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Adolescent girls' neural response to reward mediates the relation between childhood financial disadvantage and depression

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Abstract

Background—Children who experience socioeconomic disadvantage are at heightened risk for developing depression; however, little is known about neurobiological mechanisms underlying this association. Low socioeconomic status (SES) during childhood may confer risk for depression through its stress-related effects on the neural circuitry associated with processing monetary rewards.

Methods—In a prospective study, we examined the relationships among the number of years of household receipt of public assistance from age 5–16 years, neural activation during monetary reward anticipation and receipt at age 16, and depression symptoms at age 16 in 123 girls.

Results—Number of years of household receipt of public assistance was positively associated with heightened response in the medial prefrontal cortex during reward anticipation, and this heightened neural response mediated the relationship between socioeconomic disadvantage and current depression symptoms, controlling for past depression.

Conclusions—Chronic exposure to socioeconomic disadvantage in childhood may alter neural circuitry involved in reward anticipation in adolescence, which in turn may confer risk for depression.

Keywords

socioeconomic status; reward; depression; neural; medial prefrontal cortex

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Introduction

Depression is the leading cause of disability worldwide (World Health Organization, 2012), and depression that begins in youth is particularly detrimental to academic and social functioning (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996). Childhood socioeconomic disadvantage is associated with heightened prevalence of a range of mental health problems throughout the life span (Bradley & Corwyn, 2002), including heightened risk for both first onset and longer duration of depression episodes (Lorant et al., 2003; McLaughlin et al., 2011). The mechanisms underlying the profound and persistent adverse effects of low childhood SES on risk for depression are unclear. However, recent research suggests that early life socioeconomic disadvantage may negatively impact brain structure and function, elevating its relevance for understanding subsequent development of depression (Brito & Noble, 2014; Hackman, Farah, & Meaney, 2010; Kim et al., 2013; Sripada, Swain, Evans, Welsh, & Liberzon, 2014). Improved understanding of a neural mechanism linking early socioeconomic disadvantage to the development of depression in adolescence could inform novel interventions aimed at reducing the burden of both socioeconomic disadvantage and depressive illness.

The physiological stress associated with experiencing socioeconomic disadvantage in childhood may impair functioning of neural reward circuitry, which is central to mood dysfunction in depression (Forbes & Dahl, 2005; Nestler & Carlezon, 2006). Neural reward circuitry includes the striatum, medial prefrontal cortex (mPFC), orbital frontal cortex (OFC), and amygdala. The striatum has been implicated in detecting rewards and representing reward-related goals, while the OFC has been implicated in regulation of reward response (Forbes & Dahl, 2012). The mPFC is thought to play a critical role in regulation of reward response (Etkin, Egner, & Kalish, 2011; Phillips, Drevets, Rauch, & Lane, 2003), as well as processing of self-referential and social information (Amodio & Frith, 2006). Indeed, during monetary reward processing individuals with vs. without depression show reduced activation in striatal areas (Zhang, Chang, Guo, Zhang, & Wang, 2013) and heightened activation in the mPFC (Forbes & Dahl, 2012), which may reflect blunted sensitivity to rewards (via striatum), as well as suppression or difficulty sustaining reward responses through function in regions with a regulatory association with striatum (e.g., mPFC; Haber & Knutson, 2010). Indeed, alterations in neural reward circuitry are related to blunted positive affect in natural environments among individuals with depression (Forbes et al., 2009).

Furthermore, neural reward circuitry undergoes significant changes during adolescence (Galván, 2010; Luciana, Wahlstrom, Porter, & Collins, 2012; Somerville, Jones, & Casey, 2010), at the same time that first onset of depression is most likely to develop (between ages 15–18; Hankin et al., 1998; Paus, Keshavan & Giedd, 2008). Given these changes, maladaptive patterns of neural response to rewards may render adolescents particularly vulnerable to depression (Davey, Yücel, & Allen, 2008). Early chronic exposure to socioeconomic disadvantage may contribute to neural alterations that serve as a vulnerability factor. In the context of normative adolescent changes in neural reward circuitry, this vulnerability could lead to the development of depression.

