Journal of Youth and Adolescence
https://doi.org/10.1007/s10964-023-01789-4

EMPIRICAL RESEARCH

The Effect of Parents and Peers on the Neural Correlates of Risk Taking and Antisocial Behavior During Adolescence

Christy R. Rogers[1] · Virnaliz Jimenez[2] · Amanda Benjamin[3] · Karen D. Rudolph[4] · Eva H. Telzer[5]

Received: 21 December 2022 / Accepted: 9 May 2023

Abstract

Social and neurobiological factors independently associate with the development of antisocial behavior during adolescence, yet it is unclear how these factors contribute to antisocial behavior in girls. Using a longitudinal sample of 45 adolescent girls (age in years at scan: $M = 15.38$, $SD = 0.33$), this study examined the contributions of parent-adolescent relationship quality and deviant peer affiliation from 6th–8th grades along with the neural correlates of risk taking in 9th grade to later antisocial behavior. High parent-adolescent closeness in early adolescence predicted lower antisocial behavior for girls in later adolescence via lower affiliation with deviant peer groups and less activation of the medial prefrontal cortex during risk taking. Findings highlight the enduring role of parents and peers during adolescence, and the importance of investigating social relationships alongside the brain to identify a holistic understanding of the development of antisocial behavior in girls.

Keywords Antisocial behavior · Adolescence · Parents · Peers · Risk taking · fMRI

Introduction

Antisocial behavior, including behaviors such as skipping school, using alcohol and drugs, and stealing, rapidly increases across adolescence (Meeus et al., 2021) around the world (Duell et al., 2019). Such behaviors have detrimental effects, contributing to greater physical health problems, criminal behavior, substance dependence, romantic relationship violence, and psychiatric problems, as well as poorer educational achievement and occupational stability during adulthood (Odgers et al., 2008). While childhood and early adolescent factors, such as negative parent and peer influences (Dogan et al., 2007; Kwon & Telzer, 2022), are among the strongest predictors of adolescent antisocial behavior (for a review, see Icenogle and Cauffman, 2021; Sitnick et al., 2017), there is relatively less known about the psychobiological processes through which these relationships may be associated with heightened antisocial behavior in adolescence, particularly in girls. Although adolescent girls engage in antisocial behavior (Piehler & Dishion, 2007), there is emerging literature suggesting that peers (Guyer et al., 2012), parents (Padilla et al., 2022), and neurobiological processing (i.e., ventral striatum activation, see Braams & Crone, 2017) differentially affect girls' behavior compared to boys. Given that adolescent girls are at a greater propensity to start engaging in antisocial behavior during adolescence compared to boys (Odgers et al., 2008), particularly in response to experiences within peer and parental relationships (Yang & Mcloyd, 2015), it would be beneficial to better understand the effects of peers, parents, and the brain together in understanding the development of antisocial behavior in girls. Drawing from a social contextual model of delinquency during adolescence (Scaramella et al., 2002), which emphasizes the joint influences of families and peers on adolescent antisocial behavior, this study used a longitudinal design to investigate the link between parent closeness and deviant peer affiliation in early adolescence on antisocial behavior in later adolescence. Furthermore, this model was expanded upon by using a developmental neuroscience perspective by examining neural activation during

✉ Eva H. Telzer
ehtelzer@unc.edu

1 Department of Human Development and Family Sciences, Texas Tech University, Lubbock, TX, USA

2 Department of Human Development and Family Sciences, University of Illinois at Urbana–Champaign, Champaign, IL, USA

3 John Jay College of Criminal Justice, City University of New York, New York, NY, USA

4 Department of Psychology, University of Illinois at Urbana–Champaign, Champaign, IL, USA

5 Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

risky decision-making as a mechanism through which family and peer contexts contribute toward the development of girls' antisocial behavior during adolescence.

The quality of parent-adolescent relationships contributes to adolescent engagement in antisocial behavior across time, such that high parental closeness predicts lower antisocial behavior (Rogers et al., 2018a), whereas frequent negative interactions with mothers predict greater rates of later antisocial behavior across adolescence (Defoe et al., 2013). Although parents continue to affect their adolescent offspring's antisocial behavior (Cavendish et al., 2012), adolescents tend to reorient toward peers as they navigate new developmental goals and relationships (Blakemore & Mills, 2014), and oftentimes engage in greater risk-taking behavior due to peer influence (King et al., 2017). Furthermore, associating with deviant peers is linked with antisocial behavior across childhood (Ettekal & Ladd, 2015) and adolescence (Piehler & Dishion, 2007), and specifically for adolescent girls, predicts higher rates of later vandalism, fighting, and stealing (Talbott & Thiede, 1999). Given the prominence of parent influence during childhood and into adolescence, it is no surprise that harsh parenting and involvement with deviant peers during early adolescence predicts antisocial behavior in late adolescence (Neppl et al., 2016), particularly for girls (Ehrenreich et al., 2022). Because parent-adolescent relationships can serve as a source of validation and support in buffering offspring from later deviant peer affiliation and antisocial behavior (Rudolph et al., 2020), it would benefit the field of developmental science to explore *how* these social influences contribute to girls' antisocial behavior.

Developmental social neuroscience has begun to unpack the psychobiological processes by which social relationships "get under the skin." Indeed, studies have explored links between family and peer relationship quality and the neural correlates of risk taking (Telzer et al., 2017). A meta-analysis identified the medial prefrontal cortex (mPFC), ventral striatum (VS), and insula as key regions involved in adolescent decision making in social contexts (van Hoorn et al., 2019). The mPFC is associated with social motivation and emotion regulation (Somerville et al., 2014), the VS with reward processing (Schreuders et al., 2018), and the insula with integrating affective and cognitive responses during decision-making (Smith et al., 2014). A growing body of work shows that activation in these brain regions facilitates adolescent decision-making by way of social relationships. Positive relationships (e.g., parent and sibling closeness) dampen activation in the VS during risky decision-making (Qu et al., 2015) and increase activation in the VS and insula during safe decision-making (Rogers et al., 2018b), whereas negative relationships (e.g., peer conflict) exacerbate activation in the VS and insula during risky decision-making (Telzer et al., 2015). Although family (Guassi Moreira & Telzer, 2018) and peer influences (Chein et al., 2011) have been linked with adolescent neural processing during risk taking, most of this work is cross-sectional (van Hoorn et al., 2018), or examines relationship quality one year previously (Qu et al., 2015) without examining the longer-term effects of family and peer relationships. Furthermore, social neuroscience research has primarily focused on the sole role of parents or peers without unpacking their interrelation and shared neural mechanisms that underlie adolescent girls' risk taking.

