Research on Adolescence



JOURNAL OF RESEARCH ON ADOLESCENCE, 30(3), 599-615

Differential Behavioral and Neural Profiles in Youth With Conduct Problems During Risky Decision-Making

Jorien van Hoorn, and Ethan M. McCormick D University of North Carolina at Chapel Hill Michael T. Perino D
Washington University at Saint Louis

Christina R. Rogers
Texas Tech University

Eva H. Telzer University of North Carolina at Chapel Hill

Neuroimaging work has examined neural processes underlying risk taking in adolescence, yet predominantly in low-risk youth. To determine whether we can extrapolate from current neurobiological models, this functional magnetic resonance imaging study investigated risk taking and peer effects in youth with conduct problems (CP; N = 19) and typically developing youth (TD; N = 25). Results revealed higher real-life risk taking, lower risky decisions, and no peer effects on a risk-taking task in CP youth. CP youth showed greater ventral striatum (VS) activity during safe than risky decisions, whereas TD youth showed greater VS activation during risky decisions. Differential VS activity explained higher real-life risk taking in CP youth. Findings provide preliminary evidence that risk-taking behavior in youth with CD problems is characterized by differential neural patterns.

Adolescents are characterized by remarkable improvements in physical, mental, and social capabilities, while at the same time uniquely heightened levels of risk-taking behaviors that contribute to a drastic increase in morbidity and mortality (CDC; Heron, 2017; Defoe, Dubas, Figner, & Aken, 2015). This increase in health-risk behaviors such as risky driving, delinquency, and substance use is related to heightened sensation-seeking tendencies at a time when self-regulation is still maturing (Steinberg et al., 2017). Moreover, in the presence of peers, adolescents tend to take even greater risks (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011; Gardner & Steinberg, 2005). Despite the dramatic increase in morbidity and mortality, the survival rates of US high school students are at 99.96% (Willoughby, Good, Adachi, Hamza, & Tavernier, 2014). Most teenagers navigate through adolescence with heightened, albeit non-health-compromising, levels of risk taking, which can be beneficial for development, for example in forming friendships or becoming more independent from parents (Crone & Dahl, 2012; Crone, Duijvenvoorde, & Peper, 2016). Only a small percentage of teenagers engage in persistently high levels of risky and antisocial behaviors, often at great cost to society (Moffitt, 2018). Yet, most of what we know about adolescent risk taking is based on community adolescent samples engaging in low rates of health-compromising risk taking (Bjork & Pardini, 2015). Current neurobiological theories emphasize an imbalance between hyperactive reward systems (e.g., ventral striatum, VS) and protracted development of cognitive control systems (e.g., prefrontal cortex, PFC) to explain increased orientation to risk and reward during adolescence (Casey, 2015; Shulman et al., 2016; Steinberg, 2008). However, without including youth who engage in excessive rates of risk taking, our current neurobiological theories may not generalize broadly outside of normative samples. The present study employed functional magnetic resonance imaging (fMRI) to investigate behavioral and neural profiles of risk taking in a sample of youth characterized by conduct problems (CP) severe enough to result in institutional recourse.

Excessive risk taking is clustered in a small number of adolescents characterized by CP, which include behaviors such as aggression, property damage, and stealing (reviewed in Blair, Veroude,

This work was supported by grants from the National Institutes of Health (R01DA039923) and National Science Foundation (SES1459719) awarded to EHT and National Institute of Mental Health (2T32MH100019-06) awarded to MTP.

We greatly appreciate the assistance of the Biomedical Imaging Center at the University of Illinois as well as Elizabeth Lozano, Kathy Do, Heather Ross, Lynda Lin, Paul Sharp, Susannah Ivory, and Tae-Ho Lee for assistance in collecting data.

Requests for reprints should be sent to Eva H. Telzer, Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill, 235 E. Cameron Avenue, Chapel Hill, NC 27514. E-mail: ehtelzer@unc.edu

& Buitelaar, 2018). The term conduct problems has been used interchangeably with other terms, such as externalizing behaviors and antisocial behaviors (Hyde, Shaw, & Hariri, 2013; Liu, 2005), and research describes a tremendous heterogeneity in youth with CP (e.g., callous-unemotional traits, impulsivity, antisocial peers; reviewed in Moffitt, 2018; Viding, Fontaine, & McCrory, 2012). In excessive forms, conduct problems are defining features of oppositional defiant disorder (ODD) or conduct disorder (CD) (DSM-5; American Psychiatric Association, 2013; Blair et al., 2018). Here, we define conduct problems as non-normative antisocial and health-risk behaviors (e.g., aggression, property damage, stealing, and delinquency) that have resulted in institutional involvement (arrest, school suspensions, and expulsions) as these are seen as strong predictors of continuing problems into adulthood (Moffitt & Caspi, 2001), as compared to community adolescents with normative levels of risk behavior (e.g., sneaking out of the house without parents knowing). In the current study, we included adolescents based on their behavioral profile of conduct problems, rather than clinical diagnosis, because individuals may not receive a diagnosis due to limited access to clinical resources (see Blair et al., 2018), or because they do not meet all diagnostic criteria, even though their conduct problems are substantial enough to result in institutional involvement.

Two lines of research provide some insight into the potential neural correlates of risk-taking behavior in youth with conduct problems, (1) research in developmental samples examining broadly defined risk-taking behavior and (2) youth with ODD, CD, and addiction (for reviews, see Bjork & Pardini, 2015; Blair et al., 2018; Hyde et al., 2013; Rubia, 2011; Viding et al., 2012). Research largely utilizing community samples of adolescents has shown that risk-taking behavior is associated with an interplay of affective and social-cognitive processes in the brain. Indeed, a recent meta-analysis of typically developing youth found that dorsomedial prefrontal cortex (dmPFC), inferior frontal gyrus (IFG)/insula, and VS are consistently implicated in decision-making in social contexts (Van Hoorn, Shablack, Lindquist, & Telzer, 2019). Neural activation in these regions, particularly in the VS, peaks during adolescence (Galvan, 2010; Sherman, Chein, & Steinberg, 2018; Silverman, Jedd, & Luciana, 2015; Telzer, 2016), especially when peers are present (Chein et al., 2011). Empirical work suggests that increased VS activity in response to rewards is stronger for adolescents with a higher motivation

to obtain rewards (Schreuders et al., 2018) and greater likelihood of engaging in real-life risky behaviors (Galvan, Hare, Voss, Glover, & Casey, 2007; Qu, Galvan, Fuligni, Lieberman, & Telzer, 2015), underscoring that there are meaningful individual differences in affective reactivity that could explain excessive rates of risk-taking behavior.

In the second line of work, processes implicated in risky behaviors, such as reward and punishment processing, have been examined in adolescents with CD, ODD, and addiction. This research has identified deficits in CD and ODD in "hot" ventromedial orbitofrontal-limbic regions associated with motivation and affect (Bjork & Pardini, 2015; Hyde et al., 2013; Rubia, 2011). However, there has been mixed evidence for the direction of these differences in conduct disorders, that is, hyperactivation or hypo-activation of the reward system (reviewed in Byrd, Loeber, & Pardini, 2014; Tyler, White, Thompson, & Blair, 2019). For example, adolescents with antisocial behavior increased neural activation to rewards in the VS and OFC (Bjork, Chen, Smith, & Hommer, 2010), whereas youth with CD display attenuated activation in OFC during reward tasks (Finger et al., 2009; Rubia et al., 2009). Theories of atypical brain functioning in addiction further postulate the reward-deficiency hypothesis (Blum et al., 2000), which suggests that hypoactivation of reward regions may lead to more extreme risky behaviors (i.e., more excessive risks to get the same "rewarding" feeling). Finally, another recent study in delinquent adolescents illustrated a qualitatively different pattern of neural activation in the delinquent group rather than hypo- or hyperactivation. Delinquent youth recruited cognitive control regions (e.g., IFG) more in the presence of aversive cues than appetitive cues, while this pattern was reversed in the community sample (Perino, Guassi Moreira, McCormick, & Telzer, 2019). Taken together, important progress has been made in elucidating differential neurobiological profiles in youth with conduct problems, but the literature currently provides inconsistent findings regarding the direction and nature of these differences, requiring further research on this important topic.

Present Study

The goal of this study was to investigate the behavioral and neural profiles of risk taking and peer effects in a sample of adolescents with conduct problems and a sample of typically developing adolescents. During an fMRI scan, adolescents between 13 and 16 years completed a computerized risk-taking task (the Yellow Light Game; YLG;

Op de Macks et al., 2018; Pfeifer, 2015) alone and in the presence of a peer, during which they could make risky or safe choices. At the behavioral level, we expected that youth with CP would display a higher frequency of health-risk behaviors in daily life, as self-reported on the Adolescent Risk Taking Scale (Alexander, Esminger, & Johnson, 1990). Similarly, we expected higher rates of risk taking on the computerized risk-taking task, as work with other risky decision-making tasks such as the Balloon Analogue Risk Task (BART) and Iowa Gambling Task (IGT) highlights that children and adolescents with conduct disorders engage in greater risk-taking behavior on these tasks than their typically developing counterparts (Crowley, Raymond, Mikulich-Gilbertson, Thompson, Lejuez, 2006; Humphreys & Lee, 2011; Schutter, Van Bokhoven, Vanderschuren, Lochman, & Matthys, 2011).

