THESIS

SOCIOECONOMIC INEQUALITY, AMYGDALA AND VENTRAL STRIATAL CONNECTIVITY, AND AFFECTIVE OUTCOMES IN CHILDREN AND ADOLESCENTS

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ABSTRACT

SOCIOECONOMIC INEQUALITY, AMYGDALA AND VENTRAL STRIATAL CONNECTIVITY, AND AFFECTIVE OUTCOMES IN CHILDREN AND ADOLESCENTS

Socioeconomic disadvantage has been significantly associated with an increased risk for internalizing problems in children and adolescents. The neural mechanisms underlying these associations, however, are not well understood. Differences in connectivity of the amygdala and ventral striatum with the prefrontal cortex (PFC) may play an important role in these mechanisms. The goals of this study were to examine (1) the associations among socioeconomic factors, amygdala and ventral striatal resting-state functional connectivity (rsFC), and emotional outcomes in children and adolescents, (2) sex differences in associations between socioeconomic factors and amygdala and ventral striatal rsFC, and (3) interactions between socioeconomic factors and familial/genetic risk for anxiety/depression in predicting amygdala and ventral striatal rsFC. Participants were typically-developing 3- to 20-year-olds (50% male, $N = 590$) from the Pediatric Imaging, Neurocognition, and Genetics (PING) study (Jernigan et al., 2016). Resting-state fMRI, socioeconomic (family income, parental education), and self-reported positive and negative affect data were collected. Measures of familial and genetic risk for anxiety/depression were family history of anxiety/depression and genome-wide polygenic risk scores for major depressive disorder (PRS-MDD), respectively. Whole-brain, seed-based functional connectivity analyses were conducted with the ventral striatum and the amygdala as seeds. Findings indicated significant interactions between socioeconomic factors and PRS-MDD for amygdala rsFC with the frontopolar cortex. Positive and negative affect were associated with

amygdala and ventral striatum rsFC with various brain regions. Associations between socioeconomic factors and amygdala and ventral striatal rsFC and sex differences were not significant. These findings can be applied to informing the design of more effective prevention and intervention strategies to facilitate healthy emotional development.

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INTRODUCTION

Exposure to socioeconomic disadvantage during childhood is prevalent in the United States (U.S.) and worldwide. It is estimated that approximately 16 million children in the U.S. currently live below the federal poverty line (Semega et al., 2019). Childhood socioeconomic status (SES) is commonly measured using indices such as parental educational attainment, occupational prestige, and family income, but can also be measured in terms of neighborhood indicators of disadvantage (Noble & Giebler, 2020). Socioeconomic disadvantage has been consistently associated with elevated exposure to chronic stressors, including crowding/noise, family turmoil, neighborhood violence, and household chaos and unpredictability (Conger et al., 2000; Evans & Kim, 2013). Socioeconomic disadvantage has been significantly associated with an increased risk for internalizing problems (e.g., anxiety, depression) in children and adolescents (Bradley & Corwyn, 2002; Luthar, 2003; McLaughlin et al., 2012; Ramphal et al., 2020b; Slopen et al., 2010; Strohschein, 2005; Wadsworth et al., 2016). However, the neural mechanisms through which socioeconomic disadvantage may increase risk for internalizing problems in children and adolescents are not well understood.

The neural systems underlying emotion regulation may be centrally involved in the mechanisms through which socioeconomic disadvantage increases risk for internalizing problems. Emotion regulation refers to attempts to alter an emotional experience or expression, including which emotions are experienced and when and how they are experienced (Thompson, 1994). Emotion regulation difficulties increase transdiagnostic risk for various forms of psychopathology (Beauchaine & Zisner, 2017; Heleniak et al., 2016). The amygdala and ventral striatum are key affect-related brain structures, and their connections with prefrontal cortical

(PFC) regions are crucial to emotion regulation. The amygdala is a subcortical structure in the limbic system that has been strongly associated with emotion processing and detecting salient cues in the environment (e.g., threat detection) (Sergerie et al., 2008). The ventral striatum (e.g., nucleus accumbens) is a subcortical structure that is part of the basal ganglia and widely associated with positive affect and the processing of reward (Pizzagalli, 2014). In animal models, chronic stress has pronounced effects on the amygdala and ventral striatum and their connections with PFC regions (Chattarji et al., 2015; Herringa et al., 2013; Kopala-Sibley et al., 2018; Tottenham & Galvan, 2016; Tottenham & Sheridan, 2010). In clinical studies of humans, amygdala and ventral striatal structure and function have been repeatedly associated with depression and anxiety disorders (Davey et al., 2015; Davey et al., 2012; Fischer et al., 2018; Liu et al., 2021; Rakesh et al., 2020; Wang et al., 2020).

In functional magnetic resonance imaging (fMRI) studies, socioeconomic factors have been significantly associated with measures of functional connectivity (Farah, 2017; Herzberg & Gunnar, 2020), which reflect the temporal correlation and activation patterns of anatomically separated brain regions, indicating the level of functional communication between regions (Konrad et al., 2010; van den Heuvel & Hulshoff Pol, 2010). In these studies, socioeconomic factors have been significantly associated with altered amygdala and ventral striatal functional connectivity with PFC regions (see Table 1). Taken together, this work suggests that socioeconomic disadvantage may impact amygdala and ventral striatal connectivity with PFC regions, leading to variability in emotional outcomes in children and adolescents. However, these associations are not well understood. As such, one main goal of this study is to examine the associations among socioeconomic factors, amygdala and ventral striatal resting-state functional connectivity (rsFC), and emotional outcomes in children and adolescents.

Resting-State Functional Connectivity

Studies examining the brain at the network level, including research on functional connectivity, are increasingly viewed as particularly valuable and informative. Indeed, the examination of functional connectivity provides important insights into the organization of brain networks (Friston, 2011; van den Heuvel & Hulshoff Pol, 2010). As measured using fMRI, rsFC relies on the spontaneous activity of the brain when a person is not engaged in a task (Smitha et al., 2017). It reflects the coupling of spontaneous blood oxygen level-dependent (BOLD) signals in discrete brain regions or networks (Wang et al., 2012). Using rsFC methods may be particularly well-matched to the study of higher-order cognitive and emotional functions, which have been observed to rely on connections among distributed regions rather than on single regions in isolation (Gordon et al., 2020).

Amygdala Connectivity and Affective Neural Networks

Connections between the amygdala and PFC regions play an important role in the regulation of negative affect (Cisler et al., 2010; Ochsner et al., 2012). The medial PFC sends projections to the amygdala that modulate amygdala reactivity (Hariri et al., 2003; Hare et al., 2008; Milad & Quirk, 2002; Pezawas et al., 2005). Altered amygdala-mPFC connectivity has been significantly and repeatedly associated with anxiety disorders and major depressive disorder (MDD) (Cisler et al., 2010; Johnstone et al., 2007). At the level of neural networks, the amygdala is also an important node in the salience and emotion network (Menon et al., 2015), which is crucial for integrating sensory, emotional, and cognitive information (Seeley et al., 2007). The amygdala has been found to be functionally connected with nodes of the salience and emotion network including the anterior insula, dorsal ACC, and ventral striatum (Qi et al., 2021).

Ventral Striatal Connectivity and Affective Neural Networks

The ventral striatum is a central node of the reward network (Costumero et al., 2013; Romens et al., 2015), which responds to rewarding stimuli, causes a motivation to obtain rewards, is associated with the anticipation and enjoyment of rewarding stimuli, and is involved in reward-based learning (Forbes & Dahl, 2005; Tottenham & Galvan, 2016). In addition to the ventral striatum, key components of the reward circuit include the ventral pallidum, ACC, mPFC and OFC (Teicher et al., 2016). The ventral striatum is heavily involved in detecting reward cues and reward-related goals (Costumero et al., 2013; Forbes & Dahl, 2012; Tottenham & Galvan, 2016). Connections between the ventral striatum and mPFC and OFC regions are involved in the regulation of reward response (Forbes & Dahl, 2012). Anhedonia, which refers to low positive affect and reduced ability to experience pleasure, has been suggested to be due in part to deficits in reward processing at the neural level (Liu et al., 2021).