Current Study

Given that there is little understanding of how the effects of parents, peers, and the brain together inform the development of antisocial behavior in girls, this study examined the longitudinal associations of parent-adolescent closeness, deviant peer group affiliation, and brain activation during risky decision-making with later adolescent girls' antisocial behavior. Because girls can be more responsive to the benefits (i.e., parental warmth) and costs (i.e., peer victimization) of social relationships as they relate to adolescent antisocial behavior, girls may experience more neurobiological susceptibility to the effects of family and peer contexts on adolescent developmental outcomes, such as antisocial behavior. Given prior research showing that high-quality parent and peer relations are associated with less activation in the insula, VS, and mPFC during risk taking, it was hypothesized that high parent-adolescent closeness and low deviant peer affiliation across early adolescence (6th, 7th, and 8th grades) would be associated with less activation in these regions in mid adolescence (9th grade). In addition, a conceptual model tested whether high parent-adolescent closeness in early adolescence was associated with less antisocial behavior in later adolescence via deviant peer group affiliation and neural processing during risky decision-making. Specifically, it was hypothesized that high parent closeness would be associated with lower deviant peer group association, which would in turn be associated with lower activation in the brain regions of interest, which would then be associated with lower antisocial behavior. These effects were examined after controlling for previous antisocial behavior and peer victimization in 6th grade to identify the unique contribution of the variables of interest to later antisocial behavior above and beyond previous problematic behavior and peer experiences.

Methods

Participants and Procedures

Participants included 45 adolescent girls who were recruited in 9th grade ($M_{age} = 15.38$ years, $SD = 0.33$, range = 14.88–16.16 years) from a longitudinal study that tracked 636 (337 female) youth annually starting in 2nd grade (for more details, see Rudolph et al., 2014). Exclusion criteria for the study included MRI contraindications (e.g., metal implants), learning disabilities, and neurological-altering medications on the day of the scan session. Additionally, participants were recruited based on previous experiences of peer victimization given the overarching aims of the broader study, such that participants were either chronically victimized or non-victimized. Of the 50 participants who attended the scan session, 45 had useable data. There were no significant differences in the outcome variable (i.e., antisocial behavior at three, six, and nine months after the scan) between participants with ($n = 45$) and without ($n = 5$) usable scan data.

Of the 45 participants included in the final sample, the majority self-identified as White (68.9%), with 24.4% Black, 4.4% Asian, and 2.2% Latina. Participant socioeconomic backgrounds were diverse as represented by annual family income (33.3% <$29,000, 17.8% $30,000–$59,000, 20% $60,000–$89,000, and 28.9% <90,000) and highest reported education between mothers and fathers (2.2 % some high school, 24.4% high school diploma, 37.8% some college, 11.1% associate degree, 15.6% bachelor's degree, 2.2% some graduate school, and 6.7% advanced degree). Both the sample (Fowler et al., 2017; Rudolph et al., 2018) and the task of interest (Telzer et al., 2018) have been published previously, but not linked to all the constructs of interest in the current study.

The study design included participant report of their relationship with their parents and deviant peer group affiliation in 6th, 7th, and 8th grade. Questionnaires were administered in groups of 15–20 students during classroom sessions. In 9th grade, participants completed a risk-taking task during an fMRI scan. In addition, participants reported on their antisocial behavior during 6th grade (administered in classrooms), as well as 3, 6, and 9 months following the scan session (completed at home). Parents provided written consent and adolescence provided written assent in accordance with the Institutional Review Board.

Risk-Taking Task

A group membership manipulation was conducted to enhance social motivation toward risk taking. Participants were instructed that they were randomly assigned to either the blue team or the red team and were shown pictures of their team members and individuals on the opposing team. They were also told that their team was currently behind in points, and that their performance was important for their team catching up to win. This manipulation simulated a social context for risk taking.

During the fMRI scan, participants completed the Stoplight Task, which is a well-validated driving simulation used to examine performance and neural correlates of risky decision-making (Steinberg et al., 2008). Participants encountered yellow traffic lights at a series of intersections and had to decide whether to go or stop at each intersection; they were instructed to complete the course as quickly as possible. The decision to go through the yellow light is the fastest option (no delay), but may result in a crash, causing the longest delay (6 s). On the other hand, stopping at the intersection results in a short delay (3 s). Thus, deciding to go is risky, given the outcome represents the greatest penalty or benefit, whereas deciding to stop is safe. All of these task details were made explicit to participants. The task included 26 intersections, 8 of which had cars approaching on the cross street. Traffic signal timing (minimum of 8 s) and the presence of a car on the cross street varied to reduce predictability. Participants practiced the task before the scan to familiarize them with the task environment. On average, adolescents made 16.38 go decisions during practice ($SD = 3.82$, Min = 9, Max = 25, 63% intersections) and 16.27 go decisions during the scan ($SD = 4.19$, Min = 9, Max = 24; 63% intersections), and experienced 3.53 crashes during practice ($SD = 1.73$, Min = 0, Max = 8) and 5.11 crashes during the scan ($SD = 1.70$, Min = 1, Max = 8). The number of stop decisions was the inverse of the number of go decisions.

Self-Report Measures

Parent-adolescent closeness

During 6th, 7th, and 8th grade, adolescents completed 28 items from the Inventory of Parent and Peer Attachment (Armsden and Greenberg 1987) to measure how much adolescents felt they could trust, communicate with, and were supported by their parents. Items were rated on a 5-point Likert scale (1 = *almost never*, to 5 = *almost always*). Examples items included: "I trusted my parents," and, "I could count on my parents when I needed to talk," which yielded excellent reliability across measurement ($\alpha = 0.93$, 0.94, 0.96 at each wave, respectively).These items were averaged for each grade (Ms = 3.94, 3.97, 3.74, at each wave, respectively), and then averaged across the three times of assessment, which were highly correlated (rs = 0.58–0.73, ps < 0.001). Values across 6th, 7th, and 8th grades were aggregated to create a variable that reflected adolescent perceptions of parent-adolescent closeness across early adolescence, rather than just a snapshot from one year in middle school. Higher scores indicated greater closeness. The IPPA has successfully linked family and peer relationships to neural processing during risk taking in prior research (e.g., Qu et al., 2015; Telzer et al., 2013; Telzer et al., 2015).

Deviant peer group affiliation

During 6th, 7th, and 8th grade, adolescents completed a revised version of the Peer Behavior Inventory (Prinstein et al., 2001) to measure deviant peer group affiliation. Adolescents were asked to list the initials of their closest friends. Then, using a 5-point Likert scale (1 = *never*, to 5 = *very often*), adolescents answered nine items about how often these friends engaged in problematic behaviors (e.g., "hit or threatened to hit someone," "cheated on tests"). These items were averaged (*M*s = 1.45, 1.42, 1.47, at each wave, respectively), with higher scores indicating greater deviant peer group affiliation ($\alpha = 0.90$, 0.91, 0.87 at each wave, respectively). Scores were moderately to highly correlated (*r*s = 0.34–0.70, *p*s = 0.001–0.020), and averaged across the three times of assessment to represent deviant group peer affiliation across early adolescence, with higher scores indicating a greater frequency of deviancy within adolescent peer groups.

