Adaptive Adolescent Flexibility: Neurodevelopment of Decision-making and Learning in a Risky Context

Ethan M. McCormick and Eva H. Telzer

Abstract

■ Research on adolescence has largely focused on the particular biological and neural changes that place teens at risk for negative outcomes linked to increases in sensation-seeking and risky behavior. However, there is a growing interest in the adaptive function of adolescence, with work highlighting the dual nature of adolescence as a period of potential risk and opportunity. We examined how behavioral and neural sensitivity to risk and reward varies as a function of age using the Balloon Analog Risk Task. Seventy-seven children and adolescents (ages 8–17 years) completed the Balloon Analog Risk Task during an fMRI session. Results indicate that adolescents show greater learning throughout the task. Furthermore, older participants showed increased neural responses to reward in the OFC and ventral striatum, increased activation to risk in the mid-cingulate cortex, as well as increased functional OFC–medial PFC coupling in both risk and reward contexts. Age-related changes in regional activity and interregional connectivity explain the link between age and increases in flexible learning. These results support the idea that adolescents' sensitivity to risk and reward supports adaptive learning and behavioral approaches for reward acquisition. ■

INTRODUCTION

Adolescence has been largely recognized as a period of heightened risk and poor decision-making; however, adolescence is also a period of opportunity for learning and skill acquisition. Although neurodevelopmental research has begun to shed light on neural mechanisms that support changes in risk-taking and sensation-seeking behaviors during adolescence (Steinberg et al., 2008), empirical work and theoretical models of adolescent brain development focus on how these behaviors are the result of deficient or ineffective circuitry (see Telzer, 2016). Several neurobiological models have proposed that early-maturing subcortical regions coupled with slower-developing prefrontal regions underlies increased risk taking during adolescence (Steinberg, 2010; Casey, Jones, & Hare, 2008; Ernst, Pine, & Hardin, 2006), comparing adolescent behavior to a car in full throttle but with ineffective breaks (Steinberg, 2010). Although these heuristics are useful tools (see Casey, 2015; but see Pfeifer & Allen, 2016), they can pathologize adolescence as a period of deficiency and overlook the potentially adaptive role of adolescence as a period of opportunity for learning and the acquisition of new ideas, skills, and interests (Crone & Dahl, 2012).

Emerging evidence supports the idea of adolescence as a period of adaptive flexibility. Adolescent rodents (Pattwell et al., 2012), nonhuman primates (Spear, 2000), and humans (Humphreys, Lee, & Tottenham, 2013) show behavioral patterns that support increased flexibility, even at potential risk to their health and reproductive success. For instance, human adolescents show age-related increases in risk taking as well as adolescent-specific increases in learning in a risk-taking context (Humphreys et al., 2016), and adolescents show greater tolerance for ambiguity during risk taking than do adults (Tymula et al., 2012), which might promote learning during adolescence. Adolescent mice also show increased flexibility and learning when pursuing rewards (Johnson & Wilbrecht, 2011). This flexibility supports adolescents' learning of the environment and helps them gain access to food and reproductive opportunities (Vigilant et al., 2015). In light of this research, some have suggested that the unique configuration of adolescent neural systems serves an adaptive function necessary for appropriate development (Casey, 2015; Crone & Dahl, 2012).

Although no empirical studies have explored the neurodevelopment of learning and flexible behavior in risky contexts, some initial evidence highlights the potentially adaptive function of still-developing neural states for learning. Although the heuristic models utilized in adolescent neurodevelopmental research generally highlight the maladaptive nature of delayed prefrontal development (see Casey, 2015), slower maturation of PFC may actually promote an individual's ability to flexibly adapt to new contexts. For instance, early adversity (e.g., maternal deprivation, neighborhood violence) is associated with accelerated life history trajectories (Ellis, Figueredo, Brumbach, & Schlomer, 2009) including early transition to adult-like PFC functioning (Gee et al., 2013). Although this acceleration is hypothesized to serve a compensatory role, early transition to adult neural states is also associated with developmental trade-offs that can result in

University of North Carolina, Chapel Hill

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suboptimal outcomes such as decreases in plasticity and academic achievement (Shaw et al., 2006), suggesting that later-developing PFC function may be adaptive and support learning and skill acquisition.