Moreover, we expected that both groups would make more risky decisions in the presence of a peer relative to alone, given prior evidence for greater risk taking in the presence of peers (e.g., Chein et al., 2011; but see e.g., Somerville et al., 2019 for alternative results). While teens with behavioral problems tend to engage more with other deviant peers, and deviant talk between friends can reinforce risky behaviors (Dishion & Tipsord, 2011; Richmond, Laursen, & Stattin, 2019), it is unclear if youth with conduct problems are more or less influenced by peers than typically developing youth. Adolescents with poorer selfregulation and greater antisocial behavior appear to be more vulnerable to the influence of deviant peers on antisocial behavior (Gardner, Dishion, & Connell, 2008; Goodnight, Bates, Newman, Dodge, & Pettit, 2006). On the other hand, previous neuroimaging work has revealed that adolescents with CD differentiate less between emotional signals of peers, which may indicate that this group may be less influenced by peers during decision-making (Klapwijk et al., 2016).

At the neural level, we examined whether the group with conduct problems would show higher levels of reward-related processing (VS/vmPFC/ OFC) consistent with neurobiological models of risk taking (Casey, 2015; Shulman et al., 2016; Steinberg, 2008), lower levels of reward-related processing consistent with the hypo-activation account (Blum et al., 2000), or qualitatively different patterns of activation (heightened reward-related processing to different stimuli) as found in previous work on the cognitive control system in a delinquent sample (Perino, Guassi Moreira, et al., 2019).

METHOD

Participants

The total sample consisted of 44 adolescents $(M_{\rm age}(SD) = 14.47(0.85), \text{ range } 13.03-15.94 \text{ years, } 16$ females), including 19 youth with conduct problems (n = 12 males; 63%), and an age- and gendermatched typically developing sample (n = 25; n = 16 males; 64%). One additional participant with conduct problems was scanned but excluded due to technical difficulties in acquiring data. Table 1 shows an overview of descriptives separately for the CP and typically developing group. Our sample size is similar to other studies that examined clinical or high-risk populations (see Blair et al., 2018, for a review on clinical samples; see, e.g., Cservenka & Nagel, 2012, for a study of high-risk youth).

Youth with conduct problems were recruited from an alternative school for students who have been multiply suspended or were expelled from school for acts of gross legal and school misconduct. As such, we included this group based on their behavioral profile of school and legal misconduct, rather than clinical diagnosis. No information about clinical diagnosis was available for these youth. The typically developing sample was recruited via local schools, community flyers, and listservs.

We screened all participants to ensure they were free from neurological disorders, taking psychotropic medication, or any MRI contraindications. Typically developing participants were accompanied to the scan by their primary caregiver, whereas participants with conduct problems came during school time were accompanied by the school counselor. Aside from consent and answering questions, the parent and school counselor were not present when the task, fMRI scan, or questionnaires were administered. All participants and their legal guardians provided written consent and assent, and the Institutional Review Board approved all procedures.

Questionnaires

Adolescent risk taking. Adolescents completed a modified version of the Adolescent Risk Taking Scale (ART; Alexander et al., 1990) to measure how often they engaged in risky behaviors. Adolescents responded to 12 items using a four-point scale (0 = never, 1 = once or twice, 2 = several times,and 3 = many times) to indicate the frequency with which they have engaged in each risky behavior. Risky behaviors included were the following: raced

TABLE 1
Descriptive Characteristics of Youth With Conduct Problems and Typically Developing Sample and Statistical Tests

	Conduct Problems (CP) N = 19	Typically Developing (TD) N = 25	Statistical Tests	
M(SD) age	14.52 (0.87)	14.45 (0.84)	t(42) =27, p = ns	
Gender (% Males)	63%	64%	$\chi(1) = 0.00, p = ns$	
Ethnicity			•	
Hispanic	5%	0%	$\chi(1) = 9.02, p = .003$	
Black	42%	8%		
White	26%	72%		
Mixed	26%	20%		
Maternal education				
Some high school	22%	0%	t(41) = 4.86, p < .001	
Graduated high school	17%	0%		
Trade or vocational school	11%	0%		
Some college	22%	24%		
Graduated from college	22%	48%		
Graduated from med, law, or graduate school	6%	28%		
M % arrested in the past	37%	0%	$\chi(1) = 10.95, p = .001$	
M(SD), range conduct problems	2.47 (1.81)	1.16 (1.40)	t(42) = -2.716, p = .010	
	0–6	0–5	•	
M(SD), range frequency engaging with deviant peers	1.91 (0.73) 1.07–3.80	1.31 (0.29) 1.00–2.07	t(42) = -2.716, p = .001	

Note. M = mean; SD = standard deviation.

Maternal education was obtained as an indication of SES for n = 43 (n = 1 was missing for youth with conduct problems).

a car or motorcycle, did something risky or dangerous on a dare, broke a rule that their parents set just for the thrill of seeing they could get away with it, stole or shoplifted, snuck out of the house without their parents knowing, willingly rode in a car with someone who was a dangerous driver, drove in a car without a seatbelt, had sex with someone they just met, dated or hooked up with someone else's boyfriend/girlfriend, cheated on an examination or homework, cheated on a boyfriend/girlfriend, and got drunk or high at a party. Item scores were averaged to compute an overall score, with higher scores indicating a greater frequency of real-life risk-taking behavior. The modified ART scale had an adequate internal consistency (total sample α = .795; TD α = .733; CP $\alpha = .793).$

Conduct problems. Adolescents completed the Strengths and Difficulties Questionnaire (Goodman, 1997), a widely validated scale that measures common areas of emotional and behavioral difficulties with five subscales. Conduct problems were assessed with five items. Adolescents indicated the extent to which each statement was true on a 3-point Likert scale (0 = not true, 1 = somewhat true, and 2 = certainly true). Examples include "I fight a lot" and "I can make other people do what I want." Item

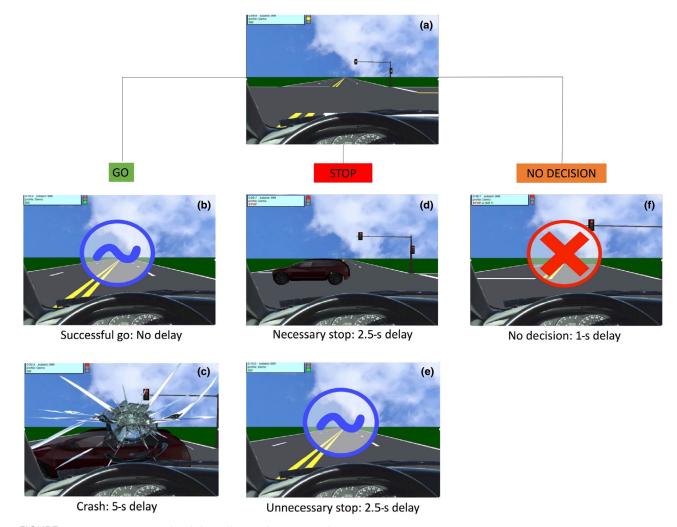
scores are summed to generate total scores on the subscales, with higher scores indicating greater behavioral difficulties. After removal of one reverse-coded item "I usually do as I am told," which had a low item-total correlation (r = .112), the conduct problems scale had a satisfactory internal consistency (total sample α = .645; TD α = .593; CP α = .654). In addition, mean interitem correlations were inspected for both groups, as this measure is more informative than α when a scale only has a few items (Piedmont, 2014). Mean interitem correlations were r = .323 for both groups, which fell in between the recommended range of 0.20–0.40 and confirmed that the scale was suitable for use with both groups.

Engaging with deviant peers. Adolescents reported on how often their friends engaged in deviant and prosocial activities within the last month (adapted from Barrera et al., 2002; see Telzer, Gonzales, & Fuligni, 2014). Participants indicated how many of their friends in the past month engaged in deviant behaviors (e.g., "Got suspended from school" and "Started a fight with someone"). The deviant subscale encompasses 15 items with a 5-point Likert scale ranging from "None" to "Almost all" of their friends (1 = None, 2 = very few, 3 = some, 4 = most, and 5 = Almost all). Items

scores are averaged to obtain a total score, with higher scores indicating a higher frequency of engaging with deviant friends. This scale had high internal consistency (total sample $\alpha = .899$; TD $\alpha = .711$; CP $\alpha = .913$).