Antisocial behavior

Adolescents completed a 13-item antisocial behavior questionnaire adapted from a questionnaire designed specifically for female adolescents (Nolen-Hoeksema et al., 2007). Participants used a 5-point scale (1 = *not at all*, to 5 = *extremely*) to indicate how much each item describes them (e.g., "I stole things," and "I cut classes or skipped school"). Reports were collected during 6th grade, and again 3, 6, and 9 months after the scan session (*M*s = 1.62, 1.53, 1.45, 1.53, at each wave, respectively), which yielded excellent reliability across time ($\alpha = 0.92$, 0.91, 0.90, 0.92 at each wave, respectively). The 6th grade assessment was used to control for previous levels of antisocial behavior to examine parental and peer effects on later adolescent antisocial behavior, above and beyond early rates of antisocial behavior. The three assessments following the scan were highly correlated (*r*s = 0.82–0.88, *p*s < 0.001), and averaged, with higher scores indicating higher levels of engagement in antisocial behavior. One participant did not complete the follow-up measures; thus, analyses examining antisocial behavior include 44 participants.

Peer victimization

Given that the sample was selected based on girls' experiences of peer victimization via their report on the Social Experiences Questionnaire—Revised, this measure was included in the analysis (Rudolph et al., 2014). Participants completed 21 items on how often they experienced relational victimization (10 items), which reflects being manipulated or harmed through relationships (e.g., "How often does another kid say they won't like you unless you do what they want you to do?"). In addition, they completed 11 items on how often they experienced overt victimization, which reflects threats or acts of physical harm (e.g., "How often do you get hit by another kid?"). Participants reported their experiences using a 5-point scale (1 = *never*, to 5 = *always*), such that higher scores reflected more frequent experiences of peer victimization. Report on the relational and covert peer victimization subscales during 6th grade were averaged and modeled as a covariate to control for the recruitment criteria of the neuroimaging sample.

fMRI Data Acquisition

Brain images were collected using a research-dedicated 3 Tesla Siemens Trio MRI scanner. The Stoplight task was presented on a computer screen and projected through a mirror. T2*-weighted echo-planar imaging (EPI; TR = 2000ms; TE = 25 ms; matrix = 92 × 92; FOV = 230 mm; 38 slices; slice thickness = 3 mm; voxel size 2.5 × 2.5 × 3 mm^3) was conducted to acquire the Stoplight task. Structural scans included a T2*-weighted matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4000 ms; TE = 64 ms; matrix = 192 × 192; FOV = 230 mm; 38 slices; slice thickness = 3 mm) and a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1900ms; TE = 2.3 ms; matrix = 256 × 256; FOV = 230 mm; sagittal plane; slice thickness = 1 mm; 192 slices). The orientation for the EPI and MBW scans was oblique axial to maximize brain coverage and to reduce noise.

fMRI Data Preprocessing and Analysis

fMRI data were preprocessed and analyzed using Statistical Parametric Mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London). Participant EPI images were spatially realigned to correct for head motion. No participant exceeded 2 mm of maximum image-image motion in any direction. Next, the realigned EPI images were coregistered to the MPRAGE, which was then segmented into cerebrospinal fluid, gray matter, and white matter. The normalization transformation matrix from the segmentation step was then applied to the functional and T2 structural images, transforming them into standard stereotactic space as defined by the Montreal Neurological Institute and the International Consortium for Brain Mapping. After normalization, the EPI images underwent spatial smoothing using an 8 mm FWHM Gaussian kernel to increase the signal-to-noise ratio in the functional images. High-pass temporal filtering of 128 s was applied to eliminate low-frequency drift across the time series. Serial autocorrelations were estimated with a restricted maximum likelihood algorithm with an auto-regressive model order of 1.

A fixed-effects general linear model was created for each participant with 4 regressors, including 2 decisions (go and stop) and 2 outcomes (pass and crash). In addition, the wait time after safe decisions and the final "game over" period were modeled to remove these from the implicit baseline. The duration of the decision trials was measured as the time at which the traffic light appeared until the participant's response time because the task was self-paced. The onset time for crashes occurred when a cross-traffic car crashed into the participant's car, and for passes occurred 2 s after the yellow light. The duration of the outcome trials was 1 s. The jittered inter-trial intervals were not explicitly modeled, and thus, represented an implicit baseline. Linear contrast images were generated from the general linear model parameter estimates.

Random-effects, whole brain analyses were conducted utilizing GLMFlex for all voxels containing values (http://mrtools.mgh.harvard.edu/index.php/GLM_Flex). The group level analyses assessed neural activation when adolescents made risky decisions (i.e., go decisions). Two separate whole-brain regression analyses were conducted, one which included parent closeness and the second which included deviant peer group association. To correct for multiple comparisons, Monte Carlo simulations were run using the group-level brain mask for risky decisions. The simulations were run through 3dClustSim within the AFNI software package (Ward, 2000; updated April 2016) and utilized the acf option to estimate the intrinsic smoothness. The simulations resulted in a minimum cluster size of 52 and 50 contiguous voxels using a $p < 0.005$ voxel-wise threshold, corresponding to $p < 0.05$, for parent closeness and deviant peer group affiliation, respectively. All reported results are available on NeuroVault (Gorgolewski et al., 2015; see https://neurovault.org/collections/DSEBVDEL/). Code is available upon request.

Analytical Plan

First, whole brain analyses were conducted to identify brain regions that significantly correlated with parent-adolescent closeness (composite across 6th, 7th, and 8th grades) and deviant peer group affiliation (composite across 6th, 7th, and 8th grades) while adolescents engaged in risky decision-making (i.e., during go decisions). Given the extensive overlap in activation in the mPFC across both whole-brain analyses, a mask was created for this portion of the mPFC for subsequent analysis. Second, a mediation model was computed using the PROCESS macro in SPSS to test deviant peer group affiliation and activation in the mPFC during risky decision-making as mediators between parent-adolescent closeness and later antisocial behavior. Peer victimization and antisocial behavior reported at 6th grade were modeled as covariates.