Socioeconomic Factors and Amygdala and Ventral Striatal rsFC

Multiple fMRI studies have examined associations between socioeconomic factors and rsFC (see Table 1). These studies have used various methods, including seed or region-of-interest (ROI) based methods.

Amygdala

A subset of the seed-based rsFC studies focused on the amygdala as a seed region in children and adolescents. In these studies, socioeconomic disadvantage has been associated with reduced rsFC between the amygdala and PFC regions. For example, lower household income at age 10 has been significantly associated with reduced rsFC between the amygdala and vmPFC at age 15 (Hanson et al., 2019). Lower parental education (but not family income) in infancy significantly predicted weaker rsFC between the amygdala and dorsal ACC in late childhood (Degeilh et al., 2019). Lower family income-to-needs ratio at preschool-age was significantly

associated with reduced rsFC between the amygdala and superior frontal cortex at school age (Barch et al., 2016). Similarly, 9-year-olds from lower income-to-needs households had stronger negative connectivity between the mOFC and the amygdala (Ip et al., 2021).

Ventral Striatum

Fewer studies have focused on associations between socioeconomic factors and ventral striatal rsFC in children and adolescents. In one study, neighborhood disadvantage was significantly associated with reduced connectivity between the ventral striatum and mPFC in adolescents (Marshall et al., 2018). In this same study, low household income was significantly associated with increased ventral striatum – lateral PFC connectivity (Marshall et al., 2018). In neonates, lower SES was significantly associated with increased rsFC between the striatum and medial PFC and frontopolar PFC (Ramphal et al., 2020b).

Amygdala and Ventral Striatal rsFC and Positive and Negative Affect

Previous research has examined rsFC in MDD and anxiety disorders or in relation to variability in depression and anxiety symptoms. This research has indicated alterations in ventral striatal-PFC and amygdala-PFC connectivity (Cheng et al., 2018; Connolly et al., 2017; Davey et al., 2012; Dillon et al., 2011; Fischer et al., 2018; Furman et al., 2011; Gabbay et al., 2013; Gong et al., 2017; Heller at al., 2009; Hamilton et al., 2011b; Hamm et al., 2014; Kaiser et al., 2015; Kenny et al., 2010; Kim et al., 2011; Liu et al., 2021; Marchand et al., 2013; Philippi et al., 2015; Rakesh et al., 2020; Tahmasian et al., 2013; Wang et al., 2020; Yang et al., 2017). Anxiety and depressive disorders are characterized by altered positive and negative affect, but few studies have examined rsFC in relation to positive and negative affect. Such research would help with understanding whether some of the rsFC differences associated with anxiety or depressive symptoms as a whole may be due to differences in positive or negative affect.

Some prior research has examined associations between positive and negative affect and amygdala and ventral striatal rsFC. Connectivity with both the ventral striatum and amygdala have been found to play roles in positive affect (Qi et al., 2021; Rohr et al., 2013), while amygdala connectivity alone may relate to negative affect (Davey et al., 2015). Frontostriatal connectivity has been found to be negatively correlated with anhedonia, a construct strongly related to positive affect, in typically developing individuals (Wang et al., 2016) and individuals with depression (Liu et al., 2021). However, more research is needed to fully elucidate these associations.

Socioeconomic Factors, Amygdala and Ventral Striatal rsFC, and Emotional Outcomes

Socioeconomic differences in amygdala and ventral striatal rsFC may partially explain socioeconomic disparities in internalizing symptoms. For example, decreased ventral striatum-mPFC rsFC has been found to mediate the association between community disadvantage and high anxiety symptoms (Marshall et al., 2018). Low SES and high anxiety have been found to be associated with reduced rsFC between the amygdala and the vmPFC (Ramphal et al., 2020a). Amygdala–lingual gyrus rsFC has been found to mediate the association between early family income-to-needs ratio and negative mood/depression at school age (Barch et al., 2016). Amygdala and ventral striatal rsFC may also mediate associations between socioeconomic factors and positive and negative affect. However, there are no studies that have examined these associations.

Sex Differences and Amygdala and Ventral Striatal rsFC

Adolescent girls are consistently shown to have a higher risk for internalizing disorders than adolescent boys, and these differences continue through adulthood (Hankin et al., 1998; Oldehinkle & Bouma, 2011). Exposure to high levels of stress, especially during puberty, has

been posited to have stronger effects on the development of mood disorders for adolescent girls than for boys (Hodes & Epperson, 2019). These and other results suggest that sex plays a role in the association between chronic stress exposure and risk for affective disorders, and this trend may extend to associations between socioeconomic factors and brain function (Allen et al., 2011; Hjelmervik et al., 2014; Rubinow & Schmidt, 2019). Thus, in the present study, sex differences in the associations between socioeconomic factors and amygdala and ventral striatal rsFC were examined.

Genetic Risk for Internalizing Disorders and Amygdala and Ventral Striatal rsFC

MDD is well established as being highly heritable (Sullivan et al., 2000). For instance, the likelihood of developing MDD is three times greater in children whose parents have MDD, compared to children whose parents do not have MDD (Weissman et al., 2006, Williamson et al., 2004). It is important to consider familial and genetic risk for internalizing disorders when examining associations between socioeconomic factors and amygdala and ventral striatal rsFC. Familial risk refers to having a parent or other family member (e.g., first-degree relative) with a history of an internalizing disorder. Genetic risk refers to impact of genetics on the probability of an individual having a specific disorder (Baptista et al., 2005). Genetic risk can be measured by creating polygenic risk scores, which are derived based on genome-wide association studies (GWAS) by aggregating the contributions of all known genetic variants associated with a phenotype (e.g., a psychiatric disorder) (Plomin & von Stumm, 2018).

According to differential susceptibility theory, certain genetic profiles may cause an individual to be more susceptible to environmental influences (Bakermans-Kranenburg & van Ijzendoorn, 2007; Ellis et al., 2011). Some individuals are more susceptible than others to a favorable or unfavorable environment, and this may cause certain outcomes to be more likely in

those environments (Boyce & Ellis, 2005). Some individuals are more susceptible than others to a favorable or unfavorable environment, and this may help explain variability in emotional and behavioral health outcomes (Boyce & Ellis, 2005). Differential susceptibility theory contends that both stressful and supportive environments have been part of the human experience throughout evolutionary history and that humans respond adaptively to both contexts (Ellis et al., 2011). In other words, environmental exposures interact with genetic factors to determine individual differences in neurobiological susceptibility to a developmental outcome (Ellis et al., 2011). In terms of statistical analyses, differential susceptibility theory implies the presence of gene-by-environment interactions whereby environmental factors may influence genetically susceptible individuals' outcomes in either direction, positively or negatively (Bakermans-Kranenburg & van Ijzendoorn, 2007).

Familial Risk for Depression

Familial risk for depression has been consistently associated with an increased risk for depression in adolescents and adults (Weissman et al., 2006; Williamson et al., 2004). At the neural level, parental depression has been associated with rsFC in children (Qiu et al., 2015; Cai et al., 2021). Parents with depression may pass down MDD vulnerability to their children via alterations within affect and cognitive control networks (Chai et al., 2016; Clasen et al., 2014; Luking et al., 2011; Shapero et al., 2019).

Polygenic Risk Scores for Major Depressive Disorder (PRS-MDD)

Polygenic risk scores for psychiatric disorders more directly reflect genetic influences on risk for psychiatric disorders. GWAS have identified genetic variants robustly associated with MDD and yielded genome-wide polygenic risk scores for MDD (PRS-MDD) that significantly predict depressive symptoms in independent samples (Howard et al., 2019; Mitchell et al., 2021).

PRS-MDD have been associated with mPFC structure in adults (Holmes et al., 2012) and decreased white matter integrity in adults (Whalley et al., 2013). However, other studies have not found significant associations between PRS-MDD and brain structure (Jansen et al., 2018; Reus et al., 2017) or function (Wang et al., 2017).

Differential susceptibility theory suggests that familial/genetic risk for depression may modulate the impact of socioeconomic factors on amygdala and ventral striatal rsFC. Therefore, gene-by-socioeconomic-factor interactions may be significant in predicting amygdala and ventral striatal rsFC.