Childhood socioeconomic disadvantage may disrupt neural reward circuitry and confer risk for depression because it involves chronic exposure to stress, and contributes to stress-related alterations in neurobiology. Socioeconomic disadvantage is a chronically stressful experience, as it inherently involves financial hardship, uncertainty about future financial security, and possible social marginalization (McEwen & Gianaros, 2010). Early life stress is associated with alterations in neural and physiological systems that regulate stress responding (Sripada et al., 2014), and also in neural reward circuitry, including mesolimbic dopamine pathways in rodents and blunted anticipatory response in adult humans' reward circuitry (Pechtel & Pizzagalli, 2011). Chronic stress is particularly likely to contribute to neurobiological dysregulation (Evans & Kim, 2007; McEwen & Gianaros, 2010). Neural activation when anticipating monetary rewards may be particularly altered by childhood socioeconomic disadvantage, because financial hardship may shape beliefs that monetary rewards are infrequent, uncertain, and unpredictable.

Evidence supports this idea, such that adults who reported experiencing low SES during childhood showed blunted activation in anterior cingulate and dorsomedial PFC (dmPFC) when receiving monetary rewards, as well as reduced connectivity between these regions and the striatum, compared to adults with average or high childhood SES (Gianaros et al., 2011). However, no prospective, developmentally sensitive studies to date have examined the relation between childhood SES and neural reward circuitry, leaving open the question of the nature of the temporal association between exposure to childhood poverty and subsequent neural reward functioning. Additionally, we are aware of no studies to date examining the relation among socioeconomic disadvantage, neural reward functioning, and depression.

In the current study, we examined the relation between childhood socioeconomic disadvantage and later function of neural reward circuitry in a prospective design, which eliminated possible recall bias. We focused on adolescence as a developmental period of alterations in reward processing that appear to be particularly relevant for the development of depression. We examined the relation between a direct measure of family income during childhood —a specific, reward-relevant facet of SES—and neural response during anticipation and receipt of monetary rewards. We focused on girls due to their disproportionately high risk for developing depression, particularly in adolescence (Hankin et al., 1998) and hypothesized that adolescent girls with a history of greater exposure to socioeconomic disadvantage would exhibit altered response in neural reward circuitry. Regions of interest included key areas of the dopamine pathway demonstrated to be sensitive to stress or low SES, i.e., the striatum, mPFC, OFC, and amygdala (Pechtel & Pizzagalli, 2011; Gianaros et al., 2011). We predicted that response in these regions would mediate the relation between childhood SES and adolescent depression symptoms after controlling for childhood depression, thus providing preliminary evidence of a mechanism of association between low SES and depression.

Method

Participants

Participants were girls recruited from the Pittsburgh Girls Study – Emotion study (PGS-E; Keenan et al., 2008), a substudy including 232 girls selected from the longitudinal Pittsburgh Girls Study (PGS; Hipwell et al., 2002; Keenan et al., 2010). Initial PGS study recruitment involved oversampling of households in low-income neighborhoods. Half of the PGS-E participants were at high risk for depression based on symptoms at age 8, while the other half were not at risk (see Keenan et al., 2008 for details). Girls and their mothers completed yearly assessments in the PGS and PGS-E from age 5–16, and could complete an fMRI scan for the first time at age 16.

The final sample for the current study—which did not differ in race, public assistance, or depressive symptoms from the full sample— included 123 16-year-old girls. Of the 232 eligible families, 38 (16%) were not seen at age 16 (15 refused participation, 21 could not be scheduled, 2 could not be contacted). Of the remaining 194 participants, 46 girls (24%) were not scanned (8 refused, 8 could not be scheduled, 3 lived out of state, 6 reported claustrophobia, 22 did not meet fMRI safety criteria). Of the remaining 147 girls, 24 girls (15%) were scanned but excluded from analyses due to excessive movement, poor coverage in whole brain or regions of interest, neural abnormalities, scanner malfunction, or poor understanding of the behavioral task. Seventy-three percent of the participants were African-American (n=90) and 27% were European-American (n=33). Written assent/consent was obtained for all participants and parents. Study procedures were approved by the Institutional Review Board of the University of Pittsburgh.