Results

Descriptive statistics and correlations of all study variables are displayed in Table 1. Two separate whole brain analyses were conducted during go decisions (9th grade), one with parent-adolescent closeness (6th, 7th, 8th grade) and a second with deviant peer group affiliation (6th, 7th, 8th grade). A negative correlation was found between parent-adolescent closeness and mPFC activation (Fig. 1, Table 2), such that higher perceptions of closeness were associated

Table 1 Descriptive statistics and correlations between variables

Time of measurement	6th grade		6th, 7th, 8th grade		9th grade (Scan)	9th-10th grade (3-, 6-, 9- months post scan)
Measure	Peer victimization	Antisocial behavior	Parent-adolescent closeness	Deviant peer group association	mPFC activation during risk taking	Antisocial behavior
Peer victimization	–	0.67***	−0.67**	0.67**	0.47**	0.53**
Antisocial behavior		–	−0.50**	0.79**	0.35*	0.48**
Parent-adolescent closeness			–	−0.66**	−0.51**	−0.46**
Deviant peer group association				–	0.54**	0.66**
mPFC activation					–	0.57**
Antisocial behavior						–
Mean	1.89	1.62	3.88	1.45	−1.72	1.52
SD	0.82	0.71	0.68	0.52	3.47	0.59

M mean; *SD* standard deviation

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$. Two-tailed significance

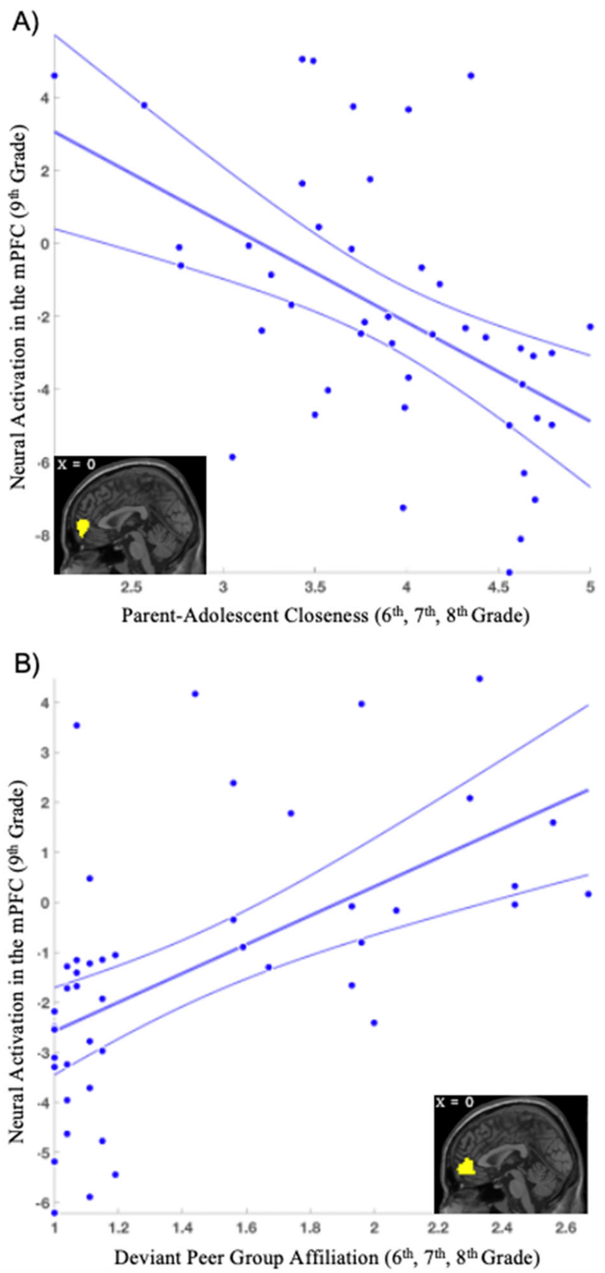

Fig. 1 $N = 45$. Neural activation in the medial prefrontal cortex (mPFC) during risky decision-making in 9th grade is correlated with **A** lower parent closeness and **B** higher deviant peer group affiliation during 6th, 7th, and 8th grades

with less activation in the mPFC during risky decision-making. Parent-adolescent closeness was also associated with less activation in the VS during risk taking. Deviant peer group affiliation was positively correlated with mPFC activation (Fig. 1), such that greater affiliation with deviant peer groups was associated with higher activation in the mPFC during risky decision-making. Notably, the mPFC region that correlated with both parent-adolescent closeness and deviant peer affiliation was nearly identical. No other

Table 2 Neural regions that associated with parent closeness and deviant peer group affiliation during go trials in the stoplight task

Predictor	Anatomical region (9th grade)	+/–	*x*	*y*	*z*	*t*	*k*
Parent-adolescent closeness (6th, 7th, 8th grade)							
	mPFC	–	3	62	–2	–4.41	261
	R medial temporal pole	–	33	26	–32	–4.99	179[a]
	R vmPFC	–	3	5	–17	–3.53	[a]
	R ventral striatum	–	18	17	–11	–3.45	[a]
	L postcentral gyrus	–	–45	–28	58	–3.57	54
	L middle temporal gyrus	–	–69	–31	–5	–3.39	71
	L paracentral lobule	+	–18	–43	46	4.69	57
	R paracentral lobule	+	15	–46	55	4.03	207
Deviant peer group affiliation (6th, 7th, 8th grade)							
	mPFC	+	12	47	1	4.71	331
	R posterior-medial frontal	–	15	–1	64	–5.14	955[b]
	R middle cingulate cortex	–	9	11	46	–4.56	[b]

L and R refer to left and right hemispheres; +/– refer to positive and negative correlation; x, y, and z refer to MNI coordinates; t refers to peak activation level in each cluster; and k refers to the number of voxels in each significant cluster. Regions that share the same superscript are part of the same cluster

All regions are significant at $p < 0.005$

regions showed overlap in activation across the two analyses. Additional regions that correlated with parent-adolescent closeness and deviant peer group affiliation are displayed in Table 2.

Given that activation in the mPFC during risky decision-making was associated with both parent-adolescent closeness and deviant peer group affiliation, analyses were conducted to test our full conceptual model, examining whether mPFC activation mediates the association between previous social influences and later adolescent antisocial behavior. To this end, a mask was created of the overlapping voxels in the mPFC clusters from the parent-adolescent closeness and deviant peer group association whole-brain analyses and extracted parameter estimates of signal intensity from the mPFC mask during risky decision-making. Next, a mediation model was computed to examine whether parent-adolescent closeness (6th, 7th, 8th grade) is associated with later antisocial behavior (9th–10th grade) via deviant peer group affiliation (6th, 7th, 8th grade) and subsequent mPFC activation during risky decision-making (9th grade). The mediator and outcome variables were regressed onto peer victimization and antisocial behavior in 6th grade to control for earlier peer victimization and previous levels of antisocial behavior. The results revealed a significant indirect effect of parent-adolescent closeness on