Current Study

The goals of this study were to examine (1) the associations among socioeconomic factors, amygdala and ventral striatal rsFC, and emotional outcomes in children and adolescents, (2) sex differences in the associations between socioeconomic factors and amygdala and ventral striatal rsFC, and (3) interactions between socioeconomic factors and familial/genetic risk for internalizing disorders in predicting amygdala and ventral striatal rsFC. Participants were typically-developing children and adolescents (3-20 years of age, 50% male, $N = 580$). Resting-state fMRI, socioeconomic data (family income, parental education), and self-reported positive and negative affect were collected. Family income and parental education were analyzed separately given their differential associations with child development and policy implications (Duncan & Magnuson, 2012). Familial and genetic risk for anxiety/depression were operationalized as family history of anxiety/depression and PRS-MDD, respectively.

I hypothesized that family income and parental education would be associated with amygdala and ventral striatal rsFC with PFC regions. I also hypothesized that there would be an interaction between socioeconomic factors and familial/genetic risk for internalizing disorders in

the prediction of amygdala and ventral striatal rsFC. Specifically, I expected that associations between socioeconomic factors and amygdala and ventral striatal rsFC may be stronger in the context of higher familial/genetic risk for internalizing disorders.

METHODS

Participants

Data used for this study came from the Pediatric Imaging, Neurocognition, and Genetics (PING) study (http://ping.chd.ucsd.edu) (Jernigan et al., 2016). This dataset contains aggregated data collected at 10 different sites in the U.S. Participants were recruited through local postings and outreach activities conducted at universities in Baltimore, Boston, Honolulu, Los Angeles, New Haven, New York, Sacramento, and San Diego. Eligibility criteria required being between 3 and 20 years of age and fluent in English. Exclusionary criteria included: history of neurological disorders or head trauma; pregnancy; diagnosis of autism spectrum disorder, bipolar disorder, schizophrenia, or intellectual disability; premature birth; prenatal exposure to illicit drugs for more than one trimester, and contraindications for MRI. Written informed consent was provided by parents for all participants younger than 18 years of age, and assent was obtained for participants 7 – 17 years of age. Those 18 years of age or older consented themselves (Jernigan et al., 2016). Seven sites (Cornell University; University of California, Davis; Kennedy Krieger Institute; Massachusetts General Hospital; University of California, San Diego; University of Massachusetts Medical School; Yale University) collected fMRI data.

A subsample of PING participants (7-21 years of age; 44% male; $n = 337$) completed web-based, self-report assessments of positive and negative affect from the PhenX Toolkit (Hamilton et al., 2011a; Jernigan et al., 2016; the PhenX RISING network et al., 2014).

Sample Characteristics

Full descriptive statistics for sample characteristics are provided in Table 2. Participants ranged in age from 3 – 21 years (49% male). Parental educational attainment ranged from 6 to 18 years of education, and annual family income ranged from $4,500 to $325,000.

Sample Sizes

In total, 590 participants have fMRI data. These data were cleaned for volume size, and data were excluded from analyses if volume size was less than 50. Eight were removed because of their rsfMRI-volumes, resulting in 582 usable scans. After removing these fMRI scans, 559 participants have fMRI and parental education data; 549 have fMRI and family income data. For positive and negative affect data, 131 participants answered questions about negative and positive affect and completed an fMRI scan. For analyses of sex differences, 574 participants' data are available. For analyses of family history of anxiety/depression and rsFC, 573 participants' data are available. For analyses of PRS-MDD and rsFC, the sample size is smaller (n = 246) due to the restriction of only participants with primarily European ancestry having PRS-MDD data (see below).

Measures

Socioeconomic Factors

Parents reported the level of educational attainment for each parent in the home and the total family income. Parental educational attainment was averaged across parents. Family income was log-transformed to correct for positive skew, consistent with previous work using this dataset (Ursache et al., 2016). Both parental education and family income data were originally collected in bins, which were recoded as the means of the bins for analysis, following from previous work (Noble et al., 2015). Family income and parental education were highly correlated (r = .57, p < .0001).

Positive and Negative Affect

The Positive and Negative Affect Schedule (PANAS) is a comprehensive measure of emotions which includes positive and negative affect subscales (Watson et al., 1988). Participants are asked to respond to 20 questions about their moods, with 10 questions referring to positive affect (e.g., interested) and 10 questions referring to negative affect (e.g., sad). Participants indicate how often they felt these emotions during the past few weeks on a 5-point Likert scale ranging from 0 (very slightly) to 5 (extremely). Higher scores indicate higher levels of positive or negative affect. The PANAS has demonstrated strong psychometric properties (Laurent et al., 1999). In the PING dataset, item scores were averaged to create positive and negative affect total scores. Therefore, total scores ranged from 1 – 5.

Family History of Anxiety/Depression

Participants indicated whether the following family members had a history of anxiety or depression (in relation to the child or adolescent participating in the study): maternal and paternal grandmother/grandfather, biological mother/father, maternal and paternal aunt/uncle, male/female sibling. These responses were summed to create a measure of the total number of family positions with a known history of anxiety or depression (Merz et al., 2018). This variable was log-transformed to correct for a positively skewed distribution.

PRS-MDD

The PING dataset includes 550,000 SNPs genotyped from saliva samples using Illumina Human660W-Quad BeadChip. Computation of polygenic scores followed steps similar to that of our collaborator's previous study (Khundrakpam et al., 2020). Steps included preparation of the data for imputation using the "imputePrepSanger" pipeline (https://hub.docker.com/r/eauforest/imputeprepsanger/) and implemented on CBRAIN (Sherif et

al., 2014) using Human660W-Quad_v1_A-b37-strand chip as reference. The next step involved data imputation with Sanger Imputation Service (the Haplotype Reference Consortium, 2016) using default settings and the Haplotype Reference Consortium, HRC (http://www.haplotype-reference-consortium.org/) as the reference panel. Using Plink 1.9 (Chang et al., 2015), the imputed SNPs were then filtered with the inclusion criteria: SNPs with unique names, only ACTG, and MAF > 0.05. All SNPs that were included had INFO scores R2 > 0.9 with Plink 2.0. Next, using polygenic score software PRSice 2.1.2 (Euesden et al., 2015) additional ambiguous variants were excluded, resulting in 4,696,385 variants being available for polygenic scoring. I filtered individuals with 0.95 loadings to the European principal component (GAF_Europe variable provided with the PING data), resulting in 526 participants. These participants were then used to compute 10 principal components with Plink 1.9. Polygenic scores based on the most recent GWAS focused on MDD were used in analyses (Howard et al., 2019). Data were clumped as per PRSice default settings (clumping distance = 250 kb, threshold $r^2 = 0.1$). The *p*-value selection threshold approach includes only those SNPs with a GWAS association p-value below a certain threshold (e.g., $p < 1 \times 10^{-8}$) in the calculation of PRS-MDD (Choi et al., 2021). These thresholds range from $p < 1 \times 10^{-8}$ to $p < 1$ (all SNPs). Analyses involving PRS-MDD were conducted using different PRS-MDD *p*-value thresholds, and the most predictive one was chosen, following previous studies (Deters et al., 2022; Judd et al., 2020; Merz et al., 2022). In the PING sample, PRS-MDD at $p < 1 \times 10^{-6}$ had the strongest correlation with depression symptoms ($p = .03$). Therefore, this PRS-MDD *p*-value threshold was used for the main analyses of rsFC.

Genetic Ancestry Factor

The PING dataset includes genetic ancestry factors (GAFs), that reflect genetic ancestry across difference racial backgrounds. GAF computation was done via supervised clustering approach implemented in the ADMIXTURE software (Alexander & Lange, 2011). GAFs represents the proportion of ancestral decent for six major populations: African, East Asian, Central Asian, American Indian, Oceanic, and European.