Childhood Socioeconomic Disadvantage

Childhood socioeconomic disadvantage was assessed annually from ages 5–16 by maternal report of whether anyone in the household had received WIC, food stamps, or “welfare or temporary assistance to needy families.” Responses indicating receipt of any form of public assistance were summed to create a count of number of years receiving public assistance (*M*=4.74, *SD*=3.48). Most participants (87%) received public assistance at some point between ages 5 and 16, with 43% receiving assistance at age 5. The mean age of receiving public assistance was 9.97 (SD = 2.68). Nine participants had missing data for one or more time points, leaving 18 missing data points (1% of data); for missing data, we imputed values indicating low risk (i.e., no public assistance). We operationalized socioeconomic disadvantage as receiving public assistance because this measure is directly related to objective financial resources and because criteria for receiving public assistance are consistent across individuals. We chose to measure number of years of public assistance because chronicity of early life stress in particular is associated with neurobiological dysregulation (Evans & Kim, 2007; Kim et al., 2013; McEwen & Gianaros, 2010).

Depression Symptoms

Current symptoms of depression (i.e., past month) were assessed in each year from ages 9–13 and at age 16 using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS) semi-structured diagnostic interview

(Kaufman et al., 1997). K-SADS assessment for ages 14–15 was not available due to a lapse in funding. The K-SADS interview was administered separately to each girl and her mother by research assistants trained and monitored by a licensed clinical psychologist (KK). Each of the nine symptoms of depression was assessed on a three-point scale (1=not present, 2=subthreshold, 3=threshold). All symptoms were assessed regardless of whether the participant endorsed the mood or anhedonia symptoms, in order to provide a continuous measure of depression symptoms. A subset of the girls' interviews (20%) was randomly selected and coded to assess inter-rater reliability, which was strong (intraclass correlation coefficient=0.96 for youth-reported and 0.89 for parent-reported symptoms). A symptom was considered present if it was endorsed at threshold level by either informant. The number of symptoms present at age 16 was summed to create a count of current depression symptoms. The number of symptoms present each year from ages 9–13 was averaged to create a count of mean past depression symptoms. Current and past depression symptom counts were log-transformed prior to analysis due to positive skew. A constant was added to depression symptom count scores prior to log transformation because raw scores included values of zero.

Reward Task

At age 16, participants completed a monetary reward guessing task with a slow event-related design in the scanner. This task reliably elicits activation in key neural reward circuitry during anticipation and receipt of monetary gains among adolescents (Forbes et al., 2009, 2010). For each trial, participants viewed an image of a blank card for 4 sec with instructions to guess, via button press, whether the value of this card would be greater than or less than five. Next, they saw an image of hands shuffling cards and an up or down facing yellow arrow (indicating trial type: potential reward or potential loss) for 6 sec. Participants then saw a screen with the "actual" card value for 500 msec, followed by a screen providing outcome feedback for 500 msec (a green up-facing arrow for win, a red down-facing arrow for loss, or a yellow circle for neutral), followed by a crosshair for 9 sec. Each trial was 20 sec in duration. The task included 24 trials (12 possible-win [i.e., win or neutral outcome], 12 possible-loss [i.e., loss or neutral outcome]; 6 win, 6 loss, and 12 neutral outcomes) administered over a single 8-minute run. Trials were presented in a fixed, pseudorandom order, with outcomes generated to match the predetermined "correct" or "incorrect" guess for each trial. Participants were informed about the potential to win or lose money on each trial. Participants were told that they would receive their winnings after the scan; all participants actually received $10.

MRI Acquisition, Processing, and Analysis

Neuroimaging was conducted on a Siemens 3.0 Tesla Tim Trio scanner. BOLD functional images were acquired using a gradient echo planar imaging sequence that included 39 axial slices (3.1 mm wide) beginning at the cerebral vertex and extending across the entire cerebrum and most of the cerebellum (TR/TE = 2000/28 msec, field of view=20 cm, matrix=64 × 64). Scanning parameters were selected to optimize BOLD signal quality while maximizing whole brain coverage. A 160-slice high-resolution sagittally acquired T1-weighted anatomical image was collected for co-registration and normalization of functional images (TR/TE=2300/2.98 msec, field of view=20 cm, matrix=256 × 240).

