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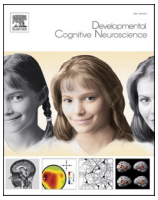

Social neural sensitivity as a susceptibility marker to family context in predicting adolescent externalizing behavior

Caitlin C. Turpyn, Nathan A. Jorgensen, Mitchell J. Prinstein, Kristen A. Lindquist, Eva H. Telzer *

University of North Carolina at Chapel Hill, 235 E. Cameron Avenue, Chapel Hill, NC, 27599-3270, USA

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ABSTRACT

Adolescence represents a period of risk for developing patterns of risk-taking and conduct problems, and the quality of the family environment is one robust predictor of such externalizing behavior. However, family factors may not affect all youth uniformly, and individual differences in neurobiological susceptibility to the family context may moderate its influence. The current study investigated brain-based individual differences in social motivational processing as a susceptibility marker to family conflict in predicting externalizing behavior in early adolescent youth. 163 adolescents (M_{age} = 12.87 years) completed an fMRI scan during which they anticipated social rewards and social punishments. For adolescents with heightened ventral striatum and amygdala blood oxygen-level dependent (BOLD) response during the anticipation of social rewards and heightened ventral striatum BOLD response during the anticipation of social punishments, higher levels of family conflict were associated with greater externalizing behavior. BOLD response when anticipating both social rewards and punishments suggested increased susceptibility to maladaptive family contexts, highlighting the importance of considering adolescent social motivation in positive and negatively valenced contexts.

1. Introduction

Problematic externalizing behavior—including substance use, rule breaking, and aggression—increase dramatically during adolescence; however, these behaviors often cluster within vulnerable individuals and do not represent the behavior of all youth (Blair et al., 2018; Guilamo-Ramos et al., 2005). To account for these individual differences, theory has suggested that some youth may be especially susceptible to salient influences in the social environment including those within the family system, as a function of developing social-affective neural sensitivities (Guyer, 2020; Schriber and Guyer, 2016), creating a context of risk for externalizing behavior. Thus, characterizing patterns of intersecting social and neurobiological function may be critical in identifying youth at greatest vulnerability for problematic trajectories of externalizing behavior.

Although adolescents show developmental increases in sensitivity in social-affective neural systems (Somerville et al., 2010), growing evidence suggests individuals may differ in their sensitivity to social information (Falk et al., 2012; Schriber and Guyer, 2016). Consistent with the *Social Reward/Social Punishment Framework*, individual differences in neural endophenotypes may result in differential responsivity to both positive and negatively valenced social cues, including processing when approaching appetitive incentives (e.g., social reward) and avoiding aversive stimuli (e.g., social punishments, Falk et al., 2012). This framework is in line with meta-analytic evidence indicating that critical subcortical regions, such as the amygdala and ventral striatum, play a role in encoding the salience of both rewards and punishments and thus are thought to be involved in a generalized motivational system (Lindquist et al., 2016; Oldham et al., 2018). Overall, previous evidence suggests that high levels of sensitivity in these neural regions (i.e., amygdala, ventral striatum) may reflect individual differences in social motivational sensitivity.

Previous evidence has also implicated amygdala and ventral striatum activation in youth with externalizing problems. For example, youth with conduct problems (but with low levels of callous/unemotional traits) have been documented to show higher amygdala activation to social threats (Sebastian et al., 2014; Viding et al., 2012), and higher ventral striatum activation to monetary loss (Crowley et al., 2010; Finger et al., 2011). Further, higher ventral striatum activation has been linked with greater sensation seeking (Bjork et al., 2008) and features of

* Corresponding author.
E-mail address: ehtelzer@unc.edu (E.H. Telzer).

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externalizing behavior (Bjork et al., 2010; Galvan et al., 2007; Jager et al., 2013; Qu et al., 2015). Notably, lower ventral striatum activation has also been documented in clinical samples of youth with conduct disorder and especially in those with higher levels of callous/unemotional traits (for review, see Blair et al., 2018). However, it has been argued that atypical neural signatures of this kind (e.g., ventral striatum hypoactivity) may be characteristic of more severe presentations of externalizing psychopathology (Bjork and Pardini, 2015). Taken together, higher sensitivity in these key areas related to social motivation may be particularly relevant for predicting externalizing behavior.

In addition to neurobiological risk factors, social environmental factors, such as the family context, have been consistently linked to externalizing phenomenon. An established body of research indicates that family conflict is a robust predictor of externalizing behavior, especially during early and middle adolescence. Family relationships characterized by high levels of negative affect, hostility, and parent-adolescent conflict are associated with higher rates of substance use (Bray et al., 2001; Shelton et al., 2008; Van Ryzin et al., 2012), aggression, and delinquency (Klahr et al., 2011; Withers et al., 2016). Although poor family functioning or conflict may confer risk for externalizing behavior via multiple pathways, models conceptualizing the development of externalizing or antisocial behavior highlight the role of social learning. In particular, parents represent an important source of social influence, including modeling the attitudes and/or norms that parents imbue, and through reinforcement and punishment of behaviors (Akers, 2011). When the parent-adolescent relationship is conflictual or characterized by high levels of negativity, adolescents may be less likely to internalize protective parental attitudes and norms related to problem behavior, thus conferring greater risk for externalizing patterns of behavior. Moreover, social learning theory also points out that parent-adolescent conflict is often associated with coercive cycles of parent-youth interactions in which problem behaviors are negatively reinforced (Akers and Jensen, 2006; Catalano and Hawkins, 1996; Patterson, 2002). Further, learned problem behaviors (e.g., aggression, oppositionality) are thought to generalize to contexts outside the home and have been associated with increased involvement with deviant peers (Granic and Patterson, 2006; Dishion and Patterson, 2016).

