Not just social sensitivity: Adolescent neural suppression of social feedback during risk taking

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1. Introduction

During the transition from childhood to adolescence there is a sharp rise in risk-taking and sensation-seeking behaviors that coincide with increases in sensitivity to social information (e.g., Steinberg, 2010; Crone and Dahl, 2012; Brener et al., 2013). These changes are accompanied by a complex neural reorganization that shapes networks involved in both risk taking and social processing (Casey et al., 2005; Tamnes et al., 2013; Mills et al., 2014; Wierenga et al., 2014; Mills et al., 2016; Vijayakumar et al., 2016). While often examined separately, risk taking and social sensitivity are clearly intertwined. For example, compared to childhood, adolescents spend considerably more time with non-related conspecifics, forming extended peer groups, as well as establishing potential romantic relationships (Larson and Richards, 1991). This social reorientation can help adolescents establish their places in adult-like social networks, promoting survival and reproduction (Nelson et al., 2005; Steinberg, 2008; Pellegrini and Long, 2003). However, there are also myriad potential downsides to these behavioral changes, as engaging with new peers can involve substantial risk of social rejection or aggressive conflicts (Wentzel and Erdley, 1993; Laird et al., 2001), and may promote risky behavior (Lewis and Lewis, 1984; La Greca et al., 2001; Miller-Johnson et al., 2003). Although the development of social processing has generated significant interest in recent years (e.g., Crone and Dahl, 2012; Blakemore and Mills, 2014), many questions remain about how adolescents integrate social information into on-going neural representations during risky-decision making. Furthermore, social information often changes dynamically in the real-world, yet most studies have primarily utilized static social inputs (e.g., still faces: Burnett et al., 2009; Guyer et al., 2009; presence versus absence: Chein et al., 2011; Telzer et al., 2015b; but see Flannery et al., 2017). To address these limitations, we examined how children and adolescents use dynamic social information during risky decision-making.

Social information processing is supported by a distributed collection of brain regions including temporo-parietal regions (e.g., TPJ, pSTS), medial prefrontal, and subcortical structures (e.g., amygdala; see Blakemore, 2008; Nelson et al., 2016 for complete review). Indeed, a growing body of work has shown that adolescents, compared with children or adults, show heightened neural responsivity to socio-emotional information. For instance, adolescents show heightened reactivity in the MPFC when being observed by peers (Somerville et al., 2013) and heightened TPJ activation when viewing social information (Burnett et al., 2009; Burnett and Blakemore, 2009), suggesting that adolescents orient towards social information to a greater extent than other age groups. This increased social orientation can interact with other domains of adolescent cognition, as both positive (Somerville et al., 2011; Perino et al., 2016) and negative (Hare et al., 2008) socio-emotional information disrupts adolescents’ cognitive control via...
heightened activation in social and affective brain regions, including the TPJ, ventral striatum, insula, and amygdala.

Adolescents’ heightened sensitivity to social information can contribute to increases in risky behavior. Indeed, a robust body of work has shown that when peers are present, adolescents prefer more immediate rewards (O’Brien et al., 2011) and are more risky, both in the lab (Gardner and Steinberg, 2005; Chein et al., 2011; Knoll et al., 2015; Silva et al., 2016) and in the real world (e.g., Prieur et al., 2001; Simons-Morton et al., 2005; Chassin et al., 2009), even when teens are presented with the potential negative outcomes of their risky decisions (Smith et al., 2014). Furthermore, enhanced neural reactivity to peers (versus parents) in a socioemotional context has been linked to increases in risky behavior (Saxbe et al., 2015). However, this social sensitivity in the context of risk may not ubiquitously contribute to increases in risk taking. For instance, while peer exclusion can increase risky behavior (Peake et al., 2013; Falk et al., 2014), prosocial feedback can modulates adolescents’ propensity to make prosocial decisions via activation in the MPFC and TPJ (van Hoorn et al., 2016a,b), and the presence of mothers decreases adolescents’ risky decisions via activation in the TPJ and fusiform (Guassi Moreira and Telzer, 2016). Thus, acting antisocially may not reflect disinhibition per se, but rather a shift of both psychological and neural resources to behaviors and cognitions aligned with those goals. This suggests that adolescents may use social information to a greater extent during goal-directed behavior, but the consequences of increased sensitivity to social information may vary based on what goals are prioritized in a given situation.

The idea that adolescents may be able to use social information flexibly is consistent with recent findings in adolescent decision-making. Adolescents are more likely than children to use feedback information to guide their future behavior (McCormick and Telzer, 2017) and research on both rodent (Johnson and Willbrecht, 2011; Pattwell et al., 2012) and human (Humphreys et al., 2015) adolescents show greater exploratory and learning behavior compared with other age groups, patterns of behavior which promote adaptive outcomes for adolescents. This raises an interesting question: if adolescents are able to flexibly use social information, are they also capable of disregarding social information when it interferes with goal-relevant behavior? While previous work examining the impact of social information on risky behavior (Chein et al., 2011; Albert et al., 2013; Telzer et al., 2015b; Guassi Moriera and Telzer, 2016) has shown that adolescents’ behavior and neural representations of safe and risky decisions are impacted by the presence of social agents, these manipulations have not directly pitted social feedback information against adolescents’ goals. Moreover, these paradigms, as well as most studies of social influence, have relied on static social contexts (e.g., presence or absence of social inputs) to examine the effect of social information on behavior. Exploring how social information updates in response to participants’ risky choices in vivo can extend our understanding of real-world risk taking, where individuals need to balance both reward- and social-related consequences of their actions in real time. Thus, our goal was to examine whether adolescents are able to ignore salient, but non-goal relevant social information in pursuit of goal-related behaviors when social information and adolescents’ goals are explicitly pitted against one another.