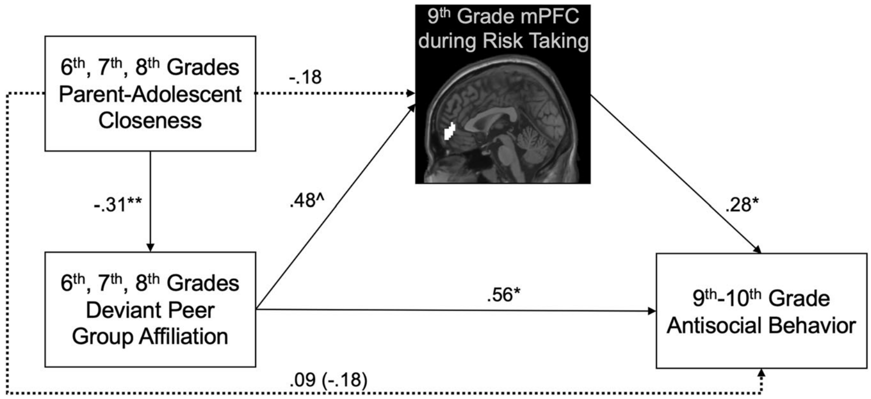

Fig. 2 $N = 44$. Mediation model examining the role of deviant peer group affiliation and medial prefrontal cortex (mPFC) activation during risk taking as mediators of the association between parent-adolescent closeness and adolescent antisocial behavior. The model adjusted for previous levels of peer victimization and antisocial behavior in 6th grade. The model evidenced a significant indirect effect [ß = −0.043, SE = 0.038, 95% CI (−0.142, −0.001)]. Coefficients are standardized. Dashed pathways are nonsignificant. ^$p = 0.053$, *$p < 0.05$, **$p < 0.01$

later adolescent antisocial behavior via lower affiliation with deviant peer groups and less activation in the mPFC during risky decision-making [ß = −0.043, SE = 0.038, 95% CI (−0.142, −0.001); Fig. 2]. In addition, deviant peer group affiliation also had a significant indirect effect on later antisocial behavior via more activation in the mPFC during risky decision-making [ß = 0.157, SE = 0.098, 95% CI (0.024, 0.412)].

Discussion

A vast majority of research focuses on understanding antisocial behavior in boys despite girls showing a higher initiation rate of antisocial behavior during early adolescence (Odgers et al., 2008), identifying a critical need to better understand the social and neurobiological factors that may place adolescent girls on antisocial trajectories. The goal of this study was to examine the influence of parents, peers, and brain activation during risk taking as joint predictors of adolescent girl antisocial behavior using a longitudinal design. Although previous research has identified parents, peers, and the neural correlates of decision-making as contributing to adolescent antisocial behavior independently and primarily in boy and mixed sex samples, this report distinguishes parent-adolescent closeness as a protective factor, and deviant peer affiliation and mPFC activation during risky decision-making as risk factors, through which girls develop antisocial behavior later in adolescence. The results highlight the salience of both parent and peer relationships for girls in early adolescence as they contribute to neural processing during risky decision-making and antisocial behavior into mid adolescence. Specifically, the mPFC was identified as a key brain region associated with both parent-adolescent closeness (less activation) and deviant peer affiliation (more activation). These findings underscore the dynamic interplay of parents, peers, and the brain on the development of antisocial behavior in girls during adolescence.

The findings show that parent-adolescent closeness and deviant peer affiliation were each associated with adolescents' neural processing while making risky decisions. Both social influences were linked to activation in the mPFC, a region associated with social motivation and regulation (Somerville et al., 2014). The results indicated that high parent-adolescent closeness was linked to less recruitment of the mPFC during risky decision-making, and that high deviant peer group affiliation was linked to more recruitment. These findings are consistent with the role of the mPFC as integral for processing social information (Somerville et al., 2013) and boosting motivation in the pursuit of rewards (Shulman et al., 2016) during adolescence, such that affiliation with deviant peers may facilitate adolescents' motivation to pursue risky behaviors whereas close parent ties may adaptively dampen this inclination. High parent-adolescent closeness was also associated with lower activation in the VS, which is consistent with previous work linking positive family influences (e.g., parent closeness and family obligation values) to lower recruitment of the VS during risky decision-making (Qu et al., 2015). The VS is consistently linked with age differences in reward processing and social influence on valuation (Schreuders et al., 2018), suggesting that close parental ties may be associated with less rewarding experiences when taking risks. Other patterns of activation that associated with parent-adolescence closeness included lower activation in the postcentral gyrus and middle temporal gyrus, as well as higher activation in the paracentral lobules. Moreover, greater affiliation with deviant peers associated with lower activation in the middle cingulate cortex (MCC), a region that has been implicated in reward processing and motivation (Becker et al., 2017). Contrary to our hypothesis and previous work with adolescent boys and girls (Telzer et al., 2015), activation in the insula was not associated with parent-adolescent closeness or deviant peer group affiliation for adolescent girls. These activation patterns may differ from previous studies of adolescent boys and mixed gender samples given that adolescent girls are uniquely socialized to respond to relationship cues (Yang & Mcloyd, 2015) and engage in specific antisocial behaviors (Bongers et al., 2011). Together, these findings corroborate the mPFC and VS as salient regions for social relationships getting "under the skin" as adolescents navigate choices to take risks or abstain from them.

Importantly, analysis of our full conceptual model showed that high parent-adolescent closeness and lower deviant peer group affiliation across middle school indirectly predicted girls' antisocial behavior in high school through less recruitment of the mPFC during risk taking. These findings underscore the importance of investigating multiple important social relationships alongside the brain to better understand adolescent girls' antisocial behavior. Because the mPFC has been implicated in regulatory and motivational processes (Somerville et al., 2014), these findings demonstrate the importance of integrating a developmental neuroscience perspective into the social contextual model of delinquency (Scaramella et al., 2002), such that relationship dynamics with parents and peers manifest neurobiologically in adolescent regulatory and motivational processing, thereby helping to shape later adolescent delinquent behavior (Dishion & Patterson, 2015). Our conceptual model highlights the complexity of psychobiological antecedents that can lay the foundation for adolescent antisocial behavior for girls, and as such, provides an opportunity for future work to investigate resiliency and risk factors in social and neurobiological processes.