Preprocessing and analysis of imaging data were conducted using Statistical Parametric Mapping software (SPM8; http://www.fil.ion.ucl.ac.uk/spm). Anatomical images were auto-segmented in SPM8 prior to analysis. Functional image preprocessing included slice time correction to the middle volume in the time-series, spatial realignment to the first volume in the time series to correct for head motion, spatial normalization to Montreal Neurological Institute (MNI) stereotaxic space using a 12-parameter affine model, and image smoothing using a Gaussian filter set at 6 mm full-width half-maximum to minimize noise and individual differences in gyral anatomy. Voxel-wise signal was ratio-normalized to the whole-brain global mean. In addition, Artifact Detection Toolbox (ART; http://www.nitrc.org/projects/artifact_detect/) was used to identify functional volumes with movement >3 SD from the subject's mean, >.5 mm scan-to-scan translation, or >.01 degrees of scan-to-scan rotation. Participants were included if they had good scan quality, ≥80% coverage in regions of interest, and excessive movement in <25% of volumes. Temporal censoring based on ART output was used to remove motion artifacts (Siegel et al., 2013).

Second-level random effects models were used to estimate neural response to rewards while accounting for scan-to-scan and between-participant variability. For each participant, condition effects were calculated at each voxel using paired *t*-tests for the contrast reward anticipation>baseline and outcome>baseline. Reward anticipation was defined as the 12 potential-win intervals (6 sec each). Reward outcome was defined as the intervals that included number presentation, arrow feedback, and the first 6 sec of cross hair during the 6 win-outcome trials (7 sec each). The last 3 sec of all 24 trials served as baseline.

Analysis of imaging data focused on a single composite ROI mask of reward regions that are related to SES and stress (Pechtel & Pizzagalli, 2001; Gianaros et al., 2011) and engaged by this monetary reward-guessing task (Forbes et al., 2009, 2010): striatum, mPFC, OFC, and amygdala. This composite ROI, which contained 17,357 voxels, was defined using PickAtlas 3.0.3 (http://fmri.wfubmc.edu/software/PickAtlas). The striatum was defined as a sphere with a 20-mm radius, centered on Talairach coordinates of (0,10,−10), and encompassing the bilateral ventral striatum (including nucleus accumbens) and dorsal striatum (Morgan, Olino, McMakin, Ryan, & Forbes, 2013). The mPFC was defined as a sphere with a 25-mm radius, centered on Talairach coordinates x = 0, y = 42, z = 18, and encompassing primarily BA32, dorsal/rostral BA24, and medial regions of BA9 and BA10 (Casement et al., 2014). Spheres were used for the striatal and mPFC ROIs to focus analyses more precisely than BA-based anatomical masks (especially for mPFC, because BA9 and BA10 have large lateral portions). The OFC was defined as BA11 and BA47, and the amygdala was defined using the human PickAtlas label. AlphaSim (http://afni.nimh.nih.gov/afni/) cluster extent threshold was calculated *a priori* to determine the minimum cluster size necessary to maintain a corrected $p<.05$ for the composite mask (threshold=243 voxels). Whole brain analyses were conducted to confirm that our ROI findings emerged when regions were unconstrained.

Regression analyses were performed in SPM8 to determine whether number of years of public assistance was associated with BOLD response in our ROIs. The focus was reward anticipation, but analyses of outcome were included for completeness. Results were saved as a mask, which contained 666 voxels, and a second regression analysis was conducted using

this mask to determine whether level of current depressive symptoms was associated with BOLD response in the clusters related to SES (i.e., conjunction analysis). That is, the mask of SES-related clusters was used for regression analysis of BOLD response on current depressive symptoms. AlphaSim extent threshold for the specific, public assistance-related ROI was 47 voxels. Because participant race was associated with both receipt of public assistance and depression, analyses included race as a covariate. To account for the potential influence of continuity of depression on the association between current depressive symptoms and neural response to reward, all regression analyses included mean past depressive symptoms as a covariate.

Finally, we conducted a formal test of whether BOLD response to reward mediated the relation between public assistance and current depression symptoms. To do so, we created a functional mask from the conjunction analysis described above and extracted mean BOLD response to task (i.e., excluding any predictors or covariates). The functional mask represented clusters that were associated with both number of years of public assistance and current depression. Next, we performed a bootstrap test of mediation using the PROCESS macro for SPSS (Hayes, 2013). This nonparametric resampling technique produced an approximation of the sampling distribution to generate point estimates and confidence intervals for indirect and total effects (Hayes, 2009). Mediation analysis tested whether BOLD response accounted for the relation between public assistance and current depression, adjusting for the two covariates (race and past depression).