Although family conflict and social motivational neurobiology represents risk factors for externalizing trajectories, recent theory suggests that these factors may not affect youth uniformly (Belsky, 2005; Boyce and Ellis, 2005; Schriber and Guyer, 2016). Individual differences in neurobiological susceptibility to the social environment may moderate the influence of the family context on youth behavior, such that adolescents who are highly tuned to the social environment, as indexed by greater functional brain responses to social rewards and punishments, may fare the worst in maladaptive family contexts and be at highest risk for externalizing problems. In contrast, adolescents who are highly tuned to the social environment but experience adaptive family contexts may be buffered from negative developmental outcomes. In other words, the same neural endophenotype (e.g., high ventral striatum, amygdala response) may confer risk or benefits depending on the social context. Moreover, adolescents exhibiting low functional brain responses to social rewards and punishments may be less susceptible and relatively resilient to their social environment, neither benefitting nor suffering from maladaptive family contexts. Thus, individual differences in social-affective neurobiology may moderate the impact of the family context on developmental outcomes in a "for better or for worse fashion" (Belsky et al., 2007).

Initial evidence has observed that aspects of family functioning interact with neurobiological sensitivity to social exclusion by peers to impact internalizing symptoms (Rudolph et al., 2018; Sequeira et al., 2019) and externalizing symptoms in adolescent youth (Schriber et al., 2018). Further, in one study of young adults, higher levels of amygdala activation to fearful faces represented a marker of susceptibility to social context (i.e., low socioeconomic resources) in predicting antisocial behavior (Gard et al., 2018). For example, heightened affective brain response (i.e., subgenual anterior cingulate cortex) in middle to late adolescent youth was associated with greater externalizing symptoms (i.e., deviance behaviors) in contexts of low family connectedness but associated with lower levels of externalizing symptoms in contexts of high family connectedness (Schriber et al., 2018). Although previous literature has shown main effects connecting youth brain function and externalizing symptoms, this study highlights that the same neural profile may serve as risk or protective factor in predicting externalizing behavior depending on the family context. Notably, no study to date has examined youth's functional brain response to both social rewards and punishments as a marker of susceptibility to the family environment. This may be a particularly important individual difference, as individuals differ in their overall sensitivity to motivationally salient social cues (Falk et al., 2012), and social cues are particularly relevant to adolescents (Crone and Dahl, 2012).

The current study sought to examine early adolescents' social motivational brain function as a marker of susceptibility to negative family contexts in predicting externalizing behavior. We examined adolescents' BOLD response during the anticipation of social rewards and punishments using a social version of the well-validated monetary incentive delay task (Knutson et al., 2000), in which adolescents anticipate and receive social feedback instead of monetary incentives. Given their role in the anticipation of rewards and punishments particularly in adolescents (Crone and Dahl, 2012; Schreuders et al., 2018; Somerville et al., 2010), as well as being key social-affective salience hubs (Lindquist et al., 2016; Oldham et al., 2018), we focused on the amygdala and ventral striatum to identify neurobiologically sensitive youth. We hypothesized that youth experiencing high levels of family conflict would show higher rates of externalizing behavior, but only among adolescents with enhanced BOLD response in amygdala and ventral striatum to social reward and punishment anticipation. Further, we expected that relatively lower levels of BOLD response in amygdala and ventral striatum to social reward and punishment anticipation would attenuate or buffer the association between family conflict and externalizing behavior, representing a pattern of relative resilience to maladaptive family contexts.

2. Materials and method

2.1. Participants

Participants included 173 adolescents (91 females) ages 11–14 (M_{age} = 12.32, SD = .60), who were recruited from three rural public middle schools in the southeast United States. Between 66.7–72.1 % of students in these schools were classified as economically disadvantaged (North Carolina School Report Cards) [NCDPI], 2017), and 69.5 % of students in the district were eligible for free or reduced-price lunch based on district reports. Participants were recruited from a larger study of 873 students in 6th and 7th grade. Participants from the larger study provided interest in being contacted for a future fMRI study. Interested participants were then called and screened on the phone for eligibility (i.e., MRI contraindications) and recruited for the fMRI study within the same academic year as the larger study. We screened 348 families, of whom 110 were ineligible due to learning disabilities, braces, head trauma or other MRI contraindications, and 65 were eligible but did not participate due to scheduling difficulties or no longer interested in participating, resulting in a total sample of 173 adolescents. Thus, of the 238 families contacted and eligible, 73 % participated.