Image Acquisition

For more detailed information about image acquisition in the PING study, see Jernigan et al. (2016). In brief, 3 Tesla (3T) scanners from different manufacturers (General Electric [GE], Siemens, and Philips) were used to acquire neuroimaging data across the seven sites that gathered fMRI data. The Siemens scanner's resting-state fMRI volumes were acquired with repetition time (TR) = 3000 ms, TE = 30 ms, and voxel size = 3 x 3 x 3.5 mm; the Philips scanner's resting-state fMRI volumes were acquired with TR = 2500 ms, TE = 30 ms, and voxel size = 2.67 x 2.67 x 3 mm; and the GE scanner's resting-state fMRI volumes were acquired with TR = 3000 ms, TE = 30 ms, and voxel size = 3 x 3 x 3 mm. The pulse sequence parameters were optimized for equivalence in contrast property. Participants were told to focus on a white cross on a black background while laying still (Darki et al., 2020). To control for the effect of motion, real-time prospective motion correction (PROMO) was used.

Image Preprocessing

For this study, I used the preprocessed fMRI data provided through PING-in-a-Box (Jernigan et al., 2016). Brain extraction was conducted using BET, a spatial smoothing using a Gaussian kernel of 5 mm and a grand-mean intensity normalization of the entire 4D dataset were performed, and finally, high pass temporal filtering was conducted to filter out high-frequency signals which may demonstrate a stronger sensitivity to motion artifacts in resting-state data

(Gaussian-weighted least-squares straight line fitting, with sigma=75.0s). As part of the PING preprocessing pipeline, the volumes were all normalized to standard MNI template after slice timing correction and realignment (Darki et al., 2020). After receiving the pre-processed data, I also performed standard motion correction (3 translation and 3 rotation parameters) and included these as regressors of no interest in my analyses at the single subject level. Following Darki et al (2020), the number of rsfMRI-volumes varied from 19 – 300 volumes across individuals due to different scanning protocols at the different sites. Any individual with volumes less than 50 were removed from analyses, resulting in 8 individuals being removed.

Imaging Analyses

I conducted seed-based functional connectivity analyses, which are used to compute the cross-correlation between the time-series of the seed regions and the rest of the brain (Lv et al., 2018). I did not have *a priori* hypotheses regarding laterality differences, and therefore used bilateral masks for the amygdala and ventral striatum in analyses. Specifically, I used the bilateral striatal ROI mask from the Oxford-GSK-Imanova Striatal Connectivity Atlas (Tziortzi et al., 2011; 2014). I used 3dcalc to only includes the nucleus accumbens, ventral caudate, and ventral putamen (Fareri et al., 2017). I used the bilateral amygdala ROI mask from NeuroVault (Doell et al., 2020). I converted both masks to MNI template. Then, I ran seed-based functional connectivity analyses on the whole brain using each mask individually. This includes calculating the time series for each voxel based on the pattern of activity of that voxel over time. These times series are derived from the BOLD signal, which reflects the change in the oxygenation level of blood in response to changes in neural activity. I then regressed the time series for each voxel in the whole brain onto the time series for the seed. For these analyses, I limited the search space using a whole-brain gray matter mask created on FSL's FAST segmentation toolbox. This

mask was then thresholded at 50% using AFNI. To analyze the resting state data, I used the general linear model (GLM) approach. First, I ran 3dDeconvolve in AFNI to calculate the t-statistics, and beta weights for each voxel in the whole brain at each time point, accounting for motion regressors (Ward, 2002). I then used 3dCalc to transform the beta weights to z scores to determine the percent signal change in each voxel (Cox, 1996). Once the percent signal change of each voxel is known, I ran a regression analysis in AFNI using 3dttest++ to determine if the connectivity for each seed to the rest of the brain differed as a function of each variable of interest. The cluster parameter was that faces or edges much touch, and intensity level was set to 3. I corrected for multiple comparisons using the most updated version of the ClustSim program in AFNI (Cox et al., 2017). In these analyses, the *p*-value threshold for significant clusters was .005, resulting in different minimum cluster sizes for each analysis dependent on the sample sizes (Lieberman & Cunningham, 2009). 3dROIstats in AFNI was used to determine the correlation coefficient between any significant clusters and the seed.

Socioeconomic Factors and Amygdala and Ventral Striatal rsFC

To examine socioeconomic differences in amygdala and ventral striatal rsFC, I ran a regression to determine if the connectivity of the ventral striatum and the amygdala differed as a function of family income or parental education. Covariates were age, sex, GAFs, and site. Minimum cluster size of 106 voxels was required to achieve significance based on output from ClustSim.

Positive and Negative Affect and Amygdala and Ventral Striatal rsFC

Associations of amygdala and ventral striatal rsFC with positive and negative affect were examined using the same approach. Covariates were age, sex, GAFs, and site. ClustSim estimated that a minimum cluster size of 88 voxels was required to achieve significance. If

family income and/or parental education was associated with an rsFC measure that was also associated with positive/negative affect, I would have then examined the mediating role of rsFC. However, there were no main effects of either socioeconomic factor for rsFC (see results section); therefore, no mediation analyses were conducted.

Sex Differences in Associations Between Socioeconomic Factors and Amygdala and Ventral Striatal rsFC

I examined interactions between socioeconomic factors and sex in predicting amygdala and ventral striatal connectivity. I centered sex (-1 = male; 1 = female) and family income/parental education. I then created a centered interaction term by multiplying the centered variables and used this interaction term in regression analyses in AFNI controlling for age, sex-centered, GAFs, site, and family income/parental education-centered. Minimum cluster size of 105 voxels was required to achieve significance based on ClustSim analysis.

Interactions Between Socioeconomic Factors and Genetic/Familial Risk for Internalizing Disorders

I then conducted analyses of interactions between socioeconomic factors and genetic/familial risk for internalizing disorders in the prediction of amygdala and ventral striatal rsFC in AFNI. One set of analyses included family history of anxiety/depression, and the other set of analyses included PRS-MDD. Each variable was centered, and interaction terms were created by multiplying the centered variables. These interaction terms were used in regression analyses in AFNI controlling for age, sex, site, family history/PRS-MDD-centered, and family income/parental education-centered. To minimize the chance of population structure explaining the PRS-MDD results, the extracted 10 first principal components (PC1-10) were used as covariates. Without controlling for those principal components, random differences in population

genomic signature can explain outcomes, if different populations also happen to differ in the outcome (Price et al., 2006). Minimum cluster size of 100 voxels was required to achieve significance for the PRS-MDD-by-socioeconomic-factor interaction and cluster size of 109 was required to achieve significance for the familial-risk-by-socioeconomic factor interaction based on ClustSim for these analyses.

I then probed significant interactions by extracting the rsFC data for the significant clusters. I imported these data into SPSS and conducted simple slopes analyses examining the associations separately at high, middle, and low levels of family income/parental education (Robinson et al., 2013). I also tested whether the simple slopes differ significantly from zero (Cohen et al., 2013).

RESULTS

Descriptive Statistics

Descriptive statistics for positive affect, negative affect, and family history of anxiety/depression can be found in Table 3.

Socioeconomic Factors and Amygdala and Ventral Striatal rsFC

Neither family income nor parental education was significantly associated with amygdala or ventral striatal rsFC. There were no significant interactions between the socioeconomic factors and age or between the socioeconomic factors and sex. Due to the lack of these main effects, no mediation analyses were conducted to examine whether amygdala or ventral striatal rsFC mediated associations between socioeconomic factors and positive and negative affect.

Positive and Negative Affect and Amygdala and Ventral Striatal rsFC

Positive Affect

Positive affect was significantly associated with rsFC between the amygdala (Figure 3) and ventral striatum (Figure 4) and multiple brain regions with cluster sizes larger than 88 voxels (Table 4) (all with $p < .005$).

Negative Affect

Negative affect was significantly associated (all with $p < .005$) with rsFC between the amygdala (Figure 5) and ventral striatum (Figure 6) and multiple brain regions with cluster sizes larger than 88 voxels (Table 5).

Interactions Between Socioeconomic Factors and Familial Risk for Internalizing Disorders

There were no significant interactions between the socioeconomic factors and family history of anxiety/depression for amygdala or ventral striatal rsFC.