In the current study, youths, aged 8–17 years, completed a novel, social version of the Balloon Analogue Risk Task (BART; Lejuez et al., 2002), during which participants saw adult faces (rather than balloons) become angrier and angrier as they made riskier decisions. Riskier decisions were coupled with a reward value but also a potential loss of all reward if participants were too risky. For each sequential risk decision, participants received increasingly negative social feedback, pitting sensitivity to social information against the goal of the task, to earn points. We examined age-related changes in participants’ risk behavior, as well as how neural regions involved in regulatory, affective, and social information processing track increasing risk and changing social feedback across each risk opportunity.

Previous research suggests several competing hypotheses, which would be reflected in different patterns of behavioral and neural responses. On the one hand, adolescents may show hypersensitivity to social information. Thus, greater social sensitivity (relative to rewards) would predict that adolescents would show aversion to increasingly negative social feedback. Indeed, adolescents show heightened affective and social brain activation (Hare et al., 2008; Somerville et al., 2011; Perino et al., 2016) in the presence of social cues, which results in impaired cognitive regulation (Perino et al., 2016). Thus, older adolescents, relative to children, would show increased sensitivity to social feedback, which would be reflected in heightened responsivity in affective/salience (e.g., amygdala, insula) and social brain (e.g., TPJ, mPFC) regions.

On the other hand, adolescents may flexibly ignore increasingly negative social information in the service of goal-directed behavior (i.e., accruing points). In this scenario, adolescents would be able to suppress social information in order to engage in risk taking and gain more points on the task. This hypothesis is consistent with findings showing that adolescents are more motivated by rewards (Crews et al., 2007; Galvan, 2010), but also more flexible in pursuing goal-relevant behavior (McCormick and Telzer, 2017). Thus, older adolescents, relative to children, would be able to flexibly ignore the increasingly negative social information in the service of goal-directed behavior (i.e., accruing points).

In order to address these questions, we examined developmental shifts in children and adolescents’ sensitivity to increasing risk coupled with negative social information during a risky decision-making task coupled with dynamic social information. To test whether adolescents show increasing sensitivity or suppressed sensitivity of social information in the service of rewards, we conducted analyses which utilized a parametric modulator (PM) to explore how neural activation adapts in response to dynamic social feedback. The parametric modulator captures neural adaptation (i.e., tracking changing task parameters) within an individual, allowing us to characterize systematic age-related differences in the degree to which children and adolescents show increased or decreased sensitivity to changing social information.

2. Methods

2.1. Participants

Fifty-six healthy children and adolescents completed an fMRI scan. One participant was excluded due to excessive head motion during the scan session (> 2.0 mm movement between slices on ≥10% of slices), leaving a final sample of fifty-five participants (30 female; M_age = 13.34 years, SD = 2.84, range = 8.1-16.5 years). Participants (43 European-American, 6 African-American, 1 Asian-American, 2 Latin-American, and 3 mixed/multiple ethnicity) provided written consent and assent in accordance with the University of Illinois’ Institutional Review Board.

2.2. Social risk taking task

Participants completed a social risk-taking task based on the well-established Balloon Analogue Risk Task (BART; Lejuez et al., 2002), which involves sequential risk decisions in pursuit of rewards (e.g., money, points). The BART indexes risky behavior by measuring participants’ willingness to pump up a balloon in order to earn rewards; however, each sequential decision to pump up a balloon parametrically increases the risk that the balloon will explode and the subject will lose all the points they might have earned on that balloon. Behavioral performance and neural reactivity during the BART have been related to real-world risk-taking behaviors (Lejuez et al., 2002; Qu et al., 2015; Telzer et al., 2015a), suggesting that this task captures externally valid components of real-world risky behavior. In our social version of the BART, the Social Analogue Risk Task (SART; adapted from Humphreys
Participants were shown the face of the "resident" for each door. Participants could choose to knock on the door, and if the resident became too angry, they would slam the door, and participants would lose all the points they had earned on that door. Thus, at any point after the first knock, participants could choose to either knock or cash-out; new face following a slam trial were separated by a random jitter (500–4000 ms). Faces were presented in a fixed order that was unknown to participants. Participants saw 12 individual faces (4 White, 4 Black, and 4 Asian; all female) each presented twice during the task, and face presentation was ordered such that no face was repeated before all faces were presented at least once. The task was self-paced, and did not advance unless participants made a decision to either knock or cash-out following at least one knock on a given trial.

Participants' willingness to engage in risky behavior was indexed by the average number of times they knocked on doors where they eventually cashed-out. Consistent with previous research (Lejuez et al., 2002; McCormick and Telzer, 2017), the number of knocks on slam trials was not included since those trials artificially curtail participants' willingness to engage in risky behavior. Negative outcomes on the task were indexed by the number of times participants experienced slam events, while positive outcomes were indexed by the total number of points participants earned on the task.