Of the 173 participants who completed the fMRI session, three were excluded from analyses due to not completing the scan, one for excessive motion, two for technical errors, one for an MRI artifact, and three for missing data on family relationship and externalizing behavior questionnaires, leading to a final sample of 163 adolescents (M_{age} = 12.87, range = 11–14 % female = 52.4). Adolescents were from diverse racial and ethnic backgrounds (57 Hispanic/Latinx, 49 White, 37 Black/African-American, 14 multi-racial, 6 other). Families reported low to

middle socioeconomic status with respect to parental reported household income (31.2 % less than $30,000, 33.8 % $30-$60,000, 32.6 % over $60,000). Adolescents and parents gave written assent/consent in accordance with the university's Institutional Review Board.

2.2. Procedures

Adolescents and their primary caregiver attended an fMRI session, during which consent and assent were obtained. Participants completed an fMRI scan that lasted approximately 1.5 h, during which they completed the Social Incentive Delay (SID) task (described below), as well as four other tasks that are not the focus of the current manuscript. Following the scan, participants completed several self-report measures using computer-assisted software in a private room, including measures of family conflict and externalizing behavior, as well as other measures which are not the focus of this manuscript. Adolescents were compensated with a monetary remuneration of $90, small prizes for completing the full scan and staying still (e.g., headphones, candy; worth $20), snacks during the visit, and a meal. Parents were compensated with a monetary remuneration of $50, as well as a meal, compensation for gas, and parking.

2.3. Questionnaire measures

2.3.1. Family conflict

Adolescents completed eight items from the Family Conflict Scale (Ruiz et al., 1998), in which the frequency of parent-adolescent conflict behaviors in the past month were rated (1 = almost never to 5 = almost always). Sample items include, "You and your parents had a serious argument or fight" and "You and your parent ignored each other." This scale has been used in previous studies with adolescent samples to measure parent-adolescent conflict and links with psychosocial and neural development (e.g., Guassi Moreira and Telzer, 2018; Telzer et al., 2014). A total mean score for all items was calculated. This measure had excellent reliability in the present sample (α = .90).

2.3.2. Externalizing behavior

Adolescents completed the externalizing subscale of the Youth Self-Report form of the Child Behavior Checklist (Achenbach and Rescorla, 2001). The CBCL scales are among the most widely used measures of youth behavioral adjustment and psychopathology. The broad-band externalizing subscale is comprised of 32 items on a 3-point scale (0 = not true of me, 1 = somewhat or sometimes true of me, 2 = true or often true of me) assessing rule-breaking behavior and aggressive behavior. T-scores were calculated based on normative references samples. The present sample ranged from 34 to 72 (M = 49.94, SD = 9.27). Approximately 7% of participants reported T-scores of 65 and above (11 participants falling in the borderline clinical range, 1 participant falling in the clinical range). This scale demonstrated excellent reliability in the present sample (α = .86).

2.4. Social incentive delay task

Participants completed the Social Incentive Delay Task (Cremers et al., 2015) while undergoing fMRI to measure neural responses when anticipating social rewards and punishments. The SID is modified from the widely used Monetary Incentive Delay Task (Knutson et al., 2000). Each trial of the SID began with a cue that signaled whether the anticipated feedback would be a reward, punishment, or neutral (500 ms). The cue was a different shape for each condition. The cue was followed by a jittered crosshair (between .48 and 3.9 s, M = 2.0 s), which was followed by the target (a white square; 300 ms), at which point participants were instructed to press a button as quickly as possible. The display of social feedback (1450 ms) was dependent on the trial type and participants' reaction time. In the reward condition, a hit (i.e., fast enough response) resulted in receiving a social reward (i.e., happy face), and a miss (i.e., too slow response) resulted in receiving a blurred face. During the punishment condition, a hit resulted in the avoidance of a social punishment (i.e., blurred face) and a miss resulted in receiving a social punishment (i.e., angry face). Both hits and misses were followed by a blurred face in the neutral condition. After the feedback, another jittered crosshair (between .51 and 4.2 s, M = 2.3 s) was presented before the next trial began. Trials were presented in an event-related design, with reward, punishment, and neutral conditions randomly ordered. Participants completed two rounds of the task, totaling 116 trials (48 reward, 48 punishment, 20 neutral).

To prevent a ceiling or floor performance effect and ensure participants performed roughly at 50 % accuracy so that they received a relatively equal amount of positive and negative feedback, the time required for a successful hit was adaptive, starting at .30 s for the first trial and adding or subtracting .02 s after a miss or hit, respectively, with an upper bound of .50 s and a lower bound of .16 s. In order to make the task motivationally salient, age-matched adolescent faces posing emotional facial expressions were utilized as rewards and punishments. The faces were photographs of ethnically diverse male and female adolescents (24 faces, 12 female) taken from the National Institute of Mental Health Child Emotional Faces Picture Set (NIMH-ChEFS). Participants were trained on the meaning of each cue and completed 12 practice trials prior to entering the scanner. Three participants only had one round of usable fMRI data from the task (due to early exit from scanner or technical issues), but were included in analyses because they met a priori requirements for the number of trials needed per condition (8 hits, or above a 15 % hit rate).