Results

Descriptive Analyses

Compared with European-American participants, African-American participants received public assistance for more years, $t(121)=-4.20$, $p<.001$ and endorsed more symptoms than European-American participants for both current depression ($t(121)= -2.73$, $p<.01$) and average past depression ($t(121)= -3.77$, $p<.001$; Table 1). Current depression was higher in those with greater number of years of public assistance, Spearman's rho=.22, $p=.01$.

Socioeconomic Disadvantage and Neural Response to Reward

Number of years of public assistance was associated with greater mPFC response to reward anticipation (666-voxel cluster; primarily BAs 8 and 9 and extending to BAs 6, 10, and 32; peak at MNI coordinates [−2, 54, 20]; $t(119)=2.95$, $p_{corrected}<.05$) (Figure 1). Consistent with these ROI results, whole-brain analyses indicated that number of years of public assistance was associated with increased mPFC response during reward anticipation (see Online Supplementary Table S1). As in previous studies, participants exhibited general response to the task in striatum and mPFC during both anticipation and outcome (see Table S2). Number of years of public assistance was not associated with response to reward anticipation in the striatum, OFC, or amygdala or with neural response to reward receipt (see Table S3 for details on response to receipt).

Socioeconomic Disadvantage, Neural Response to Reward, and Depression

Participants with more current depression symptoms showed greater response to reward anticipation in two clusters within the same region of mPFC that was associated with years of public assistance (a 193-voxel cluster; BA 6, 8, and 9; peak at [4, 54, 36]; $t(119)=3.76$, $p_{corrected}<.05$; a 94-voxel cluster; BA 9, 10, and 32; peak at [6, 52, 6]; $t(119)=3.41$, $p_{corrected}<.05$), (Figure 2). Bootstrap tests indicated that mPFC response during reward anticipation fully mediated the relation between number of years of public assistance and current depression. That is, the direct effect of public assistance on current depression was not significant after including mPFC response as a mediator (ES=0.01, 95% CI: −0.0353, 0.0198, $p=.58$).

Supplemental analyses

To clarify the potential impact of covariates and address potential confounds, we tested the association between number of years of public assistance and neural response during reward anticipation in four subgroups of participants: African Americans ($n=90$), those without a maternal history of depressive disorder ($n=67$), those without a personal history of major depressive disorder ($n=95$), and those with single parents (mode of parent relationship status across ages 5–16; $n=61$). Because inter-rater reliability of parent-reported depressive symptoms was moderate, we also examined whether our main results held for youth-reported depressive symptoms. Results did not change from those reported above (see Appendix S1 for details). We also evaluated whether years of public assistance from ages 5–10 (a potentially sensitive period of neurodevelopment) would be more predictive of neural response to reward than years of public assistance from ages 11–16. Receipt of public assistance from ages 5–10 was associated with neural response, while years of public assistance from 11–16 and mean age of public assistance were not (see Appendix).

Discussion

The current study examined whether childhood socioeconomic disadvantage is associated with altered function of neural reward circuitry in adolescence. Specifically, we examined neural reward regions previously demonstrated to be sensitive to stress or low SES (i.e., striatum, mPFC, OFC, and amygdala). Female adolescents who received public assistance for more years since they were age five showed heightened mPFC response when anticipating monetary rewards at age 16. In turn, heightened mPFC response was associated with more current depression symptoms at age 16. Furthermore, mPFC activation during reward anticipation mediated the relation between early public assistance and current depression. The relation between socioeconomic disadvantage and current depression, as well as mediation of this relationship by mPFC activation, was observed even after adjusting for race and the influence of past depression. Supplemental analyses indicated that the detrimental impact of socioeconomic disadvantage on neural reward processing was evident particularly when it occurred in early childhood (i.e., ages 5–10). Thus, experiencing chronic socioeconomic disadvantage from an early age could alter the brain's response to potential monetary rewards in adolescence and contribute to vulnerability to depression, which lends additional evidence to claims that altered reward circuitry is a mechanism in the development of depression (Forbes & Dahl, 2012; Zhang et al., 2013).