2.3. fMRI data acquisition

Imaging data were collected using a 3 T Siemens Trio MRI scanner. The BART included T2*-weighted echoplanar images (EPI) (slice thickness = 3 mm; 38 slices; TR = 2 s; TE = 25 msec; matrix = 92 × 92; FOV = 230 mm; voxel size 2.5 × 2.5 × 3 mm³). In addition, structural scans consisted of a T2*-weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4 s; TE = 64 msec; FOV = 230; matrix = 192 × 192; slice thickness = 3 mm; 38 slices) and a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1.9 s; TE = 2.3 msec; FOV = 230; matrix = 256 × 256; sagittal plane; slice thickness = 1 mm; 192 slices). To maximize brain coverage, MBW and EPI scans were obtained using an oblique axial orientation.

2.3.1. fMRI data preprocessing and analysis

The Statistical Parametric Mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK) software package was used for preprocessing and data analysis. Steps for preprocessing involved head motion correction using spatial realignment (including participants had no motion in excess of 1.8 mm slice-to-slice motion); coregistration of all images to the high-resolution T1* MPRAGE structural scan (1 participant lacked a T1 image so coregistration utilized the MBW image); and segmentation into grey matter, white matter, and cerebrospinal fluid. MPRAGE segmentation resulted in transformation matrices that were applied to MBW and EPI images to warp them into the standard stereotactic space defined by the Montreal Neurological Institute (MINI) and the International Consortium for Brain Mapping. An 8 mm Gaussian kernel, full-width-at-half maximum was used to smooth the EPI images in order to increase signal-to-noise ratios in the functional images. The hemodynamic response function was convolved for each trial using the general linear model in SPM8. A high-pass filter with a 128 s cutoff was applied to remove low-frequency drift.
across time in the time-series, and serial autocorrelations were estimated using a restricted maximum likelihood algorithm with an autoregressive model order of 1.

The SART was modeled using an event-related design. A general linear model was included in the fixed-effects model for each condition of interest, which included knock decisions, cash-out decisions, and slam events. We modeled knock decisions separately for trials that ended in cash-outs and trials that ended in slams. Following previous research (Lejuez et al., 2002; McCormick and Telzer, 2017), analyses were performed using knock decisions on trials that ended in cash-outs, as the number of knocks was artificially constrained on trials that ended in door slams. Jittered inter-trial periods were not modeled and served as the implicit baseline for the task.

In order to model sensitivity to the dynamic social information across risky decisions in the task, a parametric modulator (PM) was included for our primary condition of interest, knock events. The PM values represented the knock number for each decision trial, and all PM values were centered for each trial within a person around the average knock for each face. This PM allowed us to examine how the brain linearly tracks increasing social risk. As such, significant parameter estimates represent voxels that show neural tracking (i.e., show monotonic changes in activation that correspond to increases in social risk). Contrasts were then computed at the individual level for each person across risky decisions in the task, a parametric modulator (PM) was included for our primary condition of interest, knock events. The PM values represented the knock number for each trial within a person around the average knock for each face. This PM allowed us to examine how the brain linearly tracks increasing social risk. As such, significant parameter estimates represent voxels that show neural tracking (i.e., show monotonic changes in activation that correspond to increases in social risk). Contrasts were then computed at the individual level for each condition of interest. Our contrast therefore focused on Increasing Social Risk, which refers to the concomitant parametric increase in the negative affect displayed on the face of the resident coupled with the risk of the door slamming following successive decisions to knock.

At the group level, we conducted random-effects analyses, in which age was entered in whole brain regression analyses in order to examine whether there are age differences in neural sensitivity to increasing social risk. We tested whether age was associated with increasing sensitivity (i.e., neural regions increasingly track the changing anger level and risk within a trial) or suppressed sensitivity (i.e., neural regions show decreased tracking of the anger level and risk within a trial). Random effects, group-level analyses were run on individual subject contrasts using GLMFlex, 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). To ensure that age-related results were not driven by differences in the amount of data acquired in the self-paced task, we ran follow-up, correlation and whole-brain analyses controlling for the number of TRs and the average length of knock events in participants’ task. Neither task length (M = 261.78 TRs, SD = 56.28, range = 110-397 TRs) nor average length of knock events (M = 1.19, SD = 0.27, range = 0.58-1.94) correlated with age (TR: r = 0.19, p = 0.16; Average Length: r = 0.03; p = 0.82), and the whole-brain results remained unchanged when controlling for these variables.

Correction for multiple comparisons was run using a Monte Carlo simulation through the updated version (April, 2016) of the 3dFWHMx and 3dClustSim programs from the AFNI software package (Ward, 2000) using the group-level brain mask. The simulation resulted in a voxel-wise threshold of p < 0.005 and a minimum cluster size of 53 voxels for the whole brain, corresponding to p < 0.05, Family-Wise Error (FWE) corrected. All results are available on NeuroVault (Gorgolewski et al., 2015; see http://neurovault.org/collections/PMZPESDZ/).

3. Results

3.1. Behavioral results

3.1.1. Age-related changes in task performance

To test for age-related changes in task performance, we ran bivariate correlations between participants’ age and behavioral indices of interest (see Table 1 for descriptive information and associations between all variables of interest). Age was associated with more risk behavior (i.e., greater knocks at each door) and the total number of points earned during the task, but not with the number of slam events participants experienced. We used the methods outlined by Hayes (Hayes, 2013) to determine if the association between age and points earned was accounted for by greater knocks, when controlling for slam events. We standardized all variables, and then using 1000 sample bootstrapping, calculated the significance and magnitude of the indirect effect, as well as a bias-corrected confidence interval (CI). We found a significant indirect effect, such that increases in participants’ average number of knocks mediated the relationship between age and total points earned on the task (B = 0.43 SE = 0.11; 95% CI = [0.22, 0.65]), suggesting that with age, participants showed more optimal risk taking behavior; knocking more, but effectively cashing-in before experiencing a slam, and thereby receiving more points.