Risk-taking paradigm. The YLG (Op de Macks et al., 2018; Pfeifer, 2015) is an adaptation of the Stoplight Task (Chein et al., 2011; Gardner & Steinberg, 2005) that examines risk taking at the behavioral and neural level. In the YLG, participants are asked to drive a virtual car from the driver's point of view along a straight track, during which they encounter several intersections with yellow lights (Figure 1a). They were instructed that the goal of the game was to get through all of the intersections in the shortest amount of time. At each intersection, participants had to indicate by button press whether they wanted to accelerate and go through the yellow light (go decision) or brake before arriving at the intersection (stop decision). Deciding to accelerate through the intersection—a go decision—constitutes a risky decision and could result either in a successful go associated with no delay (i.e., if there was no other car passing through the intersection) or in a delay of 5 s in the event of a crash (i.e., if there was another car passing through the intersection). A successful go was shown on the screen with a blue tilde and a positive chiming sound (Figure 1b), whereas a crash was shown as a cracked car window, honking car, and crash sound (Figure 1c). At the behavioral level, we assessed risk taking as the percentage of go decisions out of the total decisions made (i.e., stop and go were exact opposites).

Braking before the intersection—a stop decision —resulted in a 2.5-s delay and constitutes a safe decision because participants avoided a potential crash. After a stop decision, participants saw either an approaching car and heard a honking noise



Participants completed the Yellow Light Game in the scanner.

604

(i.e., necessary stop, because going would have resulted in a crash; Figure 1d) or an empty intersection and blue tilde with a positive chiming sound (i.e., unnecessary stop, because going would have been successful; Figure 1e). At the behavioral level, safe decisions were defined as the percentage of stop decisions out of the total decisions made. Finally, if participants did not make a decision in time, this resulted in a 1-second delay, a red X on the screen, and an error noise (Figure 1f).

Participants played the YLG in the MRI scanner while alone and in the presence of a peer. Participants completed two runs of 20 intersections for each condition, totaling 80 trials across the task. The onset of the yellow light was 1,500 ms after the previous trial, which corresponded to a varying distance on the track (200-250 ft). While the runs were varied in terms of probability of crashes for each intersection, the probability of crashing was kept at 50% for each run, which prevented the task from promoting risk taking, as there was no overall advantage to either taking risks or playing it safe (Op de Macks et al., 2018). All participants completed the exact same runs in the scanner for the sake of consistency. Before entering the scanner, participants were trained on how to properly play the task by completing two practice runs, in order to account for learning effects (Kahn, Peake, Dishion, Stormshak, & Pfeifer, 2015). The practice runs were based on the same parameters as the scanner task, but unbeknownst to the participants, was slightly different in terms of the no-decisions. To train participants from not responding, no-decisions were paired with a larger 5-s delay in the practice runs.

Manipulation of Peer Context

When participants arrived to the scan, they were shown a picture of an age-, race-, and gendermatched peer accompanied by a profile that a confederate peer had completed. The participant was told that the peer was currently completing their brain scan and would be playing the same YLG as the participant. After participants practiced two rounds of the YLG themselves, the researcher pretended to communicate via cell phone to the scan technician, who indicated that the peer was ready to begin the task in the scanner. The participant was then given a notecard to read to the peer, which they said through the cell phone: "Hi, this is [PARTICIPANT'S NAME], and I'll be watching you play this round." The participant then saw a "live feed" of the peer supposedly playing the YLG

in the scanner, which was in fact a recording of a game set to be a representative teen's behavior (choosing to go approximately 50% of the time; Kahn, Peak, Dishion, Stormshak, & Pfeifer, 2015; "average" peer in Peake, Dishion, Stormshak, Moore, & Pfeifer, 2013).

During their own fMRI scan, participants completed the YLG alone and in the presence of the peer. In the *Peer* condition, the participant was told that the same peer would be watching them play now. The experimenter then played an audio recording of the same script from this supposed peer, telling the participant they were watching them play this round, a method we have employed in prior studies to examine social influence on risk taking (e.g., Guassi Moreira & Telzer, 2018; Telzer, Ichien, & Qu, 2015). The Alone and Peer conditions were counterbalanced across participants. After the scan, we asked all participants about their experience with the task and the peer, and no participant mentioned that they did not believe it or were suspicious.

fMRI Data Acquisition

Data were collected with a 3-T Siemens Trio MRI Scanner, using a 32-channel head coil. The task was presented on a computer screen, which participants could see through a mirror attached to the head coil. We obtained the functional data using T2*-weighted echoplanar images (EPI) (slice thickness = 3 mm; 38 slices; TR = 2 s; TE = 25 msec; $matrix = 92 \times 92$; FOV = 230 mm; voxel $2.5 \times 2.5 \times 3$ mm³). Structural scans were also acquired, including a T2*-weighted, matched-bandwidth (MBW; TR = 4 s; TE = 64 ms; FOV = 230; $matrix = 192 \times 192$; slice thickness = 3 mm; 38 slices) and a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1.9 s; TE = 2.32 ms;FOV = 230;matrix = 256×256 ; sagittal acquisition plane; slice thickness = 0.9 mm; 192 slices). MBW and EPI scans were collected with an oblique axial orientation to prevent signal dropout in orbital and temporal regions, thereby maximizing coverage of the brain.

fMRI Data Preprocessing and Analysis

Standard preprocessing was conducted using the FSL FMRIB's Software Library (FSL v6.0; https://fsl.fmrib.ox.ac.uk/fsl/). Data were first skull-stripped with BET (Smith, 2002). We corrected for slice-to-slice head motion using MCFLIRT (Jenkinson, Bannister, Brady, & Smith, 2002), image coregistration was done using a three-step registration procedure

(EPI to T2 to T1), and each functional image was resampled to $2 \times 2 \times 2$ mm and warped to the standard Montreal Neurological Institute 2-mm brain using FLIRT (Jenkinson et al., 2002; Jenkinson & Smith, 2001). Images were then spatially smoothed with a 6-mm FWHM Gaussian kernel, and a highpass temporal filtering with a 128-s cutoff was applied to remove low-frequency drift across time (Gaussian-weighted least squares straight line fitting; sigma = 64.0 s). Moreover, to remove artifact signals such as motion and physiological noise, we applied an independent component analysis (ICA) denoising procedure using MELODIC (Beckmann & Smith, 2004), combined with an automated signal classification toolbox (an average of 8.96% components was removed; classifier NP-threshold = 0.3; for more details, see Tohka et al., 2008).

After preprocessing, statistical analyses were conducted on the individual subjects' data using the general linear model in the Statistical Parametric Mapping software package (SPM8; Welcome Department of Cognitive Neurology, London). Each trial was convolved with the canonical hemodynamic response function. The YLG was modeled as an event-related design. In the fixed-effect model, we included two decision regressors (go and stop decisions); four outcome regressors (successful go (i.e., no car approaching), crash, necessary stop (i.e., car approaching), and unnecessary stop (i.e., no car approaching)); as well as no decisions, which were modeled in a separate junk regressor together with volumes that contained excessive motion. The time when participants were driving on the course between the intersections was modeled as an implicit baseline and controlled for basic visual characteristics. These regressors were estimated separately for the Alone and Peer conditions.

The decision phase (go and stop) was modeled with the onset of the yellow light and duration of their decision (i.e., when participants made a button press to either go or stop). The outcome phase for go decisions was modeled from either the onset of blue tilde (successful go) or onset of the crash (unsuccessful go), each with a 2.5-s duration, in order to make the outcome equitable across conditions. The outcome phase for stop decisions was modeled from the onset of the stopped car and had a duration of 2.5 s for both necessary and unnecessary stops. Note that we included the four possible outcomes in our model, but do not report on the outcome phase in the current paper, because we were specifically interested in the effects of decision-making (i.e., stop and go decisions). Participants had to have at least 20% of trials in each condition (i.e., go decision and stop

decision) in each run to be kept in neural analyses, based off of previous work using the same paradigm (Rogers, McCormick, van Hoorn, Ivory, & Telzer, 2018; Van Hoorn, McCormick, Rogers, Ivory, & Telzer, 2018). This cutoff resulted in exclusion of n=3 participants from the group with CP, which left us with N=16 in our neural analyses.

The resulting contrast images, computed at the individual level, were submitted to random-effects group-level analyses, in which we computed independent-samples *t*-tests to compare the two groups. In the current study, our main contrast of interest was go–stop decisions to examine neural activation that differed between the two groups when they made risky relative to safe decisions. We ran additional analyses to examine the peer effects for go and stop decisions separately (i.e., peer-alone go decisions; peer-alone stop decisions). We report exploratory regression analyses on the go–stop contrast using continuous CP and real-life risk taking in the supplement (see Table S1 and Figure S1).