2.5. fMRI data acquisition and preprocessing

Imaging data were collected using a 3 T Siemens Prisma MRI scanner. The SID was presented on a computer screen and projected through a mirror. A high-resolution structural T2*-weighted echo-planar imaging (EPI) volume (TR =2000 ms; TE =25 ms; matrix = 92 × 92; FOV = 230 mm; 37 slices; slice thickness = 3 mm; voxel size 2.5 × 2.5 × 3 mm3) was acquired coplanar with a T2*-weighted structural matched-bandwidth (MBW), high-resolution, anatomical scan (TR =5700 ms; TE =65 ms; matrix = 192 × 192; FOV = 230 mm; 38 slices; slice thickness = 3 mm). In addition, a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR =2400 ms; TE =2.22 ms; matrix = 256 × 256; FOV = 256 mm; sagittal plane; slice thickness = 0.8 mm; 208 slices) was acquired. The orientation for the EPI and MBW scans was oblique axial to maximize brain coverage and to reduce noise. Preprocessing was conducted using FSL (FMRIB's Software Library, version 6.0; www.fmrib.ox.ac.uk/fsl) and included the following steps: Skull stripping using BET (Smith, 2002); motion correction with MCFLIRT (Jenkinson et al., 2002); spatial smoothing with Gaussian kernel of full width at half maximum (FWHM) 6 mm; high-pass temporal filtering with a filter width of 128 s (Gaussian-weighted least-squares straight line fitting, with sigma = 64.0 s); grand-mean intensity normalization of the entire 4D dataset by a single multiplicative factor; and individual level ICA denoising for motion and physiological noise using MELODIC (version 3.15; Beckmann and Smith, 2004), combined with an automated signal classifier (Tohka et al., 2008; Neyman-Pearson threshold = .30). For the spatial normalization, the EPI data were registered to the T1 image with a linear transformation, followed by a white-matter boundary based transformation (BBR; Greve and Fischl, 2009) using FLIRT, linear and non-linear transformations to standard Montreal Neurological Institute (MNI) 2-mm brain were performed using Advanced Neuroimaging Tools (ANTs; Avants et al., 2011), and then spatial normalization of the EPI image to the MNI.

2.6. fMRI data analysis

Individual level, fixed-effects analyses were estimated using the general linear model convolved with a canonical hemodynamic

response function in SPM8. The task was modeled as event-related with eight conditions, including three anticipation conditions (reward, punishment, neutral), two outcome conditions for both reward (hit, miss) and punishment (hit, miss), and one outcome condition for neutral. Anticipation conditions were modeled as the onset of the cue and a duration of 0 s. Six motion parameters were modeled as regressors of no interest. Using the parameter estimates from the GLM, linear contrast images comparing each of the conditions of interest were calculated for each individual. The two primary contrasts of interest for this study were reward anticipation vs. neutral anticipation and punishment anticipation vs. neutral anticipation.

We employed a region-of-interest (ROI) approach of the bilateral ventral striatum and bilateral amygdala. The ventral striatum was anatomically defined from the Oxford-GSK-Imanova structural striatal atlas (Tziortzi et al., 2011), and the amygdala was anatomically defined from the Harvard-Oxford subcortical structure atlas, both included within the FSL software package (FMRIB, Oxford, UK). We chose to utilize bilateral masks because we had no a priori hypotheses regarding laterality. Using these masks, parameter estimates of signal intensity from the primary contrasts of interest were extracted. These parameter estimates were then used in subsequent regression analyses to test our primary hypotheses.

2.7. Analysis plan

Moderation analyses were conducted in SPSS (version 25, IBM) using the PROCESS macro (Hayes, 2013). We conducted two linear regression models testing the moderating effect of **BOLD response** during contrasts of interest (reward anticipation, punishment anticipation,) for each ROI (ventral striatum, amygdala) on the relationship between family conflict and adolescent externalizing symptoms. Bootstrap bias-corrected confidence intervals (95 %) were estimated, where nonzero overlapping confidence intervals indicated a significant effect. For all primary moderation analyses, predictor and moderator variables were mean-centered. Interaction effects were probed through the Johnson-Neyman technique (Bauer and Curran, 2005; Hayes and Matthes, 2009) and by examining simple slopes using small multiples (created with the R-based interActive data visualization tool; McCabe et al., 2018).

3. Results

3.1. Bivariate correlations

Table 1 provides correlation coefficients for primary study variables. Family conflict was significantly correlated with adolescent externalizing behaviors in expected directions, with greater conflict associated with greater externalizing behavior. Demographic variables, including adolescent age and biological sex were not significantly correlated with family conflict or adolescent externalizing behavior.