3.2. fMRI results

3.2.1. Age-related neural sensitivity to increasing social risk

We first examined age-related differences in how children and adolescents track increasing social risk (for main effects, see Table 2). To do this, age was entered as a continuous regressor in a whole-brain regression analysis on the contrast of increasing social risk (i.e., events where participants knocked on doors; parametric modulator representing increasing social risk). Results indicate age-related decreases in neural tracking of social risk in the bilateral insula/IFG, mid-cingulate cortex (MCC), left posterior superior temporal sulcus (pSTS), bilateral temporoparietal junction (TPJ), bilateral temporal poles, and left fusiform gyrus (Table 3). For descriptive purposes, we extracted parameter estimates from the insula, pSTS, and left TPJ and plotted them with age in Fig. 2. The scatterplots demonstrate age-related patterns in neural tracking, where the y-axis represents parametric increases in neural activation across the trials (positive values indicate monotonic linear increases in neural activation, whereas values around 0 indicate no change in neural activation across the trial). As shown in Fig. 2, children show positive tracking of social risk, that is, they show a concomitant increase in insula, TPJ, and pSTS activation as trials increase in risk and negative feedback. Across early and later adolescents, there is a transition from this positive tracking to showing neural insensitivity to changing task parameters (i.e., showing similar activation across increasing social risk). As such, older (relative to younger) participants show a decreased tracking of risk and negative affect in regions related to social cognition and regulatory processes.

In order to further explore and clarify these effects, we performed follow-up analyses examining age-related changes in activation during knock decisions (i.e., decisions to take a risk), controlling for the parametric modulator (for main effects, see Table 2). This analysis explored whether adolescents might show a lack of neural tracking because there were differences in the overall level of activation in the regions highlighted in the previous results. If, for instance, adolescents had higher overall activation in the insula or TPJ, they might show

| Variable          | M     | SD    | Range | 1   | 2   | 3   | 4   |
|-------------------|-------|-------|-------|-----|-----|-----|-----|
| 1. Age            | 13.34 | 2.84  | 8.10-16.54 | .33* | .45*** | 0.003 |
| 2. Average Knocks (# Trials) | 4.51 | 1.05 | 2.17-6.50 | .91*** | .70*** |
| 3. Total Points   | 97.34 | 18.43 | 52-129 | .33* |
| 4. Number of Slam Trials | 2.09 | 1.97 | 0-8 | 1 |

Note: * < 0.05, ** < 0.01 *** < 0.005. Numbers along the diagonal represent Pearson’s correlations.
reduced tracking due to a ceiling effect, where they are hypersensitive to the negative social information at all levels of risk decisions. Whole-brain regression analyses indicated that across risk decisions, control-related increases or decreases in any of the regions identified above (Table 3). These findings held even if we relaxed the whole-brain threshold to \( p < 0.01 \), suggesting that we were not masking potentially meaningful subthreshold results.

These findings suggest that the age-related changes in neural tracking of increasing social risk are not simply due to differences in overall sensitivity to the task, but rather specifically in differential sensitivity across levels of dynamic social feedback. If we had seen strong age-related increases in activation, the lack of tracking increasing social risk we observed in older adolescents could have been attributable to these participants being at-ceiling in terms of their neural activation, thereby reducing neural tracking because they could not increase further. However, we find no systematic differences in activation in regions which showed reduced tracking of increasing social risk, suggesting that these effects represent adolescents’ relative suppression in response to the high intensity negative social information at progressively riskier choices. These findings lend support to the hypothesis that adolescents are capable of flexibly ignoring social information when it conflicts with their goals within the task.

### 3.2.2. Links between age-related neural sensitivity to increasing social risk and task performance

Finally, we performed post-hoc tests that examined whether age-related decreases in the neural tracking of social risk were related to task performance. To do so, we extracted parameter estimates of signal intensity from significant clusters in our whole-brain regression with age. We then correlated these extracted parameter estimates with behavioral performance on the task. All regions identified in our age analysis were related to both average knocks and total points earned on the task (see Table 4). For each region, participants who showed reduced neural tracking of social risk showed greater average knocking and total points earned, suggesting that age-related decreases in tracking of social risk was beneficial for participants’ performance on the task. Next, we performed mediation analyses to see whether changes in neural tracking of increasing social risk explained the relationship between age and behavioral performance on the task. We found that neural tracking in the bilateral insula as well as the MCC accounted for the relationship between age and both the average number of knocks, as well as the total number of points they earned during the task (Table 5).