At the group level, we conducted analyses on contrasts of interest using GLM Flex, which removes outliers and sudden activation changes in the brain, partitions error terms, analyzes all voxels containing data, and corrects for variance-covariance inequality (http://mrtools.mgh.harvard.edu/index.php/GLM Flex). We corrected all analyses for multiple comparisons using Monte Carlo simulations through 3DClustSim (updated version November 2016) in the software package AFNI (Ward, 2000) and accounted for the smoothness of the data with the acf function within the 3dFWHMx command. For the main effects in the go-stop contrast, the simulation resulted in a voxel-wise threshold of p < .005and minimum cluster size of 63 voxels for the whole brain, which corresponds to p < .05, FWE clustercorrected. For the main effects in the go and stop peer-alone contrast, the simulations resulted in a voxel-wise threshold of p < .005 and minimum cluster size of 51 (go) and 70 (stop) voxels for the whole brain. Given our a priori hypotheses of activation in the VS, an anatomically small structure which typically does not survive stringent correction, we applied a small-volume correction for the VS, with a voxel-wise threshold of p < .005, and minimum cluster size of k = 20 (Giuliani & Pfeifer, 2015; Guassi Moreira & Telzer, 2018)¹. All reported results are available on NeuroVault (Gorgolewski et al., 2015; see/collections/BUYSSCLX/).

¹We ran a Monte Carlo simulation on a VS structural mask, which resulted in a minimum cluster size of 3 voxels. We used a minimum cluster size of 20 voxels to be more conservative.

RESULTS

Behavioral Analyses

First, we examined differences in real-life risk taking between youth with conduct problems (CP) and typically developing youth (TD), as measured by the ART scale. An independent-samples t-test demonstrated that CP youth showed a higher frequency of real-life risk-taking behavior ($M_{\rm CP}(SD) = 0.74(0.42)$, range 0.08-1.50) than typically developing youth ($M_{\rm TD}(SD) = 0.40(0.32)$, range 0.00-1.33; t(42) = -3.075, p = .004).

Next, we examined risk-taking behavior on the task. The mean percentages of go decisions in the Alone and Peer condition are displayed in Figure 2, separately for the CP and typically developing youth. Kolmogorov-Smirnov tests of normality showed that the percentage of go decisions in the alone condition was not normally distributed in the CP youth, D(19) = .216, p = .02. Therefore, we used nonparametric tests to assess the between- and within-subjects effects, as transforming the data did not solve this issue. The Mann-Whitney U-test for between-subjects effects showed significant differences between the CP group and the TD group, (p < .001), revealing that the CP group displayed a lower percentage of go decisions across the alone $(M_{\rm TD}(SE)=53.54(2.71),$ peer conditions $M_{\rm CP}(SE) = 34.72(3.10)$). Friedman's tests for withinsubjects effects showed that the percentage of go decisions were significantly different between the condition and peer in TD $(M_{\text{alone}}(SE) = 56.44(3.01), M_{\text{peer}}(SE) = 50.64(2.72), p$ =.007), such that they showed a risk-reducing peer effect, while CP youth did not show any differences between the alone and peer condition $(M_{\text{alone}}(SE) = 34.09(3.45),$ $M_{\text{peer}}(SE) = 35.36(3.12),$ $p = .637)^2$.

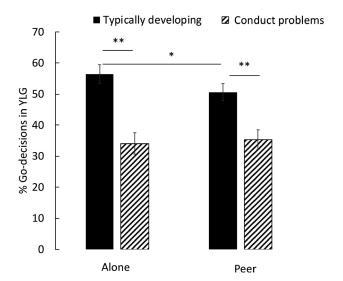


FIGURE 2 Risk-taking behavior during Yellow Light Game as measured by % of go decisions in Alone and Peer for the typically developing sample (black bars) and conduct problems sample (striped bars). Error bars indicate standard error of the mean (± 1 SEM). *p < .05 **p < .001.

Finally, in CP youth, greater real-life risk-taking behavior was associated with higher levels of risk taking on the task (i.e., percentage go decisions) in the alone condition, r(17) = .502, p = .029, but not the peer condition, r(17) = .314, p = .190. For the typically developing teens, real-life risk-taking behavior was not related to behavior on the task (ps ns).

Next, we compared reaction times between the groups in the alone and peer condition, for go and stop decisions separately. For go decisions, a main effect of Group (F(1,40) = 11.749, p < .001) indicated that youth with CP were slower to choose to go through the intersection $(M_{CP} (SE) = 1.009 (0.053))$ developing typically $(M_{TD}(SE) = 0.774 (0.044))$. For stop decisions, which included all N = 19 in the CP group, we found a main effect of Group (F(1,42) = 9.040, p = .004),revealing that the CP group was slower to choose to stop at the intersection $(M_{CP} (SE) = 0.795 (0.042);$ $M_{TD}(SE) = 0.998$ (0.048)). Results also indicated a trend-level interaction of Group X Condition, F (1,42) = 3.616, p = .064, which was qualified by a trend in the typically developing group, such that this group tended to be slightly faster in the Peer condition than Alone condition $(M_{\text{ALONE}}(SE) = 0.823$ (0.046), $M_{\text{PEER}}(SE) = 0.768$ (0.045) p = .09). Together, these findings suggest that the CP group was more cautious, as indexed by fewer risky decisions and longer reaction times, effects that did not vary across peer and alone conditions for the CP group.

²A RM ANOVA that compared the percentage of go decisions in the alone and peer conditions in the two groups using a 2 (Group: Conduct Problems (CP), Typically developing (TD)) x 2 (Condition: Alone, Peer) design, similarly yielded an interaction effect that was driven by a difference between go decisions in peer and alone for the TD group. Given the significant difference in distribution of ethnicities as well as maternal education among the typically developing and youth with conduct problems (see Table 1), as a check we also ran a RM ANOVA with ethnicity (white or other) as a covariate and a separate RM ANOVA with maternal education as a covariate, because including covariates is not possible with nonparametric tests using SPSS. Both ethnicity and maternal education showed no significant main or interaction effects, and the significant group differences in behavior were unchanged. We therefore did not include these controls in further analyses.

fMRI Analyses

Group differences in VS response during go-stop decisions. At the neural level, we first collapsed our analyses across the alone and the peer conditions to test neural differences when making risky (i.e., go) and safe (i.e., stop) decisions, given that the CP youth showed lower overall risk taking. We examined the main effects of the go-stop contrast using a 2-sample *t*-test to compare the two groups. We observed greater activity in the VS in the typically developing sample than the youth with CP (Table 2). For descriptive purposes, we extracted parameter estimates of signal intensity from the VS cluster, separately for go-baseline and stop-baseline in each group, respectively (see Figure 3). Typically developing adolescents showed greater VS activity during go decisions than stop decisions, whereas this pattern was reversed for youth with CP, such that these adolescents showed greater VS activity to stop than go decisions. In addition to the VS, group differences in neural activation for the gostop contrast yielded greater activation in the typically developing youth in left anterior cingulate cortex, right fusiform as well as motor areas and cerebellum, and greater activation in the youth with CP in the dmPFC (see Table 2 for all regions).

Next, we examined whether group differences in real-life risk-taking behavior were mediated by neural activation in the VS in the typically developing and CP sample (see Figure 4). In other words, do group differences in VS activation explain the variance in group differences in real-life risk taking? This mediation was run in the PROCESS macro in SPSS. We extracted the parameter estimates from the VS for the go-stop contrast. Group was dummy-coded, with the typically developing (TD) sample coded as 0 and the CP sample coded as 1. Findings indicated a significant indirect effect, such that CP youth displayed greater real-life risk taking via altered VS activity during go-stop decisions, & = .1881, SE = .0951, 95% CI [0.0178, 0.3994].

Group differences in VS response during decisions in peer-alone. We examined group differences during go and stop decisions in the presence of a peer versus alone using a 2-sample *t*-test. For stop decisions, TD youth showed greater activity in the temporal pole than CP youth, while CP youth displayed greater activity in the IFG (see Table 3). For go decisions, TD youth showed greater activation than CD youth in the VS, supplementary motor area, cerebellum, and inferior temporal gyrus (see

TABLE 2
Brain Regions for Main Effects of Go–Stop Across Conditions in Youth With Conduct Problems (CP) and Typically Developing (TD) Sample at p < .005, FWE Cluster-Corrected (k = 63)

MNI Coordinates Z Region k t-value y Go-Stop TD > CP 43a -8 R Ventral Striatum 3.958 R Fusiform Gyrus 222 4.354 34 -62-10173 28 -42R Cerebellum 3.696 -4679 32 L ACC 4.105 -4-2L Postcentral Gyrus 104 3.804 -58-1824 L Precentral Gyrus 3.717 -32-1450

Note. CP = conduct problems sample; TD = typically developing sample; L = left; PFC, prefrontal cortex; R = right; k = number of contiguous voxels in mm³.