Table 1
Correlations Among Primary Study Variables.

Variable	1.	2.	3.	4.	5.	6.
1. Family Conflict	1	−.03	−.10	.02	.05	.44*
2. VS Reward Ant		1	.59**	.81***	.51***	.001
3. AM Reward Ant			1	.45***	.76***	−.14
4. VS Punish Ant				1	.56***	−.01
5. AM Punish Ant					1	−.06
6. Externalizing Behavior (T-score)						1

Note: Ant = anticipation, Punish = punishment, VS = ventral striatum, AM = amygdala.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

3.2. Moderation by brain function

3.2.1. Social reward anticipation

For social reward anticipation, linear regressions revealed a significant family conflict X ventral striatum BOLD response interaction ($B = 5.33$, $SE = 1.34$, $p < .0001$, 95 % CI [2.69, 7.97], See Table 2). Simple slopes indicated that at average to high levels of ventral striatum BOLD response during social reward anticipation, greater family conflict predicted higher levels of externalizing behavior (see Fig. 1A). Johnson-Neyman significance regions indicated that the simple slope of family conflict on adolescent externalizing behavior was significant above -0.68 units of mean ventral striatum BOLD response (85.37 % of observations were within this region of significance). Thus, at low levels of ventral striatum **BOLD response** during the anticipation of social rewards, family conflict did not significantly predict adolescent externalizing behavior.

Further, linear regressions indicated a significant family conflict X amygdala BOLD response interaction ($B = 3.13$, $SE = 1.43$, $p < .05$, 95 % CI [0.30, 5.96]). Similar to patterns of ventral striatum response, simple slopes indicated that at higher levels of amygdala BOLD response during social reward anticipation, greater family conflict predicted higher levels of externalizing behavior (see Fig. 1B). Johnson-Neyman significance regions indicated that the simple slope was significant above −0.92 units of mean amygdala BOLD response, with 92.68 % of observations falling within this region. Again, at low levels of amygdala BOLD response, family conflict did not significantly predict adolescent externalizing behavior.

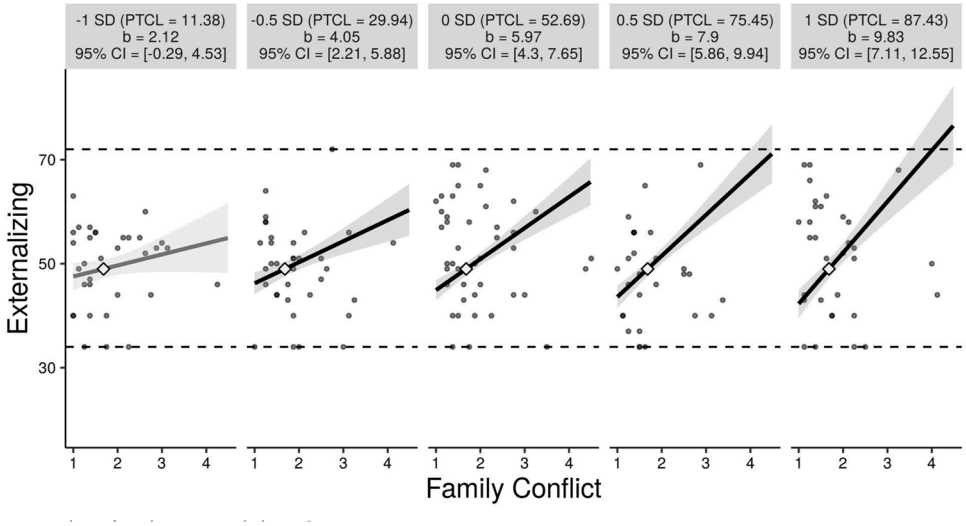
3.2.2. Social punishment anticipation

For social punishment anticipation, linear regressions also revealed a significant family conflict X ventral striatum BOLD response interaction ($B = 5.76$, $SE = 1.63$, $p < .0001$, 95 % CI [2.54, 8.98]). Decomposition of this interaction effect via simple slopes revealed a similar pattern of findings to social reward anticipation, such that at higher levels of ventral striatum BOLD response to punishment anticipation, family conflict was associated with externalizing behavior (Fig. 2). Simple slopes were significant above -0.59 units of mean ventral striatum BOLD response, with 84.15 % of observations falling within this region. Amygdala BOLD response did not moderate the association between family conflict and externalizing.

Table 2
Region of Interest Regression Analyses.