### 4. Discussion

During the transition to adolescence, neural reactivity to both rewarding and socially-salient stimuli plays an important role in determining adolescent behavior (e.g., Perino et al., 2016). Previous research has highlighted that social information influences adolescents’

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**Table 2**

Neural Regions Showing Significant Main Effects.

| Anatomical Region | +/− | BA | x | y | z | t | k |
|-------------------|------|----|---|---|---|---|---|
| **Parametrically Increasing Risk** | | | | | | | |
| L Insula/IFG* | + | −30 | 20 | −8 | 6.60 | 344 |
| R Insula/IFG* | + | 30 | 20 | −11 | 7.80 | 448 |
| R ACC | + | 24/32 | 9 | 29 | 28 | 7.73 | 532 |
| SMA* | + | 6 | 6 | 11 | 5.10 | 11 |
| L VS* | + | −9 | 5 | −2 | 7.09 | 429 |
| R VS* | + | 12 | 5 | −2 | 6.75 | 153 |
| VTA | + | 3 | −25 | −5 | 7.07 | 11 |
| R Motor Cortex | + | 4 | 45 | 19 | 58 | 6.01 | 365 |
| L IFG* | + | 46 | −21 | 23 | 43 | −7.63 | 245 |
| R IFG* | − | 46 | 18 | 38 | 40 | −6.90 | 371 |
| L IFG* | − | 45 | −45 | 35 | 13 | −6.32 | 377 |
| vmPFC* | − | 10/11 | 3 | 59 | −11 | 5.90 | 606 |
| L ITG* | + | 20 | −57 | −58 | −14 | −8.91 | 7778 |
| PCC | +/− | 23/31 | 3 | −49 | 10 | −8.21 |
| Precuneus* | − | 7 | 0 | −61 | 37 | 6.86 |
| L Hippocampus | − | 21 | −19 | −14 | 6.09 |
| R Hippocampus | − | 31 | −16 | −17 | 5.85 |
| L Amygdala | − | −24 | −4 | −14 | 4.98 |
| R Amygdala | − | 18 | −4 | −17 | 3.69 |
| R TPJ | − | 39/40 | 42 | −61 | 31 | 6.29 |
| L TPJ | − | 39/40 | −48 | 67 | 28 | −7.93 | 1466 |
| L Motor Cortex | − | 4 | −45 | 16 | 52 | −7.71 |
| **Average Risk** | | | | | | | |
| L Inferior Occipital | − | −12 | −103 | 2 | 10.24 | 2847 |
| L Occipital Gyrus | − | 18 | −100 | 7 | 9.73 |
| L Gyrus | − | 18 | 52 | −26 | 9.48 |
| L Cerebellum | + | −42 | −58 | −23 | 7.37 |
| R IFG/Al* | + | 48 | 17 | 1 | 6.01 | 784 |
| R Parahippocampal | + | 18 | −1 | −14 | 5.71 |
| R Caudate | + | 12 | 5 | 7 | 4.11 |
| L Parahippocampal | + | −15 | −7 | 5.06 | 127 |
| L IFG/Al | +/− | −45 | 11 | −2 | 4.94 | 240 |
| R Posterior MeFG | + | 6 | 20 | 64 | 4.92 | 96 |
| L Auditory Cortex | − | −42 | −31 | 7 | 10.47 | 13466 |
| R Auditory Cortex | − | 48 | −22 | 7 | 8.73 |
| R STG | − | 60 | −19 | 7 | 8.75 |
| L Hippocampus | − | −33 | −40 | −8 | 7.33 |
| L mPFC | − | −15 | 62 | 4 | 5.17 |
| R mPFC* | − | 24 | 32 | 43 | 5.30 |
| Precuneus | − | −12 | −46 | 40 | 6.22 |
| L Occipital Gyrus | − | −33 | −79 | 25 | 6.48 |
| R Fusiform Gyrus | − | 30 | −43 | −8 | 382 |

Note: L and R refer to left and right hemispheres; + and − refer to positive or negative parametric tracking for regions in Parametrically Increasing Risk and positive or negative activation for regions in Average Risk; BA refers to Brodmann Area of peak voxel; k refers to the number of voxels in each significant cluster; t refers to peak activation level in each cluster; x, y, and z refer to MNI coordinates; voxel size = 3 mm³. Supercorrelation (e.g. a, b, etc.) indicate that peak voxels are part of a contiguous cluster. MCC = Mid-Cingulate Cortex; TPJ = Temporoparietal Junction; pSTS = Posterior Superior Temporal Sulcus; ACC = Anterior Cingulate Cortex; SFG = Superior Frontal Gyrus; *regions that survive at \( p < 0.001 \), \( k = 40 \).

**Table 3**

Neural Regions Showing Significant Age-related Differences.

| Anatomical Region | +/− | BA | x | y | z | t | k |
|-------------------|------|----|---|---|---|---|---|
| **Parametrically Increasing Risk** | | | | | | | |
| L Insula | − | −45 | 17 | −8 | 3.13 | 82 |
| R Insula* | − | 36 | 17 | −8 | 4.24 | 137 |
| MCC* | − | 31 | 12 | 20 | 34 | 3.78 | 146 |
| Posterior MCC | − | 23/31 | 15 | −10 | 43 | 3.68 |
| L TPJ | − | 39/40 | −54 | −52 | 28 | 3.88 | 151 |
| R TPJ | − | 39/40 | −60 | −43 | 25 | 3.11 | 78 |
| L pSTS | − | 22 | −51 | −40 | −2 | 4.59 | 56 |
| R Temporal Pole | − | 38 | −39 | 5 | −44 | 4.25 | 91 |
| R Ventral Striatum | − | 38 | 48 | 5 | −41 | 4.27 | 59 |
| L Fusiform | − | 37 | −24 | 64 | −8 | 4.22 | 61 |
| **Average Risk Decisions** | | | | | | | |
| L Inferior Parietal Lobule | − | −54 | 37 | 37 | 3.52 | 89 |
| R Cerebellum* | − | 48 | −55 | −26 | 3.84 | 448 |