-3.947

-6

36

42

100

Go-Stop CP > TD

Dorsomedial PFC

Table 3). There were no regions more active in the CP youth than in the TD youth. For descriptive purposes, we extracted parameter estimates of signal intensity from the VS cluster, separately for go decisions in the alone condition and the peer condition for each group (see Figure 5). Typically developing adolescents showed greater VS activity during go decisions in the presence of peers than alone, whereas for youth with CP, this pattern was reversed, such that these adolescents showed greater VS activity during go decisions alone than in the presence of peers. Finally, we computed a mediation model to examine whether group differences in real-life risk-taking behavior were mediated by VS activation from the go peer-alone contrast. Findings indicated a nonsignificant indirect effect, suggesting that differences in VS activity during the presence of peers relative to alone did not mediate group difin real-life risk taking, SE = .1677, 95% CI [-0.1280, 0.5514].

DISCUSSION

This study aimed to elucidate the behavioral and neural profiles of risk taking and peer effects in a sample of adolescents characterized by school and legal misconduct. CP youth self-reported higher rates of conduct problems, greater real-life risk taking, and engaging with deviant peers, and about 40% had been arrested in the past compared to their typically developing peers who had lower rates of these behaviors. However, youth with

T-value is at local maximum.

^aSmall-volume correction p < .005, k = 20.

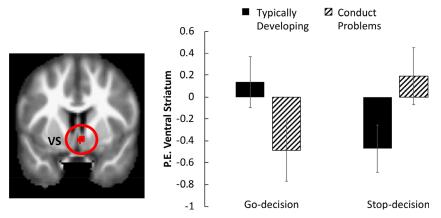


FIGURE 3 Ventral striatum (VS) activity when adolescents took risks (go decision) and played it safe (stop decision) for typically developing (black bars) and conduct problems sample (striped bars). For descriptive purposes, parameter estimates (P.E.) were extracted from Go > baseline and Stop > baseline. Error bars represent standard error of the mean (± 1 SEM). Thresholded at p < .005, small-volume-corrected (k = 20).

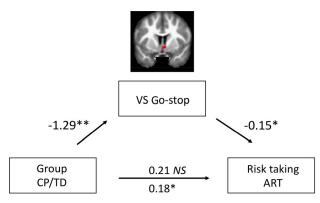


FIGURE 4 Mediation model examining the association between group and real-life risk taking. Activation in the ventral striatum (VS) during go—stop decisions was modeled as a mediator between group (typically developing [TD; coded as 0] and conduct problems [CP; coded as 1]) and frequency of risk-taking behaviors as assessed by the Adolescent Risk Taking scale. Coefficients are unstandardized. *p < .05, **p < .001.

conduct problems showed lower levels of risk taking on the computerized driving task and no effect of being observed by a peer. At the neural level, youth with conduct problems showed greater VS activity during safe decisions relative to risky decisions, whereas typically developing youth showed the reverse pattern—greater VS activity during risky decisions than safe decisions. Moreover, youth with conduct problems showed greater VS activity during risky decisions alone than with a peer, whereas typically developing youth again showed the reverse pattern. Taken together, our findings suggest that rather than hyper- or hypoactivation of reward-related neural processing, youth with conduct problems show a differential behavioral and neural profile of risk taking with

TABLE 3
Brain Regions for Main Effects of Peer-Alone Go and Stop in Youth With Conduct Problems (CP) and Typically Developing (TD) Youth at p < .005, FWE Cluster-Corrected (k = 51 for Go Decisions and k = 70 for Stop Decisions)

Region	t-value	K	MNI Coordinates			
			x	у	Z	
Peer-Alone go TD> CP						
Ventral striatum	3.503	52	6	16	-16	
Supplementary motor area	5.959	109	10	32	62	
L cerebellum	5.062	436	-8	-58	-12	
R cerebellum	4.957	153	36	-72	-22	
L inferior temporal gyrus	4.442	96	-50	-10	-32	
Peer-Alone go CP> TD	No significant regions					
Peer-Alone stop TD> CP						
R temporal pole	3.236	107	38	22	-34	
Peer-Alone stop CP> TD						
R inferior frontal gyrus	-3.274	102	42	8	30	

Note. CP = conduct problems; TD = typically developing sample; L = left; R = right; k = number of contiguous voxels in mm³. T-value is at local maximum.

and without peers, which highlights the need to explicitly recruit high-risk samples to inform our understanding of adolescent neurocognition.

Recent theoretical perspectives and empirical work have started to emphasize that the increase in health-risk behaviors during adolescence, while troublesome, is to some extent normative and beneficial to reach developmental milestones (Crone & Dahl, 2012; Van Duijvenvoorde, Peters, Braams, & Crone, 2016). However, when normative risk behaviors, like sneaking out of the house without parents knowing, turn into excessive risk taking, including aggression, stealing, and property damage, this is

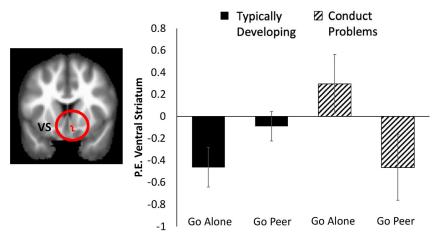


FIGURE 5 Ventral striatum (VS) activity when adolescents took risks (go decision) in the presence of a peer or alone for typically developing (black bars) and conduct problems sample (striped bars). For descriptive purposes, parameter estimates (P.E.) were extracted from Go Alone > baseline and Go Peer > baseline. Error bars represent standard error of the mean (± 1 SEM). Thresholded at p < .005, FWE cluster (k = 51).

associated with negative psychosocial outcomes and major costs for society (Bjork & Pardini, 2015; Moffitt, 2018). Previous developmental neuroscience work assessing risk taking has either typically studied adolescents formally diagnosed with conduct disorders or typically developing low-risk adolescents, from which findings are subsequently extrapolated to youth who take more excessive risks. Yet, this leaves out a substantive subgroup of adolescents with conduct problems who experience behavioral difficulties and take excessive risks. This is especially relevant because in studies of typically developing youth, participants are often excluded if they have any behavioral difficulties (Bjork & Pardini, 2015). Hence, the current study taps into this subgroup of youth selected on behavioral profiles of school and legal misconduct, rather than clinical diagnosis (Bjork & Pardini, 2015; Blair et al., 2018), thereby representing a sample of youth typically overlooked or excluded from research.

Despite previous behavioral work in clinical adolescent populations (i.e., diagnosed with CD or ODD), which collectively shows greater risk taking on experimental tasks such as the BART or IGT (Crowley et al., 2006; Humphreys & Lee, 2011; Schutter et al., 2011), the current findings rather unexpectedly showed lower levels of risk taking on the computerized risk-taking task in the CP group. Lower overall risk taking on the experimental paradigm is at odds with our self-report measures of greater risk-taking in daily life. There is currently limited research on how laboratory measures of risk taking generalize to real-world risky behaviors (Op de Macks et al., 2018). Indeed, a review on brain–behavior relations and

individual differences in risk taking in typically developing adolescents revealed that only two studies have reported a significant relation between laboratory-based and real-life risk-taking behavior (Sherman et al., 2018). It is possible that the self-report question-naire captures different aspects of risk taking than our experimental task. Yet, we did find a correlation between real-life risk taking and risky decision-making during the task in the CP group. Moreover, neural activity during the risky relative to safe decisions mediated real-life risk taking, which suggests that the task does tap into processes that underlie risky decision-making in real life.

We also note that there were no peer effects for the CP group, and peer effects in the typically developing group were in the direction of safer behavior. While risk-reducing peer effects seem surprising, this is in line with other recent work which shows that peer presence does not always lead to greater risky choices, but can also work to promote safer decisions (Somerville et al., 2019; for a recent meta-analysis see Defoe, Semon Dubas, & Romer, 2019). The absence of peer effects in the CP group is in line with previous work in adolescents with conduct disorders, which revealed less differentiation between emotional signals of peers, suggesting that they may be less influenced during decision-making (Klapwijk et al., 2016). This highlights that variables such as motivational and dispositional factors need to be explored (Foulkes & Blakemore, 2018; Perino, Guassi Moreira, et al., 2019; Perino Moreira, & Telzer, 2019).

Speculatively, risk taking in youth with conduct problems may be tied to specific conditions, with greater risk-taking behaviors in certain risk-taking contexts but not others (e.g., known, deviant peers). Future research could examine whether peer effects are present in the CP group when they are observed by their friend(s) rather than an unknown peer (cf. Chein et al., 2011), or when they are actively "egged-on" (Segalowitz et al., 2011). It will be important to examine the conditions under which peer effects occur (Somerville et al., 2019), in order to further inform our application of adolescent models of neurocognition to high-risk youth.