	B	SE	95 % CI	ΔR^2
Social Reward Anticipation				
Ventral Striatum				
Family conflict	5.87***	.84	[4.21, 7.53]	
VS	1.02	0.88	[-0.72, 2.76]	
Family conflict X VS	5.33***	1.34	[2.69, 7.97]	.07***
Amygdala				
Family conflict	5.69***	0.87	[3.97, 7.43]	
AM	−1.10	0.91	[-2.90, 0.70]	
Family conflict X AM	3.13*	1.43	[0.30, 5.96]	.03*
Social Punishment Anticipation				
Ventral Striatum				
Family conflict	5.73***	0.85	[4.06, 7.41]	
VS	0.67	0.96	[-1.23,2.57]	
Family conflict X VS	5.76***	1.63	[2.54, 8.98]	.06***
Amygdala				
Family conflict	5.69***	0.87	[3.97, 7.42]	
AM	−0.79	1.03	[-2.82, 1.24]	
Family conflict X AM	2.68	1.84	[-0.94, 6.31]	.01

Note: VS = ventral striatum, AM = amygdala, CI = Confidence interval.
* $p < .05$. **$p < .01$.
*** $p < .001$.



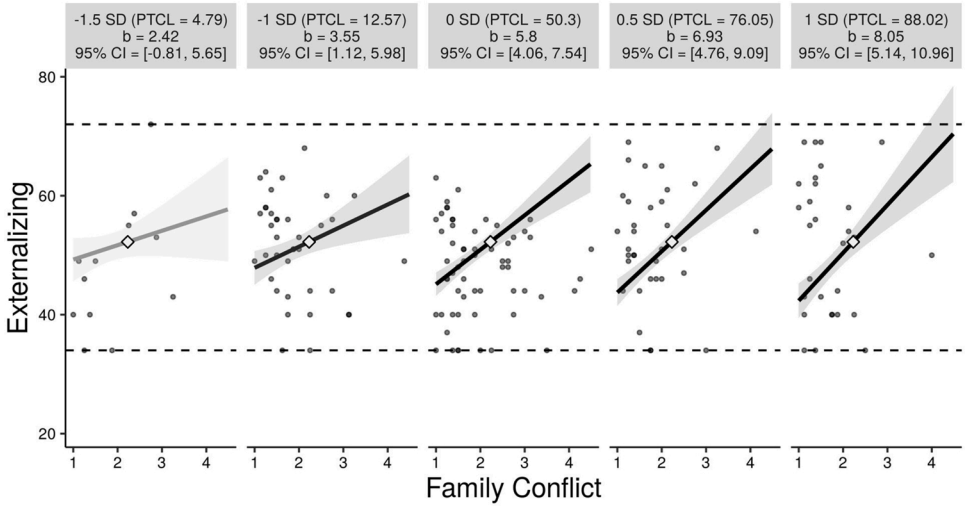


Fig. 1. Moderation by Social Reward Anticipation versus Neutral Anticipation.
A. Moderation by ventral striatum BOLD response.
B. Moderation by amygdala BOLD response.
Note: SD = standard deviation, CI = confidence interval, PTCL = percentile.

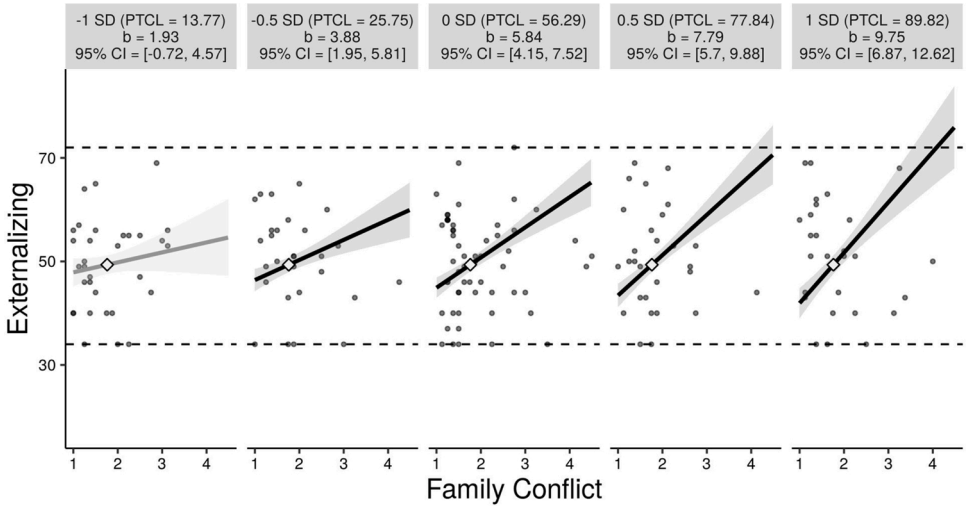


Fig. 2. Moderation by Social Punishment Anticipation versus Neutral Anticipation.
A. Moderation by ventral striatum BOLD response.
Note: SD = standard deviation, CI = confidence interval, PTCL = percentile.

4. Discussion

Adolescence represents a period of risk for risk taking and conduct problems, and the quality of the parent-adolescent relationship is one robust predictor of such externalizing behavior. However, theory suggests that the family environment may not affect all youth uniformly, as individual differences in susceptibility to the social environment may moderate its influence (Belsky, 2005; Boyce and Ellis, 2005). The current study investigated the moderating role of social motivational brain function as an index of such susceptibility to family influence. Results indicated that brain-based individual differences in social motivational processing reflect a susceptibility marker to the social environment in predicting externalizing behavior in early adolescent youth. Higher BOLD response when anticipating both social rewards and punishments suggested increased susceptibility to family conflict, highlighting the importance of considering adolescent social motivation in positive and negatively valenced contexts more comprehensively.