Note: L and R refer to left and right hemispheres; + and − refer to positive or negative correlation with age; BA refers to Brodmann Area of peak voxel; k refers to the number of voxels in each significant cluster; t refers to peak activation level in each cluster; x, y, and z refer to MNI coordinates; voxel size = 3 mm³. Supercorrelation (e.g. a, b, etc.) indicate that peak voxels are part of a contiguous cluster. MCC = Mid-Cingulate Cortex; TPJ = Temporoparietal Junction; pSTS = Posterior Superior Temporal Sulcus; ACC = Anterior Cingulate Cortex; SFG = Superior Frontal Gyrus; *regions that survive at \( p < 0.001 \), \( k = 40 \).
decision-making, demonstrating that peers (e.g., Chein et al., 2011; Albert et al., 2013) and parents (e.g., Telzer et al., 2015b; Guassi Moriera and Telzer, 2016) have important impacts on adolescent behavior. Other work has shown that human and rodent adolescents can use information more flexibly than other age groups (e.g., Johnson and Willbrecht, 2011; McCormick and Telzer, 2017). As such, while previous paradigms have shown social influence on risky behavior, they have not yet explored how adolescents dynamically use (or disregard) social information when that information might interfere with goal-directed behavior. To explore this question, we employed a novel, social adaptation of the Balloon Analogue Risk Task. We show that there are developmental changes from childhood through adolescence, such that older participants showed decreased tracking of social risk in regions of the cortex involved in affective, social, and regulatory processing.

In contrast with previous theoretical (Blakemore and Mills, 2014; Nelson et al., 2016) and empirical (Lau et al., 2011; Guyer et al., 2012; Somerville et al., 2013; Knoll et al., 2015) research highlighting increased sensitivity to social information in adolescence, our results indicated that compared with younger participants, older adolescents showed decreased sensitivity to increasing negative social feedback in both regulatory (e.g., insula/IFG and MCC), as well as social processing regions (e.g., TPJ and pSTS, fusiform). Mid-cingulate and regions of the anterior insula/IFG have been implicated in behavioral regulation (Shackman et al., 2011; Hampshire et al., 2010), as well as salience monitoring and attention (Hampshire and Owen, 2010; Menon and Uddin, 2010), while the TPJ and pSTS are involved in mentalizing processes and inferring the thoughts and intentions of others (Blakemore and Mills, 2014; van den Bos, 2011; Frith and Frith, 2006). Follow-up analyses showed that suppressed sensitivity in these regions

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**Table 4**

Associations between regions showing age-related differences in tracking of social risk and task performance.

| Neural Regions | Average Knocks | Total Points | Number Slams |
|----------------|----------------|--------------|--------------|
| **Parametrically Increasing Risk Decisions** | | | |
| R Insula | −0.52*** | −0.60*** | −0.17 |
| L Insula | −0.35* | −0.43** | −0.08 |
| MCC | −0.56*** | −0.56*** | −0.33* |
| L pSTS | −0.19 | −0.28* | 0.04 |
| L TPJ | −0.29* | −0.34* | −0.11 |
| R TPJ | −0.27* | −0.31* | −0.12 |
| R FFA | −0.28* | −0.37* | −0.02 |
| L TP | −0.25 | −0.27* | −0.11 |
| R TP | −0.27 | −0.33* | −0.07 |

Note: * < 0.05, ** < 0.005, *** < 0.001. Associations between neural regions showing age-related increases in activation and task behavioral indices.

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**Table 5**

Testing mediation of age-related differences in task performance by differences in neural tracking.

| Neural Regions | B   | SE  | 95% CI |
|----------------|-----|-----|--------|
| **Average Knocks** | | | |
| L Insula | 0.04 | 0.02 | [0.0001, 0.09] |
| R Insula | 0.08 | 0.03 | [0.04, 0.14] |
| MCC | 0.08 | 0.03 | [0.03, 0.16] |
| **Total Points** | | | |
| L Insula | 0.81 | 0.36 | [0.20, 1.65] |
| R Insula | 1.6 | 0.48 | [0.78, 2.72] |
| MCC | 1.2 | 0.55 | [0.36, 2.52] |

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Fig. 2. We found age-related decreases for the tracking of increasing social risk (i.e., parametrically more risky knocks or “knocks PM”) in regions involved in social processing and salience-monitoring (L. TPJ, L. pSTS, and R Insula highlighted).
was beneficial for older participants’ task performance, contributing to a greater number of points earned on the task. Consistent regulatory activity might well be expected, with older adolescents being more effective and consistent in deploying regulatory resources than children or younger adolescents. However, reduced sensitivity in social processing regions (i.e., TPJ) may suggest that older adolescents were able to suppress the processing of negative social information associated with increased risk, perhaps by redirecting their attention, in order to enact goal-relevant behaviors. This account is supported by the TPJ’s role in attentional processing in both social (e.g., Saxe and Kanwisher, 2003; Schurz et al., 2014) and non-social contexts (e.g., Silvetti et al., 2016; Vossel et al., 2016), and in concert with decreased fusiform tracking in older participants, supports the idea that older adolescents may be decreasing their attention to the more-negative faces. As such, reduced tracking may allow adolescents to extract information from the negative social feedback without becoming overwhelmed, promoting a more adaptive behavioral strategy in the task.