At the neural level, we first examined activation during risky decisions relative to safe decisions (go-stop) collapsed across the alone and peer condition. Risky decisions elicited greater activity in the VS for the typically developing than CP group, whereas safe decisions elicited greater activity in the VS for the CP youth than typically developing youth. The VS plays a key role in risk-taking behaviors, via processes of reward motivation and salience (Delgado, 2007; Schreuders et al., 2018; Telzer, 2016), and greater VS activity to rewards has been linked to greater motivation to pursue rewards and real-life risk taking (Galvan et al., 2007; Schreuders et al., 2018). This pattern of VS activity, which is reversed between the two groups, mirrors the behavioral findings and potentially suggests that risks were more affectively salient for the typically developing group, whereas safe decisions are more affectively salient for the CP group.

Additionally, youth with conduct problems showed greater activity in the dmPFC relative to typically developing youth. A recent meta-analysis showed that dmPFC is consistently activated when adolescents make decisions in a social context (Van Hoorn et al., 2019), and like the VS, has previously been implicated in risky decision-making (Blankenstein, Schreuders, Peper, Crone, & Duijvenvoorde, 2018; van Duijvenvoorde et al., 2015; van Leijenhorst et al., 2010). The role of the dmPFC may differ in part based on task demands and has been associated with the cognitive component of risk (Van Duijvenvoorde et al., 2015), as well as social information processing (e.g., Blakemore & Mills, 2014). While cognitive control was not assessed in the current task, recent work examined cognitive control in a delinquent sample of adolescents during fMRI. Interestingly, similar to the current findings, this study shows differences rather than deficits, such that the cognitive control of the delinquent group was poorer in the presence of aversive cues than appetitive cues, while the reverse pattern was found for typically developing youth, and differences in cognitive control were

paralleled by differences in activation in regions such as IFG and medial prefrontal cortex (Perino, Guassi Moreira, et al., 2019). Taken together, these studies suggest that adolescents with conduct problems appear to be characterized by qualitatively different neural processes than typically developing adolescents.

When we examined neural differences in the presence or absence of a peer, we found that TD adolescents showed greater VS activation during risky decision-making in the presence of peers relative to alone. This finding is consistent with prior research in normative developmental samples (Chein et al., 2011). However, this heightened VS activation in the presence of peers did not correspond to greater risk behavior in the presence of peers, as we found that adolescents made fewer risky decisions with a peer present. Speculatively, the VS signals processes of reward motivation and salience (Telzer, 2016), which may result in different behavior depending on the peer context. In the study of Chein et al. (2011), results showed increases in risk taking and VS activation when close friends were present, and therefore, adolescents may have shown heightened VS as they conformed to the (overestimated) perception of risk-taking norms of their friends (Prinstein & Wang, 2005). In the current work, a similar neural signal was associated with a risk reduction in the presence of an unknown peer, perhaps as youth conformed to a peer whose decisions were known (i.e., 50% risk). Thus, perhaps the behavioral effects of peer presence may depend on situational contexts, but may be neurally represented in similar ways, a hypothesis that should be investigated in future research.

Youth with conduct problems again showed a reversed pattern, such that the VS response during risky decisions was greater in the alone condition than in the peer, despite no behavioral differences in risk taking. This suggests that the CP group also process risky decisions in a social context qualitatively different from their typically developing counterparts. The social reorientation process of remodeling in the adolescent brain, which guides the navigation of social challenges associated with this developmental period (Blakemore & Mills, 2014; Crone & Dahl, 2012; Nelson, Jarcho, & Guyer, 2016; Nelson, Leibenluft, McClure, & Pine, 2005), may look substantially different depending on individual characteristics (e.g., behavioral disinhibition; Casey et al., 2011) or life experiences. Indeed, highrisk youth, such as those with conduct problems, often need to adapt to a more negative environment (Agnew, 1992; Hyde et al., 2013), which

necessitates greater attention to other types of cues, such as threat (Perino, Guassi Moreira, et al., 2019). Therefore, it may be possible that neural processing of reward in a social context in the CP group is substantially different from the typically develop-

The current study provides a novel insight into the behavioral and neural profiles of youth engaging in excessive risk behaviors. It can be considered a stepping stone in order to further understand adolescent neurocognition by focusing on youth who are at the greatest risk of poor psychosocial outcomes (cf. Bjork & Pardini, 2015). We recruited our high-risk adolescents from an alternative school for students who have been multiply suspended or were expelled from school for acts of gross legal and school misconduct, which puts them at heightened risk for lifetime delinquency (Moffitt & Caspi, 2001). Interestingly, mean levels of conduct problems did not appear very high in this group, suggesting some self-report measures may not fully capture the extent of the behaviors these youths engage in. Additionally, our sample size is small, and future research needs to replicate our findings in larger samples of diverse high-risk youth populations to ensure the robustness of our findings. With larger samples, it will also be possible to examine moderators of the effects, such as age, as well as peer attachment and conflict, which are especially relevant for examining peer effects in more depth. Future work may attempt to bring in adolescents with their actual friends and assess their friends' deviancy, to assess whether this impacts task behavior. Due to practical restraints in recruiting this sample, we were unable to assess and control for all potential confounding factors such as cognitive ability and socio-emotional intelligence. Previous work has shown that risk taking in mild-to-borderline intellectual disability may be better explained by low intellectual functioning rather than by comorbid behavior disorders, specifically under peer influence (Bexkens et al., 2002). As such, it would be an important next step to consider these variables in tandem with behavioral impairments in future work on this topic.

In conclusion, we employed a risk-taking task in a sample of adolescents characterized by extreme school and legal misconduct to gain traction on their behavioral and neural profiles of risk taking. Our findings show that youth with conduct problems are less risky during the experimental risk-taking task than typically developing teens, in line with neural findings which reflected more VS activity safe decisions than risky during decisions.

Interestingly, VS activity mediated higher rates of real-life risk taking in the CP group. Taken together, rather than greater reward processing as suggested by the imbalance model, or lower reward processing as suggested by the reward-deficiency hypothesis, youth with conduct problems showed a qualitatively different behavioral and neural profile during risk taking alone and in the presence of peers. These findings highlight the need for studies that bridge the gap between clinical and nonclinical samples, so that we can focus on prevention before clinical interventions are necessary.

REFERENCES

Agnew, R. (1992). Foundation for a general strain theory of crime and delinquency. Criminology, 30(1), 47-88. https://doi.org/10.1111/j.1745-9125.1992.tb01093.x

Alexander, C., Esminger, M., & Johnson, K. (1990). Adolescent Risk Taking Scale. Journal of Youth and Adolescence, 19, 409-424.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.) Washington, DC: Author.

Barrera, M., Prelow, H. M., Dumka, L. E., Gonzales, N. A., Knight, G. P., Michaels, M. L., ... Tein, J. (2002). Pathways from family economic conditions to adolescents' distress: Supportive parenting, stressors outside the family, and deviant peers. Journal of Community Psychology, 30(2), 135–152. https://doi.org/10.1002/jc

Beckmann, C. F., & Smith, S. M. (2004). Probabilistic independent component analysis for functional magnetic resonance imaging. IEEE Transactions on Medical Imaging, 23, 137–152. https://doi.org/10.1109/TMI. 2003.822821

Bexkens, A., Huizenga, H. M., Neville, D. A., d'Escury-Koenigs, A. L. C., Bredman, J. C., Wagemaker, E., & Van der Molen, M. W. (2019). Peer-influence on risktaking in male adolescents with mild to borderline intellectual disabilities and/or behavior disorders. Journal of Abnormal Child Psychology, 47(3), 543–555.

Bjork, J. M., Chen, G., Smith, A. R., & Hommer, D. W. (2010). Incentive-elicited mesolimbic activation and externalizing symptomatology in adolescents. Journal of Child Psychology and Psychiatry, 51, 827-837. https://d oi.org/10.1111/j.1469-7610.2009.02201.x

Bjork, J. M., & Pardini, D. A. (2015). Who are those "risktaking adolescents"? Individual differences in developmental neuroimaging research. Developmental Cognitive Neuroscience, 11, 56-64. https://doi.org/10.1016/j.dcn.