Despite this initial evidence, we know relatively little about neurodevelopmental mechanisms that support age-related changes in flexibility and learning. To address this gap, we examined flexible learning in the context of risk and reward contingencies. Youth ages 8–17 years completed the Balloon Analog Risk Task (BART; Lejuez et al., 2002) during an fMRI session. The BART mirrors real-world behavior in that risky behavior is rewarded up until a point but then becomes detrimental to the individual's goals. The task creates a context for investigating learning since participants can use feedback they receive on each trial to modify or reinforce their behavior (Humphreys et al., 2016). We examined age-related changes in risk-taking behavior across the task as well as age-related differences in neural activation and connectivity in motivational (e.g., ventral striatum [VS] and OFC) and regulatory (e.g., lateral PFC and anterior cingulate) regions involved in learning and goal-directed behavior. We hypothesized that adolescents would be more likely than younger participants to explore and better learn the parameters of the task. Adolescents could then utilize this learning to guide their risk-related behavior in pursuit of rewards. We further hypothesized that neurodevelopmental changes in motivational and regulatory regions would mediate these age-related increases in flexible learning.

METHODS

Participants

Eighty healthy children and adolescents completed an fMRI scan. Two participants were excluded because of excessive head motion (>2.0 mm slice-to-slice on ≥10% of slices) during the session, and an additional participant was excluded because of corrupted/missing data, leaving 77 participants in the final sample (41 girls; M_{age} = 14.23 years, SD = 2.76, range = 8.1–17.7 years). Participants (54 European American, 18 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.

Risk and Reward Task

Participants completed a version of the BART, a well-established experimental paradigm (Qu, Galvan, Fuligni, Lieberman, & Telzer, 2015; Telzer, Fuligni, Lieberman, Miernicki, & Galván, 2015; Lejuez et al., 2002) that measures participants' willingness to take risks in the pursuit of rewards. Before the scan, participants were shown a box of age-appropriate prizes and were told that the more points they earned on the task, the more prizes that they could select at the end of the neuroimaging session. In reality, all participants were allowed to choose three prizes regardless of the number of points they earned. During the scan, participants were presented