Interactions Between Socioeconomic Factors and PRS-MDD

The interaction between parental education and PRS-MDD was significant for amygdala rsFC (Table 6 and Figure 2). There was a significant interaction ($p < .0001$) between PRS-MDD and parental education for amygdala connectivity with the frontopolar cortex (Figure 1). The simple slope analyses revealed a significant negative association between PRS-MDD and amygdala – frontopolar cortex connectivity for low parental education ($\beta = -1.98$, $p < .05$) and a significant positive association between PRS-MDD and amygdala – frontopolar cortex connectivity for high parental education ($\beta = 3.97$, $p < .001$), but no significant association for moderate levels of parental education ($\beta = 1.00$, $p = .17$).

DISCUSSION

The goals of this study were to examine (1) the associations among socioeconomic factors, amygdala and ventral striatal rsFC, and emotional outcomes (positive and negative affect) in children and adolescents, (2) sex differences in associations between socioeconomic factors and amygdala and ventral striatal rsFC, and (3) interactions between socioeconomic factors and familial/genetic risk for anxiety/depression in predicting amygdala and ventral striatal rsFC. Results indicated a significant interaction between parental education and PRS-MDD in the prediction of amygdala rsFC with the frontopolar cortex. In addition, positive and negative affect were associated with amygdala and ventral striatal connectivity with multiple brain regions. There were no significant main effects of socioeconomic factors on amygdala or ventral striatal rsFC and no sex differences in the associations between socioeconomic factors and amygdala and ventral striatal rsFC. These findings are described further and interpreted below.

Socioeconomic Factors and Amygdala and Ventral Striatal rsFC

Neither socioeconomic factor was associated with rsFC of the amygdala or the ventral striatum in terms of main effects, and no significant socioeconomic factor-by-age interactions were detected. These results are inconsistent with some previous research (Barch et al., 2016; Degeilh et al., 2019; Hanson et al., 2019; Ip et al., 2021; Marshall et al., 2018; Ramphal et al., 2020a; Ramphal et al., 2020b), but consistent with a selection of studies (Sripada et al., 2014; Weissman et al., 2018).

Previous studies that have found these associations have used past SES to predict current amygdala rsFC. Studies have used SES from infancy (Degeilh et al., 2019) and preschool (Barch

et al., 2016) to predict amygdala rsFC in childhood. One study also used SES from childhood to predict amygdala rsFC in adolescents (Hanson et al., 2019). And one other study examining SES at age 9 (Ip et al., 2021) found an association between family income-to-needs ratio and amygdala rsFC at age 10. It is possible that concurrent SES may not be as strong as a predictor of amygdala rsFC as early life SES. Alternatively, it is possible that socioeconomic factors have stronger main effects on connectivity in networks more traditionally associated with cognition, as some have suggested (Sripada et al., 2014).

There were also no significant associations between socioeconomic factors and ventral striatal rsFC. One previous study found family income and neighborhood disadvantage to be significantly associated with ventral striatal-mPFC rsFC in children (Marshall et al., 2018). However, 50% of the sample earned below $40,000 while families in the PING dataset were more advantaged on average. One other study examined the impact of SES in infancy using the striatum as a seed region, finding an increase in rsFC to the mPFC at age 2 (Ramphal et al., 2020b). Of note, this study used insurance type to determine SES level, which is not comparable to the measures of SES used in the current study.

Positive and Negative Affect and Amygdala and Ventral Striatal rsFC

Negative and positive affect were both significantly associated with rsFC between the amygdala and ventral striatum and multiple brain regions. Similar to previous work (Rohr et al., 2013; Qi et al., 2021), positive and negative affect were associated with distinct patterns of neural connectivity.

Positive Affect

Positive affect was significantly associated with amygdala and ventral striatal whole brain connectivity to multiple regions. These findings are consistent with previous research in

which connectivity to the amygdala and the ventral striatum has decrease with increased positive affect (Qi et al., 2021; Rohr et al., 2013). Previous research has suggested that these negative correlations may indicate that having more positive affect requires less connectivity resources to detect salient events (Qi et al., 2021). Our findings are consistent with this theory, as amygdala and ventral striatum connectivity to regions associated with salience detection (postcentral gyrus, occipital gyrus, and calcarine fissure) decreased in connectivity as positive affect increased. These findings are supported by research that found that individuals with state positive affect had improved global information processing, but reduced selective visual attention (Rowe et al., 2007).

Negative Affect

Increased negative affect was significantly associated with increased amygdala and ventral striatal whole brain connectivity with multiple brain regions. Studies have found distinct patterns of connectivity in relation to negative affect, with connections between functionally distinct regions either increase or decreasing in connectivity with increased negative affect (Rohr et al., 2013). Increased connectivity between regions associated with vision and somatosensorial has been found to be correlated with more negative affect (Rohr et al., 2013). Decreased connectivity between motor areas and frontal regions as well as increased connectivity between the ventral striatum to motor and somatosensory regions has been found to be correlated with more negative affect (Rohr et al., 2013).

Results from this study showed that increased negative affect was associated with increased and decreased connectivity of the ventral striatum and the amygdala with a number of regions. Amygdala connectivity to regions associated with visual processing (fusiform gyrus, inferior temporal gyrus, cuneus) decreased with more negative affect. The amygdala receives

projections from the inferotemporal cortex to evaluate affective visual stimuli (Pessoa & Adolphs, 2010), and these findings suggest that a decrease in these connections is associated with an increase in negative affect. This may be due to the amygdala receiving less information about the emotional content of visual stimuli, leading to impaired emotional processing and regulation (Pessoa & Adolphs, 2010).

This study also found increased connectivity between the ventral striatum and regions associated with motor movement (BA 6) was associated with a decrease in negative affect. This may reflect the neurological underpinnings of instrumental learning, an adaptive process where an individual learns new motor responses in order to obtain positive outcomes or avoid negative outcomes (Kelley, 2004). This suggests that individuals with an increase in connectivity between these regions associated with instrumental learning will have less negative affect. This idea is supported by research that finds that those with depression have difficulty learning from rewarding stimuli, specifically that they have a reduced approach behavior to the reward (Garbusowa et al., 2022).

These results suggest that positive and negative affect have separate neural underpinnings. Therefore, combining positive and negative affective symptoms should be done with caution when examining the role of amygdala and ventral striatal function in psychiatric disorders. Different regions show differing directionality of significant connectivity with both the amygdala and ventral striatum, and future studies should focus on the complex relationship between connectivity in these regions.

Interactions Between Socioeconomic Factors and PRS-MDD

A significant interaction was found between parental education and PRS-MDD in the prediction of amygdala rsFC with the frontopolar cortex. Connectivity between the amygdala

and frontopolar cortex plays a large role in social cognition and the cognitive control of emotion (Riedel et al., 2019). Research has found that connectivity between these regions is decreased during negative emotion processing in those with depression (Kong et al., 2013; Young et al, 2016). This may reflect a decrease in the frontopolar cortex's inhibitory control over the amygdala, resulting in a delay of extinguishing negative emotions (Kong et al., 2013).

Findings from the current study show a decrease in amygdala – frontopolar cortex rsFC as PRS-MDD increases, consistent with previous findings (Kong et al., 2013; Young et al, 2016), but only for those from low SES backgrounds. Thus, PRS-MDD may impact the ability of the frontopolar cortex to decrease negative emotions brought on by the amygdala, leading to increased negative affect (Walther et al., 2012).

For children from higher SES families, however, PRS-MDD had a positive relationship with the rsFC between these regions. As PRS-MDD increased, so did amygdala – frontopolar cortex connectivity. This means high SES children with a low PRS-MDD had a low connectivity between the amygdala and the frontopolar cortex.

This positive relationship between PRS-MDD and amygdala-frontopolar cortex means that children from higher SES families had increased rsFC between these regions when their PRS-MDD was high. Increased genetic risk for depression has been associated with increased amygdala activity at rest (Hariri et al., 2002; Pezawas et al., 2005). In higher SES children, the amygdala may be overactive due to the high PRS-MDD, but the frontopolar cortex is able to continue to regulate the amygdala regardless of genetic predisposition. It is possible that higher PRS-MDD prompts resilience processes for children in higher SES environments.