Although this report sheds light on the developmental processes associated with antisocial behavior in adolescent girls, its limitations are important to address. First, because adolescent girls are underrepresented in the antisocial behavior literature and are more likely to evidence the onset of these behaviors during adolescence compared to boys (Odgers et al., 2008), the focus of the study included a sample of females, and thus, our conclusions may not generalize to adolescent boys. These social and neural processes should be examined in a larger sample of both girls and boys to investigate sex differences and universal processes in how these factors contribute to adolescent antisocial behavior. Further, it would be advantageous to examine decision-outcome associations across the task (i.e., whether decisions resulted in crashes or passing through the intersection safely) as it can disentangle advantageous versus disadvantage decision-making, and further inform how risky decision-making associates with antisocial behavior in adolescent girls (e.g., Op de Macks et al., 2018). Second, the longitudinal assessments of parent-adolescent closeness and deviant peer group affiliation at 6th, 7th, and 8th grade allowed us to examine social relationships throughout middle school, which contributed to a much-needed interdisciplinary perspective on the development of antisocial behavior in adolescent girls. It should be noted that this was also a limitation of the analytical design given that parent-adolescent closeness and deviant peer group affiliation were measured concurrently and tested sequentially within a mediation model. Caution should be taken in interpreting the sequential order of parent and peer social influence. Third, a larger sample size would allow the ability to detect small effect sizes, including the association between deviant peer group affiliation and activation in the mPFC. It would also lend to more sophisticated analyses, such as growth curve modeling (Widaman et al., 2010) to tease apart how these social influences change over time, and how these changes contribute to neural processing and antisocial behavior during adolescence. In addition, obtaining assessments of neural activation at multiple time points would benefit future research in teasing apart the direction of effect between brain function, social relationships, and antisocial behavior across adolescence. Last, there was a focus on positive aspects of parent-adolescent relationships (i.e., closeness) and negative aspects of adolescent peer relationships (i.e., delinquency of peers), and thus, future work would benefit from including positive and negative dimensions of salient social agents, as well as using a larger sample size to detect the interplay between these dimensions.

Conclusion

There is little known how the influences of parents, peers, and risk taking at the level of the brain together contribute to the development of antisocial behavior in adolescent girls. An integrated contextual and psychobiological approach was used to identify how relationships with parents and peers during early adolescence predict neural processing during risk taking and subsequent antisocial behavior during later adolescence. Parent-adolescent closeness and deviant peer group affiliation differentially associated with mPFC activity while adolescents take risks. Importantly, strong ties with parents were associated with lower antisocial behavior across adolescence, which manifests through concurrently associating with less delinquent peers and later lower activation of the mPFC during risk taking. Given the importance of investing in adolescent development and health using developmental neuroscience

(Dahl et al., 2018), this report underscores the continued influence of parents, and thus, the importance of validating and bolstering parental efforts to support adolescent success across development. In kind, it also emphasizes the negative influence peers can hold over adolescent girls in their development of antisocial tendencies. This research informs applied work with adolescent girls, such that girls who orient away from supportive friend groups toward deviant peer groups would benefit from supportive interactions with their parents.

Acknowledgements We are grateful to Michelle Miernicki, Jamie Abaied, Monica Agoston, Samirah Ali, Suravi Changlani, Megan Flynn, Inge Karosevica, Nicole Llewellyn, Jennifer Monti, Heather Ross, and Niwako Sugimura for their assistance in data collection and management.

Authors' Contributions C.R.R. conceived of the paper, conducted fMRI and regression analyses, and wrote the full manuscript; V.J. assisted with fMRI analyses and manuscript revisions; A.B. assisted with fMRI analyses and reviewed the manuscript; K.D.R. created the research design, obtained funding, and contributed to manuscript revisions; E.H.T. created the research design, obtained funding, and contributed to manuscript revisions. All authors read and approved the final manuscript.

Funding This work was supported by a University of Illinois Research Board Award and National Institute of Mental Health Grants MH68444 (to K.D.R.) and MH105655 (to K.D.R. and E.H.T.).

Data Sharing and Declaration The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Compliance with Ethical Standards

Conflict of Interest The authors declare no competing interests.

Ethical Approval All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Parents provided written consent and adolescence provided written assent in accordance with the Institutional Review Board.

References

Armsden, G. C., & Greenberg, M. T. (1987). The inventory of parent and peer attachment: Individual differences and their relationship to psychological well-being in adolescence. *Journal of Youth and Adolescence*, *16*, 427–454. https://doi.org/10.1007/BF02202939.

Becker, C. A., Flaisch, T., Renner, B., & Schupp, H. T. (2017). From thirst to satiety: the anterior mid-cingulate cortex and right posterior insula Indicate dynamic changes in incentive value. *Frontiers in Human Neuroscience*, *11*(May), 1–9. https://doi.org/10.3389/fnhum.2017.00234.

Blakemore, S.-J., & Mills, K. L. (2014). Is adolescence a sensitive period for sociocultural processing? *Annual Review of Psychology*, *65*, 187–207. https://doi.org/10.1146/annurev-psych-010213-115202.

Bongers, I. L., Koot, H. M., Van Der Ende, J., & Verhulst, F. C. (2011). Developmental trajectories of externalizing behaviors in childhood and adolescence. *Child Development*, *75*, 1523–1537.

Braams, B. R., & Crone, E. A. (2017). Peers and parents: a comparison between neural activation when winning for friends and mothers in adolescence. *Social Cognitive and Affective Neuroscience*, *12*, 417–426. https://doi.org/10.1093/scan/nsw136.

Cavendish, W., Nielsen, A. L., & Montague, M. (2012). Parent attachment, school commitment, and problem behavior trajectories of diverse adolescents. *Journal of Adolescence*, *35*, 1629–1639. https://doi.org/10.1016/j.adolescence.2012.08.001.

Chein, J., Albert, D., O'Brien, L., Uckert, K., & Steinberg, L. (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Developmental Science*, *14*, F1–10. https://doi.org/10.1111/j.1467-7687.2010.01035.x.

Dahl, R. E., Allen, N. B., Wilbrecht, L., & Suleiman, A. B. (2018). Importance of investing in adolescence from a developmental science perspective. *Nature*, *554*, 441–450. https://doi.org/10.1038/nature25770.

Defoe, I. N., Keijsers, L., Hawk, S. T., Branje, S., Dubas, J. S., Buist, K., Frijns, T., Van Aken, M. A. G., Koot, H. M., Van Lier, P. A. C., & Meeus, W. (2013). Siblings versus parents and friends: Longitudinal linkages to adolescent externalizing problems. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *54*(8), 881–889. https://doi.org/10.1111/jcpp.12049.

Dishion, T. J., & Patterson, G. R. (2015). The development and ecology of antisocial behavior. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Volume 3: risk, disorder, and adaptation* (pp. 503–541). Wiley & Sons, Inc. https://doi.org/10.1002/9780470939406.ch13

Dogan, S. J., Conger, R. D., Kim, K. J., & Masyn, K. E. (2007). Cognitive and parenting pathways in the transmission of antisocial behavior. *Child Development*, *78*, 335–349. https://doi.org/10.1111/j.1467-8624.2007.01001.x.

Duell, N., Steinberg, L., Icenogle, G., Chein, J., Chaudhary, N., Di Giunta, L., Dodge, K. A., Fanti, K. A., Lansford, J. E., Oburu, P., Pastorelli, C., Skinner, A. T., Sorbring, E., Tapanya, S., Uribe Tirado, L. M., Alampay, L. P., Al-Hassan, S. M., Takash, H. M. S., Bacchini, D., & Chang, L. (2019). Age patterns in risk taking across the world. *Journal of Youth and Adolescence*, *47*, 1052–1072. https://doi.org/10.1007/s10964-017-0752-y.