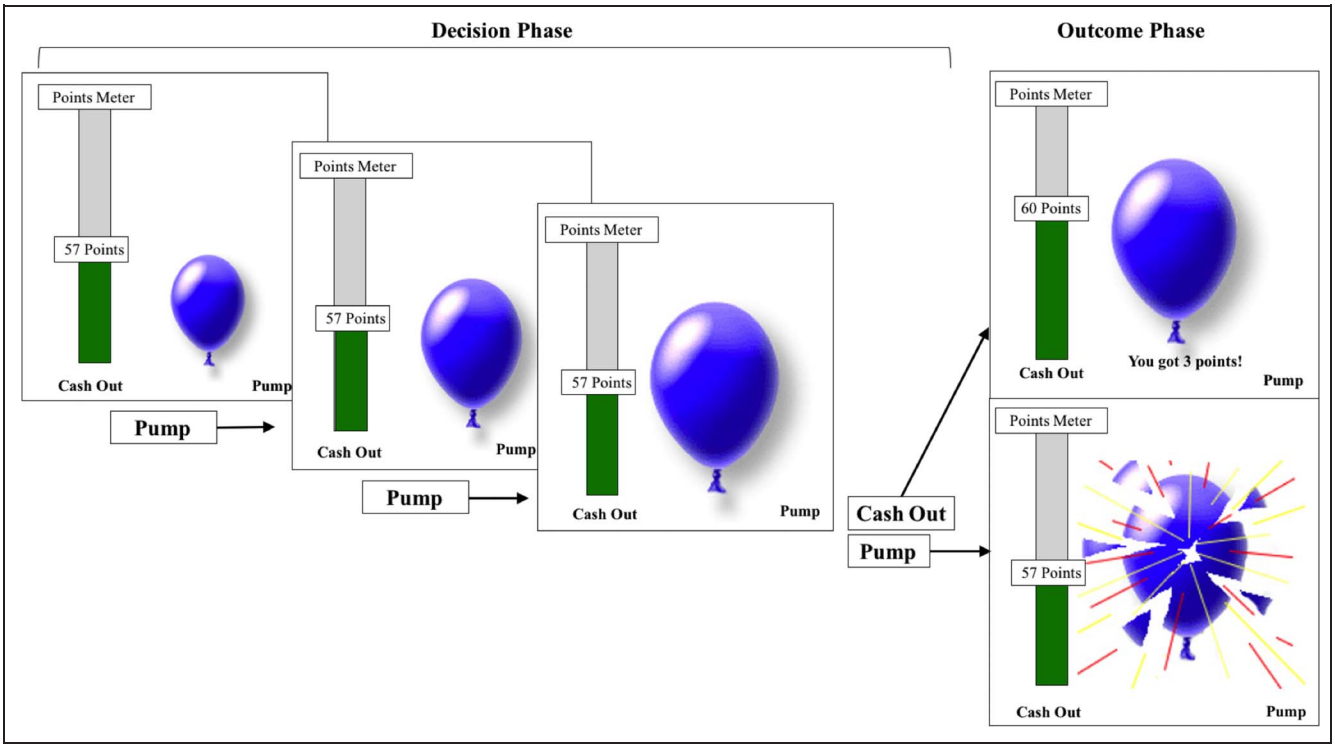

Figure 1. BART. Participants can choose to Pump to increase the size of the balloon or to Cash Out to add points to their Points Meter. However, if participants pump too many times, the balloon will explode.

with a series of 24 balloons that they could choose to pump up in order to accrue points (Figure 1). Each pump increased the risk that the balloon would explode, and if the balloon exploded, participants lost all points they had accrued from that balloon. At any point after the first pump, participants could choose to cash out their points for that balloon, which were added to their total for the task. The running total of points earned was presented on the screen as a points meter. Participants were instructed that their goal was to earn as many points as they could during the task. Each event (e.g., larger balloon following a pump, new balloon following cashed or exploded trial) was separated with a random jitter (500–4000 msec). Balloons were presented in a fixed order, with the explosion rate ranging from 4 to 10 pumps, although this was not made explicit to participants. The task was self-paced and would not advance unless the participant made the choice to either pump or cash-out.

Behavior Modeling

We measured several indices of behavior to tap risk behavior and learning on the task. "Risk behavior" represents participants' willingness to engage in risk taking. This was calculated as the average number of pumps on cashed trials. The number of pumps on explosion trials was not included because those trials are artificially constrained and end before participants have reached their maximum tolerance for risk (Lejuez et al., 2002). This metric has been used widely as an index of risk taking and is associated with higher levels of self-reported risk-taking behavior in the real world both concurrently and longitudinally (Qu et al., 2015; Telzer et al., 2015; Lejuez et al., 2002).