4.1. Social reward anticipation

Increasing levels of ventral striatum and amygdala BOLD response to socially rewarding incentives marked a pattern of susceptibility to family context, as family conflict was associated with higher rates of externalizing behavior for adolescents with greater brain response during the anticipation of social rewards. That is, adolescents experiencing both high levels of family conflict and exhibiting high BOLD response to anticipation of social rewards evinced the highest levels of externalizing behavior. These results suggest adolescents who are highly tuned to the social environment may fare the worst in harsher family contexts and be at highest risk for problematic patterns of externalizing behavior. In contrast, when adolescents exhibited low levels of ventral striatum and amygdala response during social reward anticipation, family conflict did not predict externalizing behavior, suggesting that these adolescents may be resilient to maladaptive family contexts in part as a function of their social-affective neural processing. Notably, only a small subset of youth showed this pattern of resilience (<15 %), suggesting that moderate levels of ventral striatum and amygdala BOLD response and its related susceptibility to family conflict may be relatively normative. Thus, for most youth, family conflict represented a risk factor for externalizing behavior, one that may have greater negative consequences when social motivational neural sensitivity is heightened.

Adolescents with higher levels of social motivational neural sensitivity, as indexed by brain activation when anticipating social rewards, may be particularly influenced by maladaptive social learning in the family context. Indeed, family conflict was robustly related to adolescent externalizing behavior in the current study. Previous evidence and theory suggest this may occur due to processes related to maladaptive differential reinforcement patterns in the family context, a lack of protective norms and attitudes in conflictual family contexts, and thus greater orientation towards potentially deviant peers (Akers, 2011; Akers and Jensen, 2006; Catalano and Hawkins, 1996). That is, for susceptible adolescents, the influence of these factors may be magnified due to higher levels of social salience or motivation. For example, parent-adolescent relationships characterized by high conflict may expose adolescents to an environment in which youth receive less positive reinforcement from parents and family experiences. Especially for susceptible adolescents high in social reward motivation, adolescents may seek out rewards or reinforcing social bonds outside of the home in order to compensate for the lack of rewarding experiences in the family context (Catalano and Hawkins, 1996). In other words, adolescents may tune their behavior to maladaptive rewarding contexts in response to negative family interactions and engage in greater levels of risk-taking or problem behavior.

Interestingly with respect to direct associations, family conflict was not significantly associated with youth brain function in the present study. Previous studies have shown direct associations between aspects of the parent-adolescent relationship and adolescent affective brain function (e.g., Tan et al., 2020). The present study's findings suggest that high levels of social motivation as indexed by youth brain function may serve to magnify the association between family conflict and externalizing behaviors. It may be that in this community sample, normative variations in family conflict, in contrast to more extreme variations in family functioning such as maltreatment or very harsh parenting, may not robustly influence youth brain function alone. Further, family conflict was assessed concurrently. It may be that longstanding patterns of parent-child relationship quality or attachment patterns more powerfully predict adolescent brain function in contrast to potentially changing levels of family conflict in the early adolescent period, and thus longitudinal studies of differential susceptibility are needed.

It is notable that both ventral striatum and amygdala BOLD response during the anticipation of socially rewarding incentives moderated the influence of family conflict on externalizing behavior. While ventral striatum response has been robustly implicated in reward processing across animal and human models (Daniel and Pollmann, 2014), the amygdala's role in social reward processing is less commonly explored, as its involvement in processing negatively valenced stimuli has predominated in the literature (LeDoux, 2003). However, evidence from rat and nonhuman primate studies, human lesion studies, and human functional neuroimaging suggests the amygdala's role in processing the reward value of appetitive stimuli (Baxter and Murray, 2002; O'Neill et al., 2018). Thus, in addition to classically conceptualized reward processing regions (e.g., ventral striatum), the amygdala may also play an important part in encoding the motivational salience of socially rewarding incentives and may represent a critical susceptibility marker for adolescent youth in this positively valenced context.

Further, while amygdala and ventral striatum BOLD response moderated the association between family conflict and adolescent externalizing behaviors, the present study did not reveal main effects of adolescent brain response in predicting externalizing outcomes. This null finding is in contrast with some previous literature implicating atypical ventral striatum response during reward processing in youth with conduct disorder or externalizing psychopathology (Blair et al., 2018). This null finding may be due to the present study's community sample, which largely showed normative variations in externalizing behavior. Thus, it may be that atypical neural responses may figure more prominently or independently when youth possess clinically elevated presentations of externalizing symptoms; however, for more typically developing youth, the confluence of social motivational sensitivity and negative social environment is predictive of normative variations in externalizing behavior. Future longitudinal studies are needed to assess these relations, especially as the prevalence of externalizing risk behaviors increase over time in older youth.