Limitations

There are limitations to this study that need to be considered when interpreting the findings. First, because of the cross-sectional, correlational design, no casual inferences can be made based on these findings. Future studies utilizing longitudinal designs are needed. Second, SES is thought to be a distal factor that exerts its effects on brain development through various proximal factors. Further investigations are required to unpack the role of proximal factors such as family stress, prenatal factors, cognitive deprivation, or toxin exposure in potentially mediating SES associations with brain function. Third, another limitation unique to age-related fMRI studies is the use of a single atlas to normalize all subjects to a common template. To be able to investigate brain connectivity development, using the same number of components and a common map for all subjects allowed me to avoid inducing an age-related bias into the analysis (Faghiri et al., 2019). Fourth, while the ClustSim program was used to protect against multiple comparisons in individual imaging analyses, no correction was done for the number of total analyses ran. Finally, analyses using the PANAS had much smaller sample size (n = 131), meaning findings for positive and negative affect will need to be replicated with a larger sample size. Future research should also examine how oversampling for very low SES may impact these findings, as the PING dataset does not have a significantly large number of low SES individuals.

Conclusion

While this study was unable to find a significant main effect of SES on the rsFC of the amygdala or the ventral striatum as I hypothesized, these null results indicate that a more nuanced look at neural development of children in high-risk environments is necessary. Specifically, these findings show that future research aiming to better understand this dynamic will want to take genetic risk into consideration. My findings show that genetic risk for depression may interact with a child's environment to alter their rsFC in areas associated with

the salience network. Similarly, my findings suggest that the neural underpinnings related to negative and positive affect are complex and unique. Overall, my findings contribute to a better understanding of the neural mechanisms underlying socioeconomic differences in risk for internalizing problems. My hope is that these findings, along with future research, can inform policy and practices to facilitate healthy emotional development for individuals across the socioeconomic spectrum.

TABLES

Table 1. Associations between socioeconomic factors and resting-state functional connectivity (rsFC) in children and adolescents

Authors	**Sample size**	**Age range or mean (*M*) and *SD***	**Sex (% and/or *n* female)**	**Socioeconomic measures**	**rsFC measurement approach**	**Seeds or regions-of-interest (ROIs)**	**Results**
Barch et al. (2016)	105	*M* = 9.93 years, *SD* = 1.31, age range = 7-12 years at scan	51% female; *n* = 54	Family income-to-needs ratio	Whole brain rsFC	Hippocampus, amygdala	Lower income-to-needs ratio at preschool age was associated with reduced connectivity between hippocampus and amygdala and a number of regions at school age, including the superior frontal cortex, lingual gyrus, posterior cingulate, and putamen
Brody et al. (2019)	91	Poverty assessed during adolescence ; scanned at age 25	52% female	Poverty (income-to-needs ratio < 1)	Connectivity averaged across all nodes of the central executive and emotion regulation networks	Multiple ROIs in the central executive and emotion regulation networks	More years spent living in poverty was associated with less connectivity in the central executive and emotion regulation networks among young adults who received low supportive parenting but not among those who

Authors	Sample size	Age range or mean (*M*) and *SD*	Sex (% and/or *n* female)	Socioeconomic measures	rsFC measurement approach	Seeds or regions-of-interest (ROIs)	Results
							received high supportive parenting
Dégeilh et al. (2020)	28	SES measured at 7 months of age, fMRI scan at 10 – 11 years old	61% female	Parental education and family income	Whole-brain rsFC	Amygdala, hippocampus	Higher parental education in infancy predicted stronger rsFC between the left and right hippocampi and the right amygdala with the dorsal ACC, and between the left amygdala and bilateral angular gyrus in children.
Gellci et al. (2019)	57	6 – 17 years; *M* = 10.41; *SD* = 2.89	53% female	Household income, neighborhood disadvantage (via zip codes)	Selected ROIs from each network of interest and determined connectivity between each of the ROIs	Multiple ROIs from the salience and emotion network, default mode network, sensorimotor network, visual network, dorsal attention network, fronto-parietal network, language network, and cerebellar network	Children living in more distressed communities had fewer connections between the ACC and the left supramarginal gyrus. Lower household income was associated with lower global efficiency of the ACC but had no effect on the supramarginal gyrus.

Authors	**Sample size**	**Age range or mean (*M*) and *SD***	**Sex (% and/or *n* female)**	**Socioeconomic measures**	**rsFC measurement approach**	**Seeds or regions-of-interest (ROIs)**	**Results**
Gur et al. (2019)	9498	*M* = 14.2; *SD* = 3.7; 8 – 21 years of age	51.7% female	Census-based geocoding variables obtained with the participants' addresses	CBF measured with arterial spin–labeled MRI, resting-state fMRI measures of ReHo and ALFF	Cerebellum, basal ganglia, striatum, limbic system, frontal, parietal, temporal, and occipital lobes	Low SES was associated with reduced ReHo and ALFF, most pronounced in frontoparietal regions
Hanson et al. (2019)	87	*M* = 15.2 years, *SD* = .67 at scan; 14-17 years of age	43.5% female	Family income	Focused on amygdala-vmPFC connectivity	Amygdala	Lower household income at age 10 was related to lower rsFC between the amygdala and vmPFC at age 15
Marshall et al. (2018)	100	6-17 years; *M* = 11.32, *SD* = 2.74	63 females	Family income, neighborhood disadvantage (via zip codes)	Focused on ventral striatal connectivity with PFC regions	Right and left ventral striatum	Neighborhood disadvantage was associated with reduced positive connectivity between the ventral striatum and mPFC; lower household income was associated with increased rsFC between the ventral striatum and the cerebellum, inferior temporal lobe, and lateral PFC.

Authors	Sample size	Age range or mean (*M*) and *SD*	Sex (% and/or *n* female)	Socioeconomic measures	rsFC measurement approach	Seeds or regions-of-interest (ROIs)	Results
Owens et al. (2020)	6543	9-10 years	50% female	Parental education, household income	Network to network connectivity	Default mode network and dorsal attention network	Anticorrelation of the default mode network and the dorsal attention network is negatively correlated with household income
Rakesh et al. (2021a)	7618	9-10 years	50% female	Neighborhood disadvantage (area deprivation index)	Within- and between-network connectivity were calculated for 12 predefined resting-state networks; within-network connectivity reflects the average of the correlation over all pairs of regions within a network	System-level functional connectivity computed for the auditory, cingulo-opercular, cingulo-parietal, dorsal attention, default mode, frontoparietal, retrosplenial temporal, sensorimotor, salience, ventral attention, and visual networks	Higher neighborhood disadvantage was associated with lower rsFC both within and between networks. The strongest associations were with connectivity between the default mode network and both higher-order (e.g., ventral attention network) and sensory systems (e.g., auditory network) and connectivity within higher-order networks including the cingulo-opercular network, ventral attention network, and dorsal attention network.
Rakesh et al., (2021b)	9475	9-10 years		Parental education, income-to-needs ratio,	Within- and between-network connectivity	Auditory, cingulo-opercular,	SES measures had both common and distinct effects on rsFC, with sensory-motor systems and

Authors	Sample size	Age range or mean (*M*) and *SD*	Sex (% and/or *n* female)	Socioeconomic measures	rsFC measurement approach	Seeds or regions-of-interest (ROIs)	Results
				neighborhood disadvantage	were calculated for 12 predefined resting-state networks	cingulo-parietal, dorsal attention, default mode, frontoparietal, retrosplenial temporal, sensorimotor, salience, ventral attention, and visual networks	cognitive networks (e.g., fronto-parietal network) particularly implicated. The association between neighborhood disadvantage and sensorimotor network connectivity was less pronounced in the presence of high income-to-needs ratio.
Ramphal et al. (2020)	112 infants and 46 2-year-olds at follow up	Full term & preterm infants scanned within 4 days of birth or term equivalent age. Follow up at 2 years old	59% female infants; 54% female 2-year-olds	Health Insurance type (Public health considered low SES and private health considered high SES)	Whole-brain functional connectivity maps	Striatum, medial PFC, ventrolateral PFC, dorsal anterior cingulate cortex	Lower SES children had decreased functional connectivity between the right ventrolateral PFC and a nearby cluster in the right ventrolateral PFC, as well as increased functional connectivity between the left striatum and clusters in both the left medial and right frontopolar PFC.
Sripada et al. (2014)	26 adults with a history of childhoo	9 years of age when family income was measured;	44% female	Family income (low-income group compared to middle-income group)	Specifically interested in the default mode network and salience	Posterior cingulate cortex, dorsal anterior cingulate cortex	Childhood poverty was associated with reduced default mode network connectivity but was not significantly associated