"Learning" was indexed by participants' feedback sensitivity or how likely they are to use information from the previous trial to guide their behavior on each subsequent trial and to adapt when their current behavior is resulting in maladaptive outcomes (Humphreys et al., 2015). To obtain this index, we used hierarchical linear modeling (Raudenbush & Bryk, 2002), in which trials (24 total) were nested within participants, and the outcome variable was the number of pumps on a given trial. We modeled whether the number of pumps on a given trial varied depending on the outcome of the previous trial. Consistent with prior studies (Ashenhurst, Bujarski, Jentsch, & Ray, 2014; Mata, Hau, Papassotiropoulos, & Hertwig, 2012), our Level 1 equation was

$$\begin{aligned}\text{Number of Pumps}_{ij} = {} & b_{0j} + b_{1j}\left(\text{Explosion}_{(N-1)}\right) \\ & + b_{2j}\left(\text{Explosion}_{(N)}\right) \\ & + b_{3j}(\text{Trial Number}) + \varepsilon_{ij}\end{aligned}$$

Total pumps on a particular trial (i) for a particular adolescent (j) was modeled as a function of the average number of pumps across the task (b_{0j}) and whether the previous trial (b_{1j}) was an explosion or cash-out (coded $\text{Explosion}_{(N-1)} = 0$; $\text{Cash-Out}_{(N-1)} = 1$). In addition, we included two controls, including whether the current trial resulted in an explosion or a cash-out (b_{2j}; coded $\text{Explosion}_{(N)} = 1$; $\text{Cash-Out}_{(N)} = 0$) and the trial number ($b_{3j}$).

To use the learning index in our neural and behavioral analyses, we extracted empirical Bayes estimates for each participant. Empirical Bayes estimates are optimally weighted averages that combine individual average slopes by combining estimates from both the individual and the group and "shrink" individual specific estimates toward the overall mean (Diez-Roux, 2002). The extracted estimate represents individual differences in how participants change their subsequent behavior (both magnitude and direction) based on the type of feedback they received on the prior trial. Larger positive values (e.g., >0) are indicative of greater learning (i.e., participants increase pumps following a cashed balloon but decrease pumps following an exploded balloon), whereas values closer to zero indicate little or no learning (i.e., participants increased or decreased their pump behavior at random with respect to previous feedback). Although negative values (e.g., <0) are possible, this would indicate that participants were increasing pumps after explosions and decreasing pumps after cash-outs, an especially irrational strategy.

Additional behavioral measures included *number of explosions* or the number of times participants pumped balloons until they popped as well as *total points* earned on the task, which represents participants' successful acquisition of resources. Higher total point values are indicative of more optimal behavior on the task.

fMRI Data Acquisition

Imaging data were collected using a 3-T Siemens Trio MRI scanner (Siemens, Berlin, Germany). The BART included T2*-weighted EPI (slice thickness = 3 mm, 38 slices, repetition time [TR] = 2 sec, echo time [TE] = 25 msec, matrix = 92 × 92, field of view [FOV] = 230 mm, voxel size = 2.5 × 2.5 × 3 mm^3). In addition, structural scans consisted of a T2*-weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4 sec, 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 sec, 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.

fMRI Data Preprocessing and Analysis

Preprocessing and data analysis utilized Statistical Parametric Mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK) software

package. Preprocessing steps involved spatial realignment to correct for head motion (included participants had no motion in excess of 1.5 mm between-slice motion); coregistration of all images to the high-resolution T1* MPRAGE structural scan; and segmentation into gray matter, white matter, and cerebrospinal fluid. Transformation matrices used in MPRAGE segmentation were applied to MBW and EPI images to warp them into the standard stereotactic space defined by the Montreal Neurological Institute (MNI) and the International Consortium for Brain Mapping. EPI images (voxel size = 3 mm^3) were smoothed using an 8-mm Gaussian kernel, FWHM to increase signal-to-noise ratios in the functional images. The general linear model in SPM8 was then used to convolve each trial with a canonical hemodynamic response function. Low-frequency drift across the time series was removed using a high-pass temporal filter with a 128-sec cutoff, and a restricted maximum likelihood algorithm with an autoregressive model order of 1 was used to estimate serial autocorrelations.

The BART was modeled using an event-related design with trial duration corresponding to participant RT on a given pump or cash-out or using the average RT across the task on explosions. Fixed-effects models included a general linear model for each condition of interest, which included pump decisions, cash