4.2. Social punishment anticipation

We also found that family conflict was associated with higher levels of externalizing behavior among youth showing moderate to high levels of ventral striatum BOLD response during the anticipation of social punishments. Consistent with social reward anticipation, heightened ventral striatum response reflected a pattern of neurobiological susceptibility to social context, reinforcing theory suggesting that some individuals may be more sensitive to social influence via both social approach and avoidance motivations (Falk et al., 2012). Moreover, for a subset of adolescents showing low levels of ventral striatum BOLD response to social punishment anticipation, family conflict was not significantly related to externalizing behavior. For these youth, lower motivation to avoid social punishments may buffer adolescents from potential negative effects of maladaptive family contexts. Again, similar to results with respect to social reward anticipation, only a relatively small subset of adolescents showed this lack of association with externalizing behavior, suggesting that this pattern of relative resilience may be less typical.

Adolescents with higher social punishment sensitivity may be susceptible to family conflict via multiple pathways. Broadly, family conflict, which may inherently include more frequent social punishments (e.g., anger, harsh parent-adolescent interaction, punitive discipline practices), is predictive of higher levels of externalizing behavior. In conflictual parent-adolescent relationships, parents may model more angry or aggressive response styles and contingencies, which may lead to poorer emotion regulation or even aggression in youth (Morris et al., 2007). Youth with higher social punishment sensitivity may be particularly prone to internalizing or attuned to such parental cues and negative interactions in the family context, including parent-adolescent conflict, thus exacerbating their negative consequences on behavior.

It is notable that while ventral striatum response to social punishment anticipation differentiated susceptible youth, amygdala response during social punishment anticipation did not. This is especially noteworthy given that ventral striatum and amygdala response during the anticipation of social punishments were moderately correlated across the present sample. While results broadly support the notion of social motivational sensitivity in negative and positively valenced contexts (both social reward and punishment anticipation) as a susceptibility index, this finding may suggest that the ventral striatum is particularly important in avoidance motivations. However, previous evidence showed that amygdala activation to negatively valenced social stimuli (i.e., fearful faces) moderated the association between low socioeconomic resources and antisocial behavior in young adults in a manner consistent with differential susceptibility (Gard et al., 2018). It is unclear if these discrepant findings are due to sample characteristics, unique interactive effects with family functioning, or task design. Thus, further research is certainly needed to replicate the present study's finding with respect to BOLD response in this social context with respect to family functioning.

4.3. Limitations and future directions

Several limitations of the current study should be acknowledged. First, the current study does not take into account the longitudinal relations between family functioning, externalizing behavior, and adolescent BOLD response to social information. Especially given evidence suggesting reciprocal relations between parent-adolescent relationships and youth behavior, future research assessing their mutual influence across time and the pathways by which neural functioning may modulate these relations in needed. For instance, it is possible that youth high in social motivational sensitivity may more highly elicit negative family conflict interactions over time, which in turn may lead to greater externalizing behaviors. Relatedly, the present study focused on susceptibility with respect to the family context specifically. However, other important sources of social influence are critical in understanding the development of externalizing behavior in adolescents, especially including peer relationships (e.g., Telzer, 2016; Telzer et al., 2020;). It will be important for future studies to consider the unique and differential sources of social influence across adolescent development with respect to neurobiological susceptibility. Second, the present study focused on an index of maladjustment (i.e., externalizing behavior), precluding its ability to fully explore the *advantage* or potential benefits conferred by heightened social motivational sensitivity at the neural level. Indeed, differential susceptibility theory highlights that the same neural endophenotype may confer advantages or disadvantages based on environmental contingencies. In this way, the present study better characterizes an interactive pattern reflective of diathesis stress (Jolicoeur-Martineau et al., 2020). Future studies should explore the full range of developmental outcomes, both adaptive and maladaptive, to speak to the potential cross-over interactive effects characteristic of differential susceptibility. Finally, the current study did not investigate a clinical population of externalizing youth, and thus it remains to be seen if the current findings generalize to clinical samples of adolescents. To this point, in contrast with previous literature, the present study did not find that amygdala or ventral striatum BOLD response independently predicted adolescent externalizing behavior, which may be related to this community sample's lower rate of externalizing behavior. Nonetheless, the current study demonstrates a pattern of neurobiological susceptibility (in the interaction of brain function and family conflict) emerging in a community sample of youth.

4.4. Conclusions

Despite these limitations, the current study provides evidence of neurobiological susceptibility in adolescent youth to one salient social context, the family environment, in predicting externalizing behavior. These results shed light on individual differences in social-affective neurobiology that may serve to confer risk or protect against social influence during this critical developmental period for risk-taking and problem behavior. Individual differences in neurobiological susceptibility may have significant implications for the efficacy of parent and family-focused preventative interventions, as adolescents high in neurobiological susceptibility to social influence may benefit the most from parent and family intervention. Thus, greater understanding of susceptibility factors is key for targeting at-risk youth and informing future prevention.

Data statement

Data from this study are unavailable to access because study participants did not consent to the public use of their data, so supporting data is not available.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.dcn.2021.100993.

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