocorticoids are associated with childhood adversity, depressive symptoms and reduced global and lobar grey matter in Generation Scotland. Transl Psychiatry 11:523.
129. Klinger-König J, Frenzel S, Hannemann A, Wittfeld K, Bülow R, Friedrich N, *et al.* (2021): Sex differences in the association between basal serum cortisol concentrations and cortical thickness. Neurobiol Stress 15:100416.
130. Lu S, Gao W, Wei Z, Wu W, Liao M, Ding Y, *et al.* (2013): Reduced cingulate gyrus volume associated with enhanced cortisol awakening response in young healthy adults reporting childhood trauma. PLoS One 8:e69350.
131. Echouffo-Tcheugui JB, Conner SC, Himali JJ, Maillard P, DeCarli CS, Beiser AS, *et al.* (2018): Circulating cortisol and cognitive and structural brain measures: The Framingham Heart Study. Neurology 91:e1961–e1970.
132. Stomby A, Boraxbekk CJ, Lundquist A, Nordin A, Nilsson LG, Adolfsson R, *et al.* (2016): Higher diurnal salivary cortisol levels are related to smaller prefrontal cortex surface area in elderly men and women. Eur J Endocrinol 175:117–126.
133. Carrion VG, Weems CF, Richert K, Hoffman BC, Reiss AL (2010): Decreased prefrontal cortical volume associated with increased bedtime cortisol in traumatized youth. Biol Psychiatry 68:491–493.
134. Feola B, Dougherty LR, Riggins T, Bolger DJ (2020): Prefrontal cortical thickness mediates the association between cortisol reactivity and executive function in childhood. Neuropsychologia 148:107636.
135. Liu J, Chaplin TM, Wang F, Sinha R, Mayes LC, Blumberg HP (2012): Stress reactivity and corticolimbic response to emotional faces in adolescents. J Am Acad Child Adolesc Psychiatry 51:304–312.
136. Chen R, Muetzel RL, El Marroun H, Noppe G, van Rossum EFC, Jaddoe VW, *et al.* (2016): No association between hair cortisol or cortisone and brain morphology in children. Psychoneuroendocrinology 74:101–110.
137. Sheikh HI, Joanisse MF, Mackrell SM, Kryski KR, Smith HJ, Singh SM, Hayden EP (2014): Links between white matter microstructure and cortisol reactivity to stress in early childhood: Evidence for moderation by parenting. NeuroImage Clin 6:77–85.
138. Kircanski K, Sisk LM, Ho TC, Humphreys KL, King LS, Colich NL, *et al.* (2019): Early life stress, cortisol, frontolimbic connectivity, and depressive symptoms during puberty. Dev Psychopathol 31:1011–1022.
139. Wheelock MD, Goodman AM, Harnett NG, Wood KH, Mrug S, Granger DA, Knight DC (2021): Sex-related differences in stress reactivity and cingulum white matter. Neuroscience 459:118–128.
140. Aldao A, Gee DG, De Los Reyes A, Seager I (2016): Emotion regulation as a transdiagnostic factor in the development of internalizing and externalizing psychopathology: Current and future directions. Dev Psychopathol 28:927–946.
141. Chen E, Paterson LQ (2006): Neighborhood, family, and subjective socioeconomic status: How do they relate to adolescent health? Health Psychol 25:704–714.
142. Kassem MS, Lagopoulos J, Stait-Gardner T, Price WS, Chohan TW, Arnold JC, *et al.* (2013): Stress-induced grey matter loss determined by MRI is primarily due to loss of dendrites and their synapses. Mol Neurobiol 47:645–661.
143. Mills KL, Tamnes CK (2014): Methods and considerations for longitudinal structural brain imaging analysis across development. Dev Cogn Neurosci 9:172–190.

144. Tooley UA, Bassett DS, Mackey AP (2021): Environmental influences on the pace of brain development. Nat Rev Neurosci 22:372–384.
145. Teissier A, Le Magueresse C, Olusakin J, Andrade da Costa BLS, De Stasi AM, Bacci A, *et al.* (2020): Early-life stress impairs postnatal oligodendrogenesis and adult emotional behaviour through activity-dependent mechanisms. Mol Psychiatry 25:1159–1174.