Blair, R. J. R., Veroude, K., & Buitelaar, J. K. (2018). Neuro-cognitive system dysfunction and symptom sets: A review of fMRI studies in youth with conduct problems. Neuroscience & Biobehavioral Reviews, 91, 69-90. https://doi.org/10.1016/j.neubiorev.2016.10.022

- 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
- Blankenstein, N. E., Schreuders, E., Peper, J. S., Crone, E. A., & van Duijvenvoorde, A. C. (2018). Individual differences in risk-taking tendencies modulate the neural processing of risky and ambiguous decision-making in adolescence. *NeuroImage*, 172, 663–673. https://doi.org/10.1016/j.neuroimage.2018.01.085
- Blum, K., Braverman, E. R., Holder, J. M., Lubar, J. F., Monastra, V. J., Miller, D., ... Comings, D. E. (2000). The reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive and compulsive behaviors. *Journal of Psychoactive Drugs*, 32(sup1), 1–112. https://doi.org/10.1080/02791072.2000.10736099
- Byrd, A. L., Loeber, R., & Pardini, D. A. (2014). Antisocial behavior, psychopathic features and abnormalities in reward and punishment processing in youth. *Clinical Child and Family Psychology Review*, 17(2), 125–156. https://doi.org/10.1007/s10567-013-0159-6
- Casey, B. J. (2015). Beyond simple models of self-control to circuit-based accounts of adolescent behavior. Annual Review of Psychology, 66, 295–319. https://doi.org/10.1146/annurev-psych-010814-015156
- Casey, B. J., Somerville, L. H., Gotlib, I. H., Ayduk, O., Franklin, N. T., ... Shoda, Y. (2011). Behavioral and neural correlates of delay of gratification 40 years later. *Proceedings of the National Academy of Sciences*, 108, 14998–15003. https://doi.org/10.1073/pnas.1108561108
- Center for Disease Control and Prevention, National Center for Health Statistics; Heron, M. (2017). Deaths: Leading causes for 2014. *National Vital Statistics Reports*, 65, 1–96.
- 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(2), F1–F10. https://doi.org/ 10.1111/j.1467-7687.2010.01035.x
- Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, *13*, 636–650. https://doi.org/10.1038/nrn3313
- Crone, E. A., van Duijvenvoorde, A. C., & Peper, J. S. (2016). Annual research review: Neural contributions to risk-taking in adolescence: Developmental changes and individual differences. *Journal of Child Psychology and Psychiatry*, 57, 353–368. https://doi.org/10.1111/jcpp.12502
- Crowley, T. J., Raymond, K. M., Mikulich-Gilbertson, S. K., Thompson, L. L., & Lejuez, C. W. (2006). A risk-taking "set" in a novel task among adolescents with serious conduct and substance problems. *Journal of the American Academy of Child & Adolescent Psychiatry*, 45 (2), 175–183. https://doi.org/10.1097/01.chi. 0000188893.60551.31

- Cservenka, A., & Nagel, B. J. (2012). Risky decision-making: An FMRI study of youth at high risk for alcoholism. *Alcoholism: Clinical and Experimental Research*, *36*, 604–615. https://doi.org/10.1111/j.1530-0277.2011.01650.x
- Defoe, I. N., Dubas, J. S., Figner, B., & van Aken, M. A. (2015). A meta-analysis on age differences in risky decision making: Adolescents versus children and adults. *Psychological Bulletin*, 141(1), 48–84. https://doi.org/10.1037/a0038088
- Defoe, I. N., Semon Dubas, J., & Romer, D. (2019). Heightened adolescent risk-taking? Insights from lab studies on age differences in decision-making. *Policy Insights from the Behavioral and Brain Sciences*, 6(1), 56–63. https://doi.org/10.1177/2372732218801037
- Delgado, M. R. (2007). Reward-related responses in the human striatum. *Annals of the New York Academy of Sciences*, 1104(1), 70–88. https://doi.org/10.1196/annals.1390.002.
- Dishion, T. J., & Tipsord, J. M. (2011). Peer contagion in child and adolescent social and emotional development. *Annual Review of Psychology*, 62, 189–214. https://doi.org/10.1146/annurev.psych.093008.100412
- Finger, E. C., Marsh, A. A., Mitchell, D. G., Reid, M. E., Sims, C., Budhani, S., ... Blair, J. R. (2009). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry*, *65*, 586–594. https://doi.org/10.1001/archpsyc.65.5.586
- Foulkes, L., & Blakemore, S. J. (2018). Studying individual differences in human adolescent brain development. *Nature Neuroscience*, *21*, 315–323. https://doi.org/10.1038/s41593-018-0078-4
- Galvan, A. (2010). Adolescent development of the reward system. *Frontiers in Human Neuroscience*, 4, 116–124. https://doi.org/10.3389/neuro.09.006.2010
- Galvan, A., Hare, T., Voss, H., Glover, G., & Casey, B. J. (2007). Risk-taking and the adolescent brain: Who is at risk? *Developmental Science*, 10(2), F8–F14. https://doi.org/10.1111/j.1467-7687.2006.00579.x
- Gardner, M., & Steinberg, L. (2005). Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology*, 41, 625–635. https://doi.org/10.1037/0012-1649.41.4.625
- Gardner, T. W., Dishion, T. J., & Connell, A. M. (2008). Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology*, *36*, 273–284. https://doi.org/10.1007/s10802-007-9176-6
- Giuliani, N. R., & Pfeifer, J. H. (2015). Age-related changes in reappraisal of appetitive cravings during adolescence. *NeuroImage*, 108, 173–181. https://doi.org/10.1016/j.neuroimage.2014.12.037
- Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, 38, 581–586. https://doi.org/10.1111/j. 1469-7610.1997.tb01545.x

- Goodnight, J. A., Bates, J. E., Newman, J. P., Dodge, K. A., & Pettit, G. S. (2006). The interactive influences of friend deviance and reward dominance on the development of externalizing behavior during middle adolescence. Journal of Abnormal Child Psychology, 34, 573-583. https://doi.org/10.1007/s10802-006-9036-9
- Gorgolewski, K. J., Varoquaux, G., Rivera, G., Schwarz, Y., Ghosh, S. S., Maumet, C., ... Yarkoni, T. (2015). NeuroVault.org: A web-based repository for collecting and sharing unthresholded statistical maps of the human brain. Frontiers in Neuroinformatics, 9, 8.
- Guassi Moreira, J., & Telzer, E. H. (2018). Mother still knows best: Maternal influence uniquely modulates adolescent reward sensitivity during risk taking. Developmental Science, 21(1), e12484.
- Humphreys, K. L., & Lee, S. S. (2011). Risk taking and sensitivity to punishment in children with ADHD, ODD, ADHD+ ODD, and controls. Journal of Psychopathology and Behavioral Assessment, 33, 299-307. https://doi.org/10.1007/s10862-011-9237-6
- Hyde, L. W., Shaw, D. S., & Hariri, A. R. (2013). Understanding youth antisocial behavior using neuroscience through a developmental psychopathology lens: Review, integration, and directions for research. Developmental Review, 33, 168-223. https://doi.org/10.1016/ j.dr.2013.06.001
- Jenkinson, M., Bannister, P., Brady, M., & Smith, S. (2002). Improved optimization for the robust and accurate linear registration and motion correction of brain images. NeuroImage, 17, 825-841. https://doi.org/10. 1006/nimg.2002.1132
- Jenkinson, M., & Smith, S. (2001). A global optimisation method for robust affine registration of brain images. Medical Image Analysis, 5(2), 143-156. https://doi.org/ 10.1016/S1361-8415(01)00036-6
- Kahn, L. E., Peake, S. J., Dishion, T. J., Stormshak, E. A., & Pfeifer, J. H. (2015). Learning to play it safe (or not): Stable and evolving neural responses during adolescent risky decision-making. Journal of Cognitive Neuroscience, 27, 13-25. https://doi.org/10.1162/jocn_a_
- Klapwijk, E. T., Lelieveld, G. J., Aghajani, M., Boon, A. E., van der Wee, N. J., Popma, A., ... Colins, O. F. (2016). Fairness decisions in response to emotions: A functional MRI study among criminal justice-involved boys with conduct disorder. Social Cognitive and Affective neuroscience, 11(4), 674-682.
- Liu, J. (2005). Childhood externalizing behavior: Theory and implications. Journal of Child and Adolescent Psychiatric Nursing, 17, 93–103. https://doi.org/10.1111/j. 1744-6171.2004.tb00003.x
- Moffitt, T. E. (2018). Male antisocial behavior in adolescence and beyond. Nature Human Behavior, 2, 177-186.
- Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescencelimited antisocial pathways among males and females. Development and Psychopathology, 13(2), 355–375.