The current study offers an exciting new perspective on adolescents’ sensitivity to social information. By pitting participants’ sensitivity to social stimuli against their sensitivity to risk and reward, we explored whether children and adolescents were able to selectively disregard social feedback when it conflicts with their goals. Results suggest that older adolescents’ success on the task is related to suppressed neural tracking of increasing social risk. This indicates that adolescents’ goals have a significant impact on how they process and attend to information in their environment in order to guide their behavior, and substantiates the hypothesis that adolescents are able to flexibly use social information rather than simply showing increased sensitivity regardless of context. Previous findings suggest the importance of taking into account adolescents’ goals when observing the impact of social information on their decisions. For instance, increases in risky behavior following peer rejection (Peake et al., 2013; Telzer et al., 2017) may partially reflect a top-down, conscious shift in adolescents’ social goals (e.g., impressing the observer) rather than a change in bottom-up motivational processes. This account may be more consistent with work on parental influence on risky behavior (e.g., Telzer et al., 2015b), where adolescents may consciously shift their behavior to safer patterns in order to avoid negative evaluation and feedback from their mothers. Testing these ideas is an important next step for the study of social influence on adolescent risk behavior.

Future work should additionally address some remaining questions. First, our sample examined these processes in children and adolescents; however, previous work has shown that significant development continues in regions of social processing well into early adulthood (Blakemore, 2008; Braams and Crone, 2017). Thus, future research should extend the age range examined here to explore whether goal-directed suppression of social information continues to improve or if these effects are specific to adolescence. Furthermore, future work should examine other metrics of maturation (i.e., pubertal development) that may contribute to increases in flexibility during risky decision-making in social contexts above and beyond age by itself. The current study also utilized a cross-sectional design in order to provide coverage of a wide range of ages across childhood and adolescence. However, follow-up longitudinal work will be necessary to confirm if within-person changes in the tracking of social risk follows the same pattern and is similarly adaptive. Finally, while we showed that changes in neural tracking in affective and social regions partially mediated age-related improvements in performance, our sample was relatively small, and future work should include more participants in order to better corroborate these findings.

The task used in the current manuscript also has the advantage of using dynamic social information that depends on participants’ own behavior. Dynamic social feedback in our task offers an advantage to addressing questions of developmental changes in processing social information by increasing the salience of the conflict between social information and participants’ own goals. However, the current study used adult faces as our dynamic social stimuli. An interesting remaining question is whether adolescents display the same reduced neural tracking of social information if age-matched stimuli were used (see Peake et al., 2013; Flannery et al., 2017 examples). Adolescence is a time of changing social relationships with both peers and adults (e.g., Nelson et al., 2016). Previous research has shown different reactions to parent and peer faces (Leibenluft et al., 2004) and peer, parent, and adult presence during risk taking (e.g., Chein et al., 2011; Telzer et al., 2015b; Guassi Moreira and Telzer, 2016), and as such, reactivity to same-age versus adult faces may differentially predict changes in risky behavior (e.g., Saxe et al., 2015), especially if adolescents become less motivated by adult disapproval across development. Future research should compare how adolescent risk taking changes in the presence of both peer and adult negative feedback to assess the generalizability of these results.

Finally, due to the nature of the SART, increasing risk of losing on a given trial and increasingly negative social feedback were linked, which prevents us from examining potential developmental change in the two processes separately. Although the reduced tracking in regions involved in social processing is suggestive of changes in social sensitivity driving these effects, future research should attempt to tease apart these two competing considerations by comparing social risk with other forms of risk (e.g. monetary). Furthermore, inherent in our theory is that increased risk-taking – and the negative consequences that follow – are often coincidentally driven by goal-directed behaviors. Future work should extrapolate to groups engaging in more destructive versus adaptive risk-taking to assess if the same neural patterns are observed or if there are differences in neural processing which explain why some may engage in dangerous risks versus those who engage in adaptive risks. Finally, since social information used in the current study was inherently negative, future work should extend these findings to contexts where positive social feedback is paired with risky behavior to see if adolescents are also able to flexibly use positive social information.

In conclusion, we implemented a novel, social version of the BART, combining risky decision-making with dynamic social feedback in order to examine adolescents’ ability to disregard social information during goal-directed behavior. Our results suggest that while social information may be particularly important for adolescents’ decision-making processes, they can also ignore that information in the pursuit of other goals. These findings support the idea that the increase in risky behavior in the presence of peers seen in previous research (e.g., Chein et al., 2011) may reflect a top-down shift in adolescents’ goals rather than a bottom-up motivational shift in reward-sensitivity, suggesting that adolescents can flexibly use or ignore social information in goal-relevant pursuits (Crone and Dahl, 2012). This exciting new perspective can not only help enrich our understanding of how sensitivity to social and rewarding stimuli contributes to adolescent behavior, but also the context-dependent nature of that sensitivity.

Conflict of interest
None.

References
Albert, D., Chein, J., Steinberg, L., 2013. The teenage brain peer influences on adolescent decision making. Curr. Dir. Psychol. Sci. 22 (2), 114–120.
Blakemore, S.J., Mills, K.L., 2014. Is adolescence a sensitive period for sociocultural processing? Annu. Rev. Psychol. 65, 187–207.
Blakemore, S.J., 2008. The social brain in adolescence. Nat. Rev. Neurosci. 9 (4), 267–277.
Braams, B.R., Crone, E.A., 2017. Peers and parents: a comparison between neural activation when winning for friends and mothers in adolescence. Soc. Cogn. Affect. Neurosci. 12 (3), 417–426.
Brener, N.D., Kann, L., Shanklin, S., Kinchen, S., Eaton, D.K., Hawkins, J., Flint, K.H., 2013. Methodology of the youth risk behavior surveillance system—2013. Morb. Mort. Wkly. Rep. 62 (1), 1–20.
Burnett, S., Blakemore, S.J., 2009. Functional connectivity during a social emotion task in adolescents and in adults. Eur. J. Neurosci. 29 (6), 1294–1301.
Burnett, S., Bird, G., Moll, J., Frith, C., Blakemore, S.J., 2009. Development during adolescence of the neural processing of social emotion. J. Cogn. Neurosci. 21 (9), 1736–1750.