Although our reward task elicited activation in key reward regions (see Table S2), including the striatum and mPFC, socioeconomic disadvantage was only associated with mPFC response when anticipating rewards (see Table S3 for details on outcome results). Specifically, socioeconomic disadvantage was associated with heightened response in dorsal mPFC (BA9 and 10) and pregenual anterior cingulate cortex (pgACC). The dmPFC has been implicated in social processing (Denny, Kober, Wager, & Ochsner, 2012), cognitive control (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004), and automatic affect regulation (Phillips et al., 2003), and the pgACC is postulated to regulate affective responding (Etkin et al, 2011). Consistent with these functions, when disadvantaged adolescents are presented with cues predicting upcoming monetary reward, they may recruit more top-down cognitive resources to support complex and varied reactions, including disappointment, positive affect, dampening of positive affect, and social comparison. Adolescents who experience chronic socioeconomic disadvantage may develop expectations that money is fleeting and unpredictable, or that hopeful anticipation of pleasant outcomes is unfounded. Further, although public assistance was not associated with neural response to reward *outcome*, this association could emerge in larger samples or those with different sociodemographic characteristics. Adolescents who are currently depressed also show heightened mPFC activation during reward anticipation, which has been interpreted as potentially indicative of heightened regulation of striatal reward responding, resulting in blunted reward sensitivity (see Forbes & Dahl, 2012 for review). Although the present study assessed neural function and depression concurrently, it is possible that among adolescents who experience chronic low income, the development of unusual cognitive and affective responses to reward leads to risk of developing depression.

Our findings indicate that it is critical to consider the developmental context of socioeconomic disadvantage. Although mean age of receipt of public assistance was unrelated to mPFC response to reward, the association between number of years of public assistance and mPFC response held for girls who received public assistance in childhood (i.e., age 5–10) but not for those who received public assistance in adolescence (i.e., age 11–15). Early experience of socioeconomic disadvantage could have especially strong consequences for brain development, potentially putting girls on a path toward disrupted reward responding and depression. Additionally, there could a lag between experience of disadvantage and altered neural response to reward.

Along these lines, our findings differ from those of a previous study in healthy adults, in which low childhood SES was associated with blunted mPFC and ACC response to reward (Gianaros et al., 2010). Unlike that study, we examined reward processing in adolescents, assessed SES prospectively and at multiple time points, and focused on a measure directly related to family income. Previous research has emphasized parental education, which may capture different aspects of SES (i.e., financial resources vs. social status). In addition to methodological differences, these discrepant findings could reflect developmental changes in reward processing (Forbes & Dahl, 2012), with altered sensitivity in reward circuitry during adolescence, changing associations between SES and reward response with age, or expectations of financial independence in adulthood.

Family income is associated with numerous other stressors that may be relevant to developmental differences, including objective and perceived social status, income-to-needs ratio, access to resources, exposure to violence and environmental toxins, and family characteristics (e.g., single parent home, parental health problems). Furthermore, families with low income may vary in exposure to these stressors. In the current study, the relation between SES and neural reward processing was not due to maternal depression or single parent status, or moderated by race, suggesting that financial disadvantage may be particularly impactful for neural reward circuitry. However, given considerable evidence that stress influences neural reward circuitry (Pechtel & Pizzagalli, 2011), it is important to prospectively examine the impact of other aspects of SES on reward circuitry to determine specificity of effects.

Despite the current study's design strengths, it had limitations that suggest directions for future studies. First, neural reward response and depression were measured concurrently, and the association of mPFC response with both socioeconomic disadvantage and depression could reflect the correlation between SES and depression. Also, as we were not able to predict first onset or recurrence of depressive episodes, other longitudinal studies should employ multiple neuroimaging and diagnostic time points. Second, SES likely influences a range of cognitive and affective processes, and broadening investigations to other constructs may shed light on the specificity of our results to reward processing. Third, SES is associated with altered response in many brain regions (see Table S1; Hackman et al., 2010; Kim et al., 2013; Sripada et al., 2014), and it will be informative to examine multiple cognitive and affective networks. Fourth, our sample only included female adolescents, who typically have rates of depression twice as high as those of male adolescents (Hankin et al., 1998), and overall level of depressive severity was low. Finally, additional research is needed to determine if the alterations in neural reward circuitry we observed are associated with differences in reward-motivated behavior and subjective experience of reward.