- Nelson, E. E., Jarcho, J. M., & Guyer, A. E. (2016). Social re-orientation and brain development: An expanded and updated view. Developmental Cognitive Neuroscience, 17, 118-127. https://doi.org/10.1016/j.dcn. 2015.12.008
- Nelson, E. E., Leibenluft, E., McClure, E., & Pine, D. S. (2005). The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. Psychological Medicine, 35(2), 163-174. https://doi.org/10.1017/S0033291704003915
- Op de Macks, Z. A., Flannery, J. E., Peake, S. J., Flournoy, J. C., Mobasser, A., Alberti, S. L., ... 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
- Peake, S. J., Dishion, T. J., Stormshak, E. A., Moore, W. E., & Pfeifer, J. H. (2013). Risk-taking and social exclusion in adolescence: Neural mechanisms underlying peer influences on decision-making. NeuroImage, 82, 23-34https://doi.org/10.1016/j.neuroimage.2013.05.061
- Perino, M. T., Guassi Moreira, J. F., McCormick, E. M., & Telzer, E. H. (2019). Apples to apples? Neural correlates of emotion regulation differences between high and low risk adolescents. Social Cognitive and Affective Neuroscience, 14, 827-836. https://doi.org/10.1093/sca n/nsz063
- Perino, M. T., Moreira, J. F. G., & Telzer, E. H. (2019). Links between adolescent bullying and neural activation to viewing social exclusion. Cognitive, Affective, & Behavioral Neuroscience, 19, 1467–1478. https://doi.org/ 10.3758/s13415-019-00739-7
- Pfeifer, J. H. (2015). Yellow Light Game. Retrieved March 18, 2018, from https://dsn.uoregon.edu/research/yel low-light-game/.
- Piedmont, R. L. (2014). Interitem correlations. In A. C. Michalos (Ed.), Encyclopedia of quality of life and well-being research. Dordrecht, The Netherlands: Springer.
- Prinstein, M. J., & Wang, S. S. (2005). False consensus and adolescent peer contagion: Examining discrepancies between perceptions and actual reported levels of friends' deviant and health risk behaviors. Journal of Abnormal Child Psychology, 33, 293-306. https://doi. org/10.1007/s10802-005-3566-4
- Qu, Y., Galvan, A., Fuligni, A. J., Lieberman, M. D., & Telzer, E. H. (2015). Longitudinal changes in prefrontal cortex activation underlie declines in adolescent risk Journal of Neuroscience, 35, 11308-11314. https://doi.org/10.1523/JNEUROSCI.1553-15.2015
- Richmond, A. D., Laursen, B., & Stattin, H. (2019). Homophily in delinquent behavior: The rise and fall of friend similarity across adolescence. International Journal of Behavioral Development, 43(1), 67-73.
- Rogers, C. R., McCormick, E. M., van Hoorn, J., Ivory, S., & Telzer, E. H. (2018). 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
- Rubia, K. (2011). "Cool" inferior frontostriatal dysfunction in attention-deficit/hyperactivity disorder versus "hot" ventromedial orbitofrontal-limbic dysfunction in conduct disorder: A review. *Biological Psychiatry*, 69 (12), e69–e87. https://doi.org/10.1016/j.biopsych.2010.09.023
- Rubia, K., Smith, A. B., Halari, R., Matsukura, F., Mohammad, M., Taylor, E., & Brammer, M. J. (2009). Disorder-specific dissociation of orbitofrontal dysfunction in boys with pure conduct disorder during reward and ventrolateral prefrontal dysfunction in boys with pure ADHD during sustained attention. *American Journal of Psychiatry*, 166(1), 83–94. https://doi.org/10.1176/appi.ajp.2008.08020212
- 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
- Schutter, D. J., Van Bokhoven, I., Vanderschuren, L. J., Lochman, J. E., & Matthys, W. (2011). Risky decision making in substance dependent adolescents with a disruptive behavior disorder. *Journal of Abnormal Child Psychology*, 39, 333–339. https://doi.org/10.1007/ s10802-010-9475-1
- Segalowitz, S. J., Santesso, D. L., Willoughby, T., Reker, D. L., Campbell, K., Chalmers, H., & Rose-Krasnor, L. (2011). Adolescent peer interaction and trait surgency weaken medial prefrontal cortex responses to failure. Social Cognitive and Affective Neuroscience, 7(1), 115–124. https://doi.org/10.1093/scan/nsq090
- Sherman, L. E., Steinberg, L., & Chein, J. (2018). Connecting brain responsivity and real-world risk taking: Strengths and limitations of current methodological approaches. *Developmental Cognitive Neuroscience*, 33, 27–41. https://doi.org/10.1016/j.dcn.2017.05.007
- 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
- Silverman, M. H., Jedd, K., & Luciana, M. (2015). Neural networks involved in adolescent reward processing: An activation likelihood estimation meta-analysis of functional neuroimaging studies. *NeuroImage*, 122, 427– 439. https://doi.org/10.1016/j.neuroimage.2015.07.083
- Smith, S. M. (2002). Fast robust automated brain extraction. Human Brain Mapping, 17, 143–155. https://doi.org/10.1002/hbm.10062.
- Somerville, L. H., Haddara, N., Sasse, S. F., Skwara, A. C., Moran, J. M., & Figner, B. (2019). Dissecting "peer presence" and "decisions" to deepen understanding of peer influence on adolescent risky choice. *Child Devel-opment*, 90(6), 2086–2103.

- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, 28(1), 78–106. https://doi.org/10.1016/j.dr.2007.08.002
- Steinberg, L., Icenogle, G., Shulman, E. P., Breiner, K., Chein, J., Bacchini, D., ... Fanti, K. A. (2017). Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation. *Developmental Science*, 21(2), e12532https://doi.org/10.1111/desc. 12532
- Telzer, E. H. (2016). Dopaminergic reward sensitivity can promote adolescent health: A new perspective on the mechanism of ventral striatum activation. *Developmental Cognitive Neuroscience*, 17, 57–67. https://doi.org/10.1016/j.dcn.2015.10.010
- Telzer, E. H., Gonzales, N., & Fuligni, A. J. (2014). Family obligation values and family assistance behaviors: Protective and risk factors for Mexican-American adolescents' substance use. *Journal of Youth and Adolescence*, 43, 270–283. https://doi.org/10.1007/s10964-013-9941-5
- Telzer, E. H., Ichien, N. T., & Qu, Y. (2015). Mothers know best: Redirecting adolescent reward sensitivity toward safe behavior during risk taking. *Social Cognitive and Affective Neuroscience*, 10, 1383–1391. https://doi.org/10.1093/scan/nsv026
- Tohka, J., Foerde, K., Aron, A. R., Tom, S. M., Toga, A. W., & Poldrack, R. A. (2008). Automatic independent component labeling for artifact removal in fMRI. *NeuroImage*, 39, 1227–1245. https://doi.org/10.1016/j.neuroimage.2007.10.013
- Tyler, P. M., White, S. F., Thompson, R. W., & Blair, R. J. R. (2019). Applying a cognitive neuroscience perspective to disruptive behavior disorders: Implications for schools. *Developmental Neuropsychology*, 44(1), 17–42. https://doi.org/10.1080/87565641.2017.1334782
- Van Duijvenvoorde, A. C., Huizenga, H. M., Somerville, L. H., Delgado, M. R., Powers, A., Weeda, W. D., ... Figner, B. (2015). Neural correlates of expected risks and returns in risky choice across development. *Journal of Neuroscience*, 35, 1549–1560. https://doi.org/10.1523/JNEUROSCI.1924-14.2015
- Van Duijvenvoorde, A. C., Peters, S., Braams, B. R., & Crone, E. A. (2016). What motivates adolescents? Neural responses to rewards and their influence on adolescents' risk taking, learning, and cognitive control. *Neuroscience & Biobehavioral Reviews*, 70, 135–147. https://doi.org/10.1016/j.neubiorev.2016.06.037
- Van Hoorn, J., McCormick, E. M., Rogers, C. R., Ivory, S. L., & Telzer, E. H. (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/nsy071
- Van Hoorn, J., Shablack, H., Lindquist, K. A., & Telzer, E. H. (2019). Incorporating the social context into neurocognitive models of adolescent risk-taking: A neuroimaging meta-analysis. *Neuroscience and Biobehavioral Reviews*, 101, 129–142.

- Van Leijenhorst, L., Moor, B. G., de Macks, Z. A. O., Rombouts, S. A., Westenberg, P. M., & Crone, E. A. (2010). Adolescent risky decision-making: Neurocognitive development of reward and control regions. NeuroImage, 51(1), 345-355. https://doi.org/10.1016/j. neuroimage.2010.02.038
- Viding, E., Fontaine, N. M. G., & McCrory, E. J. (2012). Antisocial behavior in children with and without callous-unemotional traits. Journal of the Royal Society of Medicine, 105, 195-200.
- Ward, B. D. (2000). Simultaneous inference for fMRI data. Retrieved from https://afni.nimh.nih.gov/pub/dist/d oc/manual/AlphaSim.pdf (April 2, last accessed date).
- Willoughby, T., Good, M., Adachi, P. J., Hamza, C., & Tavernier, R. (2014). Examining the link between adolescent brain development and risk taking from a social-developmental perspective. Brain and Cognition, 89, 70-78.

Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. (a) Regression with conduct problems on the go-stop contrast across all participants. (b) real-life risk taking on the go-stop contrast across all participants.

Table S1. Brain Regions for Regressions with conduct problems (CP) and Real-Life Risk Taking Behavior (ART) on Go-Stop Contrast Across All **Participants**