Ehrenreich, S. E., Jouriles, E. N., Mortensen, J. A., Meter, D. J., & Underwood, M. K. (2022). Peer communication about antisocial Activities as a mediator of interparental conflict in mid-adolescence and externalizing problems in late adolescence. *Journal of Child and Family Studies*, *31*(11), 3221–3233. https://doi.org/10.1007/s10826-022-02427-6.

Ettekal, I., & Ladd, G. W. (2015). Developmental pathways from childhood aggression-disruptiveness, chronic peer rejection, and deviant friendships to early-adolescent rule breaking. *Child Development*, *86*, 614–631. https://doi.org/10.1111/cdev.12321.

Fowler, C. H., Miernicki, M. E., Rudolph, K. D., & Telzer, E. H. (2017). Disrupted amygdala-prefrontal connectivity during emotion regulation links stress-reactive rumination and adolescent depressive symptoms. *Developmental Cognitive Neuroscience*, *27*, 99–106. https://doi.org/10.1016/j.dcn.2017.09.002.

Gorgolewski, K. J., Varoquaux, G., Rivera, G., Schwarz, Y., Ghosh, S. S., Maumet, C., Sochat, V. v., Nichols, T. E., Poldrack, R. A., Poline, J.-B., Yarkoni, T., & Margulies, D. S. (2015). NeuroVault.org: a web-based repository for collecting and sharing unthresholded statistical maps of the human brain. *Frontiers in Neuroinformatics 9*, https://doi.org/10.3389/fninf.2015.00008.

Guassi Moreira, J. F., & Telzer, E. H. (2018). Mother still knows best: Maternal influence uniquely modulates adolescent reward sensitivity during risk taking. *Developmental Science*, *21*, 1–11. https://doi.org/10.1111/desc.12484.

Guyer, A. E., Choate, V. R., Pine, D. S., & Nelson, E. E. (2012). Neural circuitry underlying affective response to peer feedback in adolescence. *Social Cognitive and Affective Neuroscience*. https://doi.org/10.1093/scan/nsr043.

Icenogle, G., & Cauffman, E. (2021). Adolescent decision making: a decade in review. *Journal of Research on Adolescence, 31*(4), 1006–1022. https://doi.org/10.1111/jora.12608

King, K. M., McLaughlin, K. A., Silk, J., & Monahan, K. C. (2017). Peer effects on self-regulation in adolescence depend on the nature and quality of the peer interaction. *Development and Psychopathology*, 1–13. https://doi.org/10.1017/S0954579417001560

Kwon, S.-J., & Telzer, E. H. (2022). Social contextual risk taking in adolescence. *Nature Reviews Psychology*, *1*(7), 393–406. https://doi.org/10.1038/s44159-022-00060-0.

Meeus, W., Vollebergh, W., Branje, S., Crocetti, E., Ormel, J., van de Schoot, R., Crone, E. A., & Becht, A. (2021). On imbalance of impulse control and sensation seeking and adolescent risk: an intra-individual developmental test of the Dual Systems and Maturational Imbalance Models. *Journal of Youth and Adolescence*, *50*, 827–840. https://doi.org/10.1007/s10964-021-01419-x.

Neppl, T. K., Dhalewadikar, J., & Lohman, B. J. (2016). Harsh parenting, deviant peers, adolescent risky behavior: Understanding the meditational effect of attitudes and intentions. *Journal of Research on Adolescence*, *26*(3), 538–551. https://doi.org/10.1111/jora.12212.

Nolen-Hoeksema, S., Stice, E., Wade, E., & Bohon, C. (2007). Reciprocal relations between rumination and bulimic, substance abuse, and depressive symptoms in female adolescents. *Journal of Abnormal Psychology*, *116*, 198–207. https://doi.org/10.1037/0021-843X.116.1.198.

Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R., Thomson, W. M., & Caspi, A. (2008). Female and male antisocial trajectories: from childhood origins to adult outcomes. *Development and Psychopathology*, *20*(2), 673–716. https://doi.org/10.1017/S0954579408000333.

Op de Macks, Z. A., Flannery, J. E., Peake, S. J., Flournoy, J. C., Mobasser, A., Alberti, S. L., Fisher, P. A., & Pfeifer, J. H. (2018). Novel insights from the Yellow Light Game: Safe and risky decisions differentially impact adolescent outcome-related brain function. *NeuroImage*, *181*, 568–581. https://doi.org/10.1016/j.neuroimage.2018.06.058.

Padilla, J., Updegraff, K. A., McHale, S. M., Umaña-Taylor, A. J., & Jager, J. (2022). Longitudinal associations between Mexican-origin youth's relationships with parents, siblings, and friends and individual adjustment. *Journal of Social and Personal Relationships*, 026540752211194. https://doi.org/10.1177/02654075221119428.

Piehler, T. F., & Dishion, T. J. (2007). Interpersonal dynamics within adolescent friendships: dyadic mutuality, deviant talk, and patterns of antisocial behavior. *Child Development*, *78*, 1611–1624. https://doi.org/10.1111/j.1467-8624.2007.01086.x.

Prinstein, M. J., Boergers, J., & Spirito, A. (2001). Adolescents' and Their Friends' Health-Risk Behavior: Factors That Alter or Add to Peer Influence. *Journal of Pediatric Psychology*, *26*(5), 287–298. https://doi.org/10.1093/jpepsy/26.5.287.

Qu, Y., Fuligni, A. J., Galvan, A., & Telzer, E. H. (2015). Buffering effect of positive parent–child relationships on adolescent risk taking: a longitudinal neuroimaging investigation. *Developmental Cognitive Neuroscience*, *15*, 26–34. https://doi.org/10.1016/j.dcn.2015.08.005.

Rogers, C. R., McCormick, E. M., van Hoorn, J., Ivory, S. L., & Telzer, E. H. (2018a). Neural correlates of sibling closeness and association with externalizing behavior in adolescence. *Social Cognitive and Affective Neuroscience*, *13*, 977–988. https://doi.org/10.1093/scan/nsy063.

Rogers, C. R., McCormick, E. M., van Hoorn, J., Ivory, S. L., & Telzer, E. H. (2018b). Neural correlates of sibling closeness and association with externalizing behavior in adolescence. *Social Cognitive and Affective Neuroscience*, *13*, 977–988. https://doi.org/10.1093/scan/nsy063.

Rudolph, K. D., Davis, M. M., Modi, H. H., Fowler, C., Kim, Y., & Telzer, E. H. (2018). Differential susceptibility to parenting in adolescent girls: Moderation by neural sensitivity to social cues. *Journal of Research on Adolescence*, 1–15. https://doi.org/10.1111/jora.12458.