Conclusions

In sum, female adolescents exposed to more chronic socioeconomic disadvantage in childhood show heightened mPFC response when anticipating monetary rewards. This response mediated the relationship between early socioeconomic disadvantage and adolescent depression. Similar alterations in neural reward response have been demonstrated in individuals with diagnoses of depression. As such, the present results provide further support for a neural mechanism linking childhood socioeconomic status with development of depression.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key Points

- Childhood socioeconomic disadvantage is associated with heightened risk for depression.
- The present study is the first to examine the prospective relationship between childhood socioeconomic status and neural response to reward in adolescence.
- Adolescents with more exposure to socioeconomic disadvantage showed heightened response to reward anticipation in the medial prefrontal cortex (mPFC).
- Heightened mPFC response was associated with more concurrent depressive symptoms, and mediated the relationship between childhood socioeconomic disadvantage and adolescent depressive symptoms.
- Disrupted neural reward processing may underlie the pathway from early socioeconomic disadvantage to adolescent depression.

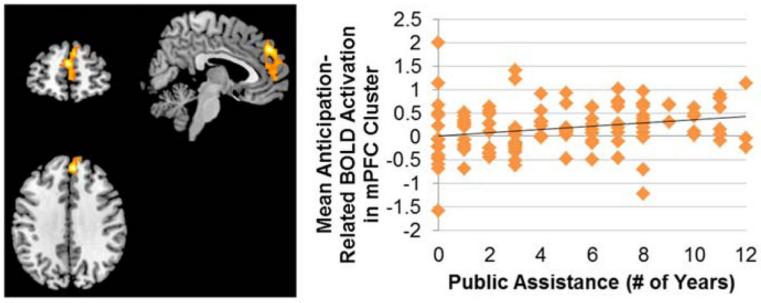

Figure 1.

Association between number of years receiving public assistance and mPFC response during reward anticipation (*beta*=0.03, R^2=0.05).

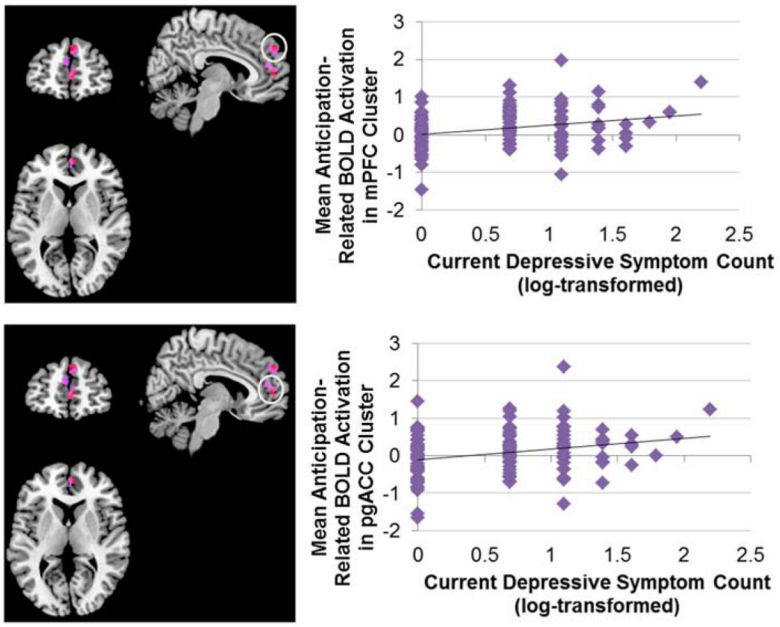

Figure 2.

Association between current (age 16) depression symptom count and dmPFC response during reward anticipation within the previously identified public assistance-related clusters (*beta*=0.24, R^2=0.07) and pgACC (*beta*=0.28, R^2=0.06).

Table 1

Descriptive statistics for receipt of public assistance and depression for African-American (n=90) and European-American (n=33) participants. Public assistance reflects number of years received from ages 5–16. Current depression is a symptom count at age 16. Past depression is the mean symptom count from ages 9–13.

	All		African-American		European-American	
	Mean	**SD (range)**	**Mean**	**SD (range)**	**Mean**	**SD (range)**
Public Assistance	4.74	3.48 (0–12)	5.49	3.34 (0–12)	2.70	3.04 (0–11)
Current Depression Symptoms	1.17	1.35 (0–8)	1.37	1.40 (0–8)	0.64	1.03 (0–4)
Past Depression Symptoms	2.06	1.51 (0–7)	2.35	1.52 (0–7)	1.25	1.17 (0–3.6)

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