146. Matosin N, Halldorsdottir T, Binder EB (2018): Understanding the molecular mechanisms underpinning gene by environment interactions in psychiatric disorders: The FKBP5 model. Biol Psychiatry 83:821–830.
147. Kozorovitskiy Y, Gross CG, Kopil C, Battaglia L, McBreen M, Stranahan AM, Gould E (2005): Experience induces structural and biochemical changes in the adult primate brain. Proc Natl Acad Sci USA 102:17478–17482.
148. Smail MA, Smith BL, Nawreen N, Herman JP (2020): Differential impact of stress and environmental enrichment on corticolimbic circuits. Pharmacol Biochem Behav 197:172993.
149. Francis DD, Diorio J, Plotsky PM, Meaney MJ (2002): Environmental enrichment reverses the effects of maternal separation on stress reactivity. J Neurosci 22:7840–7843.
150. Engel ML, Gunnar MR (2020): The development of stress reactivity and regulation during human development. Int Rev Neurobiol 150:41–76.
151. Johnson AB, Mliner SB, Depasquale CE, Troy M, Gunnar MR (2018): Attachment security buffers the HPA axis of toddlers growing up in poverty or near poverty: Assessment during pediatric well-child exams with inoculations. Psychoneuroendocrinology 95:120–127.
152. Brody GH, Yu T, Nusslock R, Barton AW, Miller GE, Chen E, *et al.* (2019): The protective effects of supportive parenting on the relationship between adolescent poverty and resting-state functional brain connectivity during adulthood. Psychol Sci 30:1040–1049.
153. Whittle S, Vijayakumar N, Simmons JG, Dennison M, Schwartz O, Pantelis C, *et al.* (2017): Role of positive parenting in the association between neighborhood social disadvantage and brain development across adolescence. JAMA Psychiatry 74:824–832.
154. Fani N, Harnett NG, Bradley B, Mekawi Y, Powers A, Stevens JS, *et al.* (2022): Racial discrimination and white matter microstructure in trauma-exposed black women. Biol Psychiatry 91:254–261.
155. Sarullo K, Barch DM, Smyser CD, Rogers C, Warner BB, Miller JP, *et al.* (2023): Disentangling socioeconomic status and race in infant brain, birth weight, and gestational age at birth: A neural network analysis [published online May 22]. Biol Psychiatry Glob Open Sci.
156. Brain Development Cooperative Group (2012): Total and regional brain volumes in a population-based normative sample from 4 to 18 years: The NIH MRI study of normal brain development. Cereb Cortex 22:1–12.
157. Piccolo LR, Merz EC, He X, Sowell ER, Noble KG, Pediatric Imaging, Neurocognition, Genetics Study (2016): Age-related differences in cortical thickness vary by socioeconomic status. PLoS One 11: e0162511.
158. Colich NL, Rosen ML, Williams ES, McLaughlin KA (2020): Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. Psychol Bull 146:721–764.
159. Farrell MR, Holland FH, Shansky RM, Brenhouse HC (2016): Sex-specific effects of early life stress on social interaction and prefrontal cortex dendritic morphology in young rats. Behav Brain Res 310:119–125.
160. Shansky RM, Hamo C, Hof PR, Lou W, McEwen BS, Morrison JH (2010): Estrogen promotes stress sensitivity in a prefrontal cortex–amygdala pathway. Cereb Cortex 20:2560–2567.
161. Raffington L, Prindle J, Keresztes A, Binder J, Heim C, Shing YL (2018): Blunted cortisol stress reactivity in low–income children relates to lower memory function. Psychoneuroendocrinology 90:110–121.
162. Barch DM, Shirtcliff EA, Elsayed NM, Whalen D, Gilbert K, Vogel AC, *et al.* (2020): Testosterone and hippocampal trajectories mediate relationship of poverty to emotion dysregulation and depression. Proc Natl Acad Sci USA 117:22015–22023.
163. Troller-Renfree SV, Costanzo MA, Duncan GJ, Magnuson K, Gennetian LA, Yoshikawa H, *et al.* (2022): The impact of a poverty reduction intervention on infant brain activity. Proc Natl Acad Sci USA 119:e2115649119.