Casey, B.J., Tottenham, N., Monti, M.P., Cato, E., Peña, M., La Morgia, C., Anderson, A., 2005. The social re-orientation of adolescents: an expanded and updated view. Dev. Cogn. Neurosci. 17, 118–127.

Casey, B.J., Tottenham, N., Liston, C., Durston, S., 2012. Understanding adolescence as a period of social–affective engagement and goal flexibility. Nat. Rev. Neurosci. 13 (9), 636–650.

Falk, E.B., Cascio, C.N., O'Donnell, M.B., Carp, J., Tinney, F.J., Bingham, C.R., Simons-Morton, B.G., 2014. Neural responses to exclusion predict social sensitivity in adolescence. J. Adolesc. Health 54 (5), 522–531.

Flannery, J.E., Giuliani, N.R., Flourny, J.C., Pfeifer, J.H., 2017. Neurodevelopmental changes across adolescence in viewing and labeling dynamic peer emotions. Dev. Cogn. Neurosci. 25, 113–127.

Galván, A., 2010. Adolescent development of the reward system. Front. Hum. Neurosci. 4, 6.

Gardner, M., Steinberg, L. 2005. Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: an experimental study. Dev. Psychol. 41 (4), 605.

Gorgolewski, K.J., Varoquaux, G., Rivera, G., Schwartz, 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. Front. Neuroinform. 9, 8.

Guastella, J.F., Silvetti, M., Lasaponara, S., 2016. Mother–child touch sensitivity toward safe behavior during risk taking. Dev. Sci. 19 (2), 259–268.

Guyer, A.E., Chaste, V.R., Pine, D.S., Nelson, E.E., 2012. Neural development supporting the understanding of peer-based motivation. Dev. Sci. 15 (6), 1638–1648.

Hayes, A.F., 2013. Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach. Guildford Publications.

Hampshire, A., Chamberlain, S.R., Monti, M.M., Dunn, J., Owen, A.M., 2010. The role of the right inferior frontal gyrus: inhibition and attentional control. Neuroimage 50 (3), 1313–1319.

Hare, T.A., Tottenham, N., Galván, A., Voss, H.U., Glover, G.H., Casey, B.J., 2008. Biological substrates of emotional reactivity and regulation in adolescence during an experimental go-nogo task. Psychol. Med. 38 (13), 2127–2134.

Hayes, A.F., 2013. Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach. Guildford Publications.

Hampshire, A.R., Galán, C.A., Tottenham, N., Lee, S.S., 2015. Impaired social decision-making mediates the association between ADHD and social problems. J. Abnorm. Child Psychol. 1–10.

Knoll, L.J., Magis-Weinberg, L., Speekenbrink, M., Blakemore, S.J., 2015. Social influence on risk perception during adolescence. Psychol. Sci. 26 (5), 583–592.

La Greca, A.M., Prinstein, M.J., Fetter, M.D., 2001. Adolescent peer crowd affiliation: linkages with health-risk behaviors and close friendships. J. Pediatr. Psychol. 26 (3), 131–139.

Laird, R.D., Jordan, K.Y., Dodge, K.A., Pettit, G.S., Bates, J.E., 2001. Peer rejection in childhood, involvement with antisocial peers in early adolescence, and the developmental course of antisocial behavior. J. Child Psychol. 1–10.

Leibenluft, E., Lejuez, C.W., Read, J.P., Kahler, C.W., Richards, J.B., Ramsey, S.E., Stuart, G.L., Brown, T.M., 2004. Mothers’ neural activation in response to pictures of their children and other children. Biol. Psychiatry 56 (4), 225–232.

Leibenluft, E., Gobbini, M.I., Harrison, T., Haxby, J.V., 2004. Mothers’ neural activation in response to pictures of their children and other children. Biol. Psychiatry 56 (4), 225–232.

Leibenluft, E., Lejuez, C.W., Read, J.P., Kahler, C.W., Richards, J.B., Ramsey, S.E., Stuart, G.L., Brown, T.M., 2004. Mothers’ neural activation in response to pictures of their children and other children. Biol. Psychiatry 56 (4), 225–232.

Lau, J.Y., Guyer, A., Tone, E.B., Jenness, J., Parrish, J.M., Pine, D.S., Nelson, E.E., 2009. Measuring the neural correlates of anticipated peer evaluation in adolescence. Child Dev. 80 (4), 1000–1015.

Laird, R.D., Jordan, K.Y., Dodge, K.A., Pettit, G.S., Bates, J.E., 2001. Peer rejection in childhood, involvement with antisocial peers in early adolescence, and the developmental course of antisocial behavior. J. Child Psychol. 1–10.

Levine, J., Gordon, J., Fraser, D., Johnson, D.C., Jing, D., Elliott, M.D., Pattwell, S.S., Soliman, E.M., McCormick, et al. 2018. 134–141 Developmental Cognitive Neuroscience 30 (2018) 134–141.