Rudolph, K. D., Lansford, J. E., Agoston, A. M., Sugimura, N., Schwartz, D., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2014). Peer victimization and social alienation: predicting deviant peer affiliation in middle school. *Child Development*, *85*, 124–139. https://doi.org/10.1111/cdev.12112.

Rudolph, K. D., Monti, J. D., Modi, H., Sze, W. Y., & Troop-Gordon, W. (2020). Protecting youth against the adverse effects of peer victimization: why do parents matter? *Journal of Abnormal Child Psychology*, *48*(2), 163–176. https://doi.org/10.1007/s10802-019-00576-9.

Rudolph, K. D., Troop-Gordon, W., Monti, J. D., & Miernicki, M. E. (2014). Moving against and away from the world: The adolescent legacy of peer victimization. *Development and Psychopathology*, *26*, 721–734. https://doi.org/10.1017/S0954579414000340.

Scaramella, L. V., Conger, R. D., Spoth, R., & Simons, R. L. (2002). Evaluation of a social contextual model of delinquency: a cross-study replication. *Child Development*, *73*, 175–195. https://doi.org/10.1111/1467-8624.00399.

Schreuders, E., Braams, B. R., Blankenstein, N. E., Peper, J. S., Güroğlu, B., & Crone, E. A. (2018). Contributions of reward sensitivity to ventral striatum activity across adolescence and early adulthood. *Child Development*, *89*, 797–810. https://doi.org/10.1111/cdev.13056.

Shulman, E. P., Smith, A. R., Silva, K., Icenogle, G., Duell, N., Chein, J., & Steinberg, L. (2016). The dual systems model: Review, reappraisal, and reaffirmation. *Developmental Cognitive Neuroscience*, *17*, 103–117. https://doi.org/10.1016/j.dcn.2015.12.010.

Sitnick, S. L., Shaw, D. S., Weaver, C. M., Shelleby, E. C., Choe, D. E., Reuben, J. D., Gilliam, M., Winslow, E. B., & Taraban, L. (2017). Early childhood predictors of severe youth violence in low-income male adolescents. *Child Development*, *88*, 27–40. https://doi.org/10.1111/cdev.12680.

Smith, A. R., Steinberg, L., & Chein, J. (2014). The role of the anterior Insula in adolescent decision making. *Developmental Neuroscience*, *36*, 196–209. https://doi.org/10.1159/0003589188.

Somerville, L. H., Jones, R. M., Ruberry, E. J., & Dyke, J. P. (2014). Medial prefrontal cortex and the emergence of self-conscious emotion in adolescence. *Psychological Science*, *24*, 1554–1562. https://doi.org/10.1177/0956797613475633.Medial.

Somerville, L. H., Jones, R. M., Ruberry, E. J., Dyke, J. P., Glover, G., & Casey, B. J. (2013). The medial prefrontal cortex and the emergence of self-conscious emotion in adolescence. *Psychological Science*, *24*, 1554–1562. https://doi.org/10.1177/0956797613475633.

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. *Developmental Psychology*, *44*, 1764–1778. https://doi.org/10.1037/a0012955.

Talbott, E., & Thiede, K. (1999). Pathways to antisocial behavior among adolescent girls. *Journal of Emotional and Behavioral Disorders*, *7*, 31–39. https://doi.org/10.1177/106342669900700104.

Telzer, E. H., Fuligni, A. J., Lieberman, M. D., & Galvan, A. (2013). Meaningful family relationships: Neurocognitive buffers of adolescent risk taking. *Journal of Cognitive Neuroscience*, *25*, 374–387.

Telzer, E. H., Fuligni, A. J., Lieberman, M. D., Miernicki, M. E., & Galván, A. (2015). The quality of adolescents' peer relationships modulates neural sensitivity to risk taking. *Social Cognitive and Affective Neuroscience*, *10*, 389–398. https://doi.org/10.1093/scan/nsu064.

Telzer, E. H., Miernicki, M. E., & Rudolph, K. D. (2018). Chronic peer victimization heightens neural sensitivity to risk taking. *Development and Psychopathology*, *30*, 13–26. https://doi.org/10.1017/S0954579417000438.

Telzer, E. H., Rogers, C. R., & van Hoorn, J. (2017). Neural correlates of social influence on risk taking and substance use in adolescents. *Addictions Report*, *4*, 333–341. https://doi.org/10.1007/s40429-017-0164-9.

van Hoorn, J., McCormick, E., Rogers, C., Ivory, S., & Telzer, E. (2018). Differential effects of parent and peer presence on neural correlates of risk taking in adolescence. *Social Cognitive and Affective Neuroscience*, *13*, 945–955. https://doi.org/10.1093/scan/nsy024/4965846.

van Hoorn, J., Shablack, H., Lindquist, K. A., & Telzer, E. H. (2019). Incorporating the social context into neurocognitive models of adolescent decision-making: a neuroimaging meta-analysis. *Neuroscience and Biobehavioral Reviews*, 1–14. https://doi.org/10.1016/j.neubiorev.2018.12.024

Ward, B. D. (2000). Simultaneous inference for fMRI data [WWW]. Available: https://afni.nimh.nih.gov/pub/dist/doc/manual/AlphaSim.pdf.

Widaman, K. F., Ferrer, E., & Conger, R. D. (2010). Factorial invariance within longitudinal structural equation models: Measuring the same construct across time. *Child Development Perspectives*, *4*, 10–18. https://doi.org/10.1111/j.1750-8606.2009.00110.x.Factorial.

Yang, G. S., & Mcloyd, V. C. (2015). Do parenting and family characteristics moderate the relation between peer victimization and antisocial behavior? A 5-year longitudinal study. *Social Development*, *24*(4), 748–765. https://doi.org/10.1111/sode.12118.

Christy Rogers is an Assistant Professor at Texas Tech University. Their major research interests include identifying the roles that families serve in the development of social, cognitive, and neural processes that support adolescent well-being.

Virnaliz Jimenez is a doctoral student at the University of Illinois at Urbana–Champaign. Her major research interests include examining adolescent adjustment and resilience in Black and Latinx families.

Amanda Benjamin is a doctoral student at John Jay College of Criminal Justice, City University of New York. Her major research interests include understanding juvenile law and evidence-based policy reform.

Karen Rudolph is a Full Professor at the University of Illinois at Urbana–Champaign. Her major research interests include identifying risk and protective processes that amplify or attenuate vulnerability to psychopathology across development, particularly during adolescence.

Eva Telzer is an Associate Professor at the University of North Carolina at Chapel Hill. Her major research interests include examining how social and cultural processes shape adolescent brain development, with a focus on both prosocial and antisocial behaviors, family and peer relationships, and long-term psychological well-being.