Authors	Sample size	Age range or mean (*M*) and *SD*	Sex (% and/or *n* female)	Socioeconomic measures	rsFC measurement approach	Seeds or regions-of-interest (ROIs)	Results
	d poverty; 26 matched controls from middle-income families	24 years of age at scan			network; correlations were examined between average time courses in the seed ROIs and all other voxels of the brain	/ supplementary motor area	with connectivity in the salience network
Su et al. (2021)	76	Mean age at scan = 14.1 years; *SD* = .5; age range = 13-15 years	47.4% female	Parental education and family income at 1 month	ROI to ROI; The time course for each of the seed regions was correlated with each other to generate rsFC values	Left IFG, left anterior STG, left posterior STG, and right anterior STG	Parental education was significantly positively associated with left IFG – left aSTG rsFC and left aSTG – left pSTG rsFC. No association was found between family income and language-related rsFC.
Tooley et al. (2020)	1012	8 – 22 years; *M* = 15.78	55% female	Neighborhood SES, maternal education	Clustering coefficient - a measure of the amount of connectivity	360 ROIs, which collectively comprised the cerebral cortex	High SES participants had stronger positive associations between age and clustering than low SES participants, and this effect was most

Authors	Sample size	Age range or mean (*M*) and *SD*	Sex (% and/or *n* female)	Socioeconomic measures	rsFC measurement approach	Seeds or regions-of-interest (ROIs)	Results
					in a node's immediate neighborhood		pronounced for regions in the limbic, somatomotor, and ventral attention systems.
Weissman et al. (2018)	68	16 years of age at scan	35 females	Family income-to-needs ratio and family income-to-needs ratio change between 10 – 16 years of age	Focused on the default mode network; whole-brain connectivity; also seed-to-seed connectivity	PCC, mPFC	Adolescents from high- and low-income families did not differ in their rsFC. For adolescents in families with lower incomes, connectivity patterns depended on income slope. Low-income adolescents whose income increased demonstrated greater connectivity between the PCC and the mPFC and between the PCC and the right IFG. Increases in income were associated with greater connectivity of the mPFC with the right IFG and the left superior parietal lobule regardless of mean income.

Note. The salience and emotion network is also known as the cingulo-opercular network. The central executive network is also known as the fronto-parietal network.

rsFC Resting state functional connectivity, *ROI* Region of interest, *SES* Socioeconomic status, *fMRI* Functional magnetic resonance imaging, *ReHo* Regional homogeneity, *ALFF* Amplitude of low-frequency fluctuations, *PFC* Prefrontal cortex, *mPFC* Medial prefrontal cortex, *vmPFC* Ventromedial prefrontal cortex, *IFG* Inferior frontal gyrus, *STG* superior temporal gyrus, *aSTG* anterior

superior temporal gyrus, *pSTG* posterior superior temporal gyrus, *ACC*, anterior cingulate cortex, *PCC* posterior cingulate cortex, *CBF* cerebral blood flow

Table 2. Descriptive statistics for sample characteristics (N = 582)

	Mean (SD) or n (%)	**Range**
Age (years)	13.59 (4.93)	3.17 – 21
Sex		
Male	282 (49.1%)	
Female	292 (50.9%)	
Parental education (years)	15.09 (2.27)	6 – 18
Family income (U.S. dollars)	99,444 (78,069)	4,500 - 325,000
Genetic ancestry		
African	0.15 (0.29)	0 – 1
American Indian	0.05 (0.11)	0 – 1
Central Asian	0.04 (0.16)	0 – 1
East Asian	0.09 (0.26)	0 – 1
European	0.68 (0.37)	0 – 1
Oceanic	0.00 (0.01)	0 – 1

Table 3. Descriptive statistics for positive affect, negative affect, and family history of anxiety/depression

	n	**Mean (SD) or n (%)**	**Range**
Positive affect	131	3.68 (0.66)	1.83 – 5
Negative affect	131	1.87 (0.63)	1 – 3.87
Family history of anxiety/depression	573	-0.51 (0.63)	-1 – 1.08

Note. Sample sizes reflect the number of participants with the data specified as well as fMRI data.

Table 4. Whole brain effects of positive affect on amygdala and ventral striatal resting-state functional connectivity (rsFC)

			MNI coordinates for peak voxel				
Region	**BA**	**Voxels**	**X**	**Y**	**Z**	**Mean connectivity (z score)**	**Beta Coefficients**
Amygdala							
Left calcarine fissure	18	425	-2	-80	-8	.008	-.014
Left postcentral gyrus	1	382	-46	-38	62	.008	-.014
Right paracentral lobule	5	320	0	-36	62	.005	-.001
Right thalamus	24	292	-2	28	4	.007	.036
Left supplemental motor area	6	208	0	8	54	.006	-.024
Right superior temporal gyrus	22	138	68	-40	24	.004	-.010
Left cerebellum crus 1	19	116	-32	-78	-20	.012	-.021
Right middle occipital gyrus	19	108	28	-74	26	.004	-.014
Right Inferior Parietal Gyrus	40	97	46	-40	52	.005	-.008
Cerebellar Vermis 3	36	96	0	-36	-6	.004	.007
Right Entorhinal Cortex	28	96	16	-12	-26	.015	-.024
Left Temporal Gyrus	13	94	-42	-10	-6	.004	.005
Ventral striatum							
Right superior frontal gyrus	6	195	18	26	56	.006	-.021
Right cerebellum 4	37	170	-26	-34	-26	.006	.010
Left cerebellum crus 1	19	139	38	-82	-22	.015	-.028
Left lingual gyrus	18	134	0	-60	8	.012	-.015
Left supplemental motor area	6	98	0	6	50	.009	-.021
Left cerebellum 9	37	94	-4	-50	-64	.001	.010
Left calcarine fissure	18	93	-2	-98	-8	.012	-.015

Note. BA, Brodmann's area. MNI, Montreal Neurological Institute.

Table 5. Whole brain effects of negative affect on amygdala and ventral striatal resting-state functional connectivity (rsFC)

			MNI coordinates for peak voxel				
Region	**BA**	**Voxels**	**X**	**Y**	**Z**	**Mean connectivity (z score)**	**Beta Coefficients**
Amygdala							
Left precentral gyrus	4	5523	-24	-28	56	.004	.006
Right angular gyrus	39	1833	58	-66	30	.004	.008
Right parahippocampal gyrus	34	1204	12	-12	-24	.005	.019
Left middle occipital gyrus	18	554	-10	-104	2	.004	.010
Right inferior temporal gyrus	37	491	52	-64	-18	.002	-.022
Left fusiform gyrus	36	343	-28	-26	-28	.008	-.013
Inferior frontal gyrus – opercular part	44	316	56	16	28	.003	-.056
Right cuneus	19	292	10	-90	34	.005	-.012
Left ventral superior temporal sulcus	21	217	-56	-30	-4	.005	.008
Primary auditory cortex	41	182	-68	-50	0	.009	.027
Left hippocampus	28	155	-32	-10	-20	.003	.005
Left inferior temporal gyrus	37	149	-68	-40	24	.002	-.022
Left superior temporal gyrus	13	141	-40	-10	-6	.005	-.006
Left lateral occipital sulcus	18	139	-42	-90	4	.004	.010
Right fusiform gyrus	37	137	32	-44	0	.003	-.022
Left median cingulate	32	121	0	14	36	.004	-.022
Intraparietal sulcus	39	116	32	-46	36	.003	.008
Right posterior TE2	41	111	54	-26	-30	.009	.027
Right cerebellum 4	37	305	10	-38	-8	.007	.012
Right cerebellum 5	19	169	10	-60	-10	.006	.008
Left cerebellum 10	36	162	-2	-18	-43	.009	-.022
Right cerebellum 9	37	139	0	-54	-54	.003	.034
Right cerebellum 8	37	121	10	-58	-62	.003	.019
Right cerebellum 10	36	105	18	-28	-40	.009	-.022
Right postcentral gyrus	1	97	28	-38	74	.006	.006
Left postcentral gyrus	1	95	-56	-20	56	.006	.006
Left rolandic operculum	4	93	-40	-4	12	.004	.006
Ventral striatum							
Left fusiform gyrus	19	3323	-34	-50	0	.009	.005
Left superior frontal language area	6	1541	-6	22	66	.006	.010

Left supplementary motor area and cingulate cortex	6	1508	-10	2	74	.006	. 010
Right insula	13	269	40	-8	-8	.005	.007
Right middle frontal gyrus	8	247	44	12	46	.005	-.023
Left parahippocampal gyrus	36	209	-18	-12	-28	.007	.015
Left supramarginal gyrus	39	200	-66	-46	32	.007	-.009
Left middle temporal gyrus	21	169	-52	-22	-6	.005	.007
Right fusiform gyrus	20	144	46	-22	-34	.005	.017
Left cuneus	18	133	0	-94	18	.006	-.010
Right postcentral gyrus	6	126	-42	10	32	.006	. 010
Left area 8c	13	121	34	-6	10	.005	.007
Left lateral intraparietal area	39	118	28	-50	44	.007	-.009
Right ventral intraparietal Area	39	116	24	-66	58	.007	-.009
Left cerebellum 9	37	746	-6	-50	-66	.005	.019
Left cerebellum 6	19	2613	-32	-66	-18	.009	.020
Left superior frontal gyrus, medial	9	94	8	-48	40	.006	.003
Left subcentral gyrus	9	93	8	-48	40	.007	.015
Left inferior frontal gyrus, opercular part	47	92	54	-50	-32	.005	-.019

Note. BA, Brodmann's area. MNI, Montreal Neurological Institute.

Table 6. Socioeconomic factor-by-PRS-MDD interactions for amygdala resting-state functional connectivity (rsFC)

				MNI coordinates for peak voxel			
PRS-MDD p-value threshold	**Region**	**BA**	**Voxels**	**X**	**Y**	**Z**	**Mean connectivity (z score)**
Parental education-by-PRS-MDD							
$p < 1 \times 10^{-6}$	Frontopolar cortex	10	207	2	66	0	.012

Note. BA, Brodmann's area. MNI, Montreal Neurological Institute. PRS-MDD, Polygenic risk score for major depressive disorder

FIGURES

Figure 1. Polygenic risk score for major depressive disorder (PRS-MDD) by parental education interaction for frontopolar cortex – amygdala resting-state functional connectivity

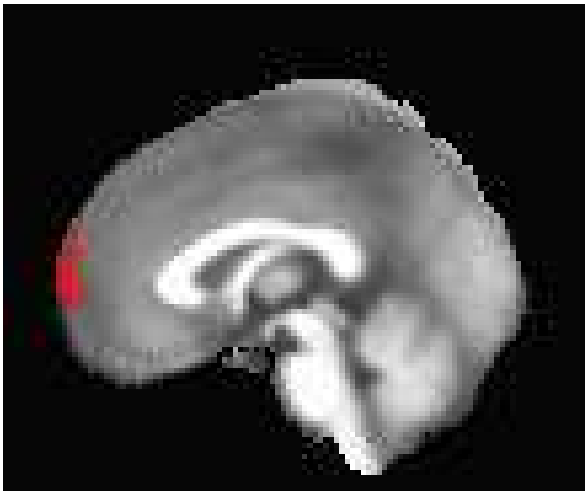

Figure 2. The red area shows an increase in connectivity between the frontopolar cortex and the amygdala correlated with the interaction between parental education and polygenic risk score for major depressive disorder

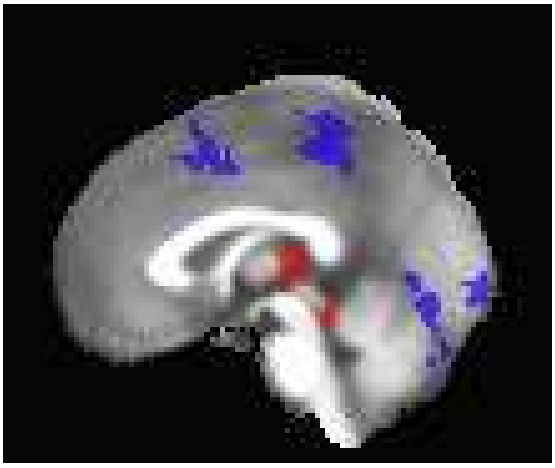

Figure 3. Whole brain connectivity with the amygdala associated with positive affect. The red areas indicate increased activity to the amygdala and the blue areas indicate a decrease in connectivity to the amygdala.

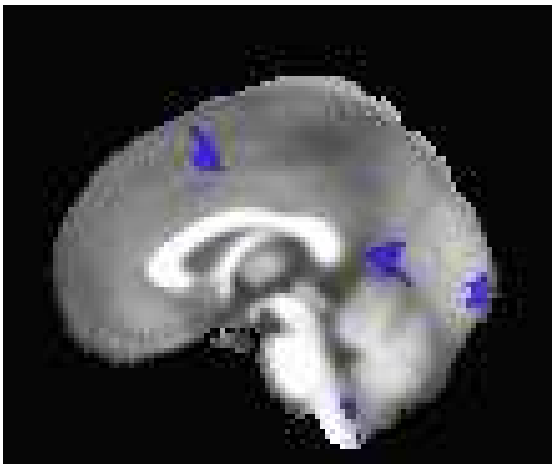

Figure 4. Whole brain connectivity with the ventral striatum associated with positive affect. The blue areas indicate a decrease in connectivity to the ventral striatum.

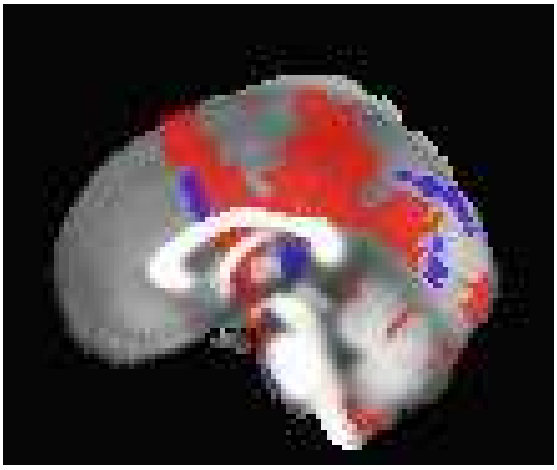

Figure 5. Whole brain connectivity with the amygdala associated with negative affect. The red areas indicate increased activity to the amygdala and the blue areas indicate a decrease in connectivity to the amygdala.

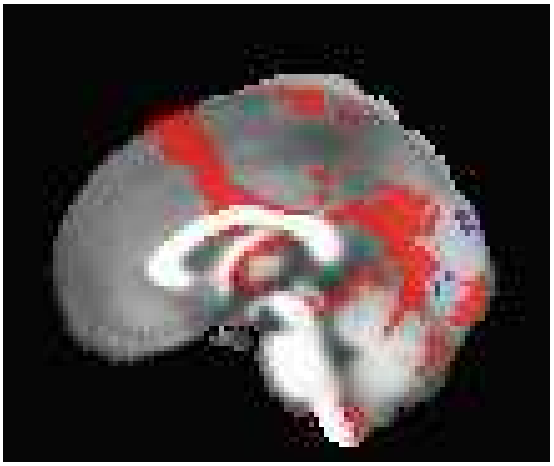

Figure 6. Whole brain connectivity with the ventral striatum associated with negative affect. The red areas indicate increased activity to the ventral striatum and the blue areas indicate a decrease in connectivity to the ventral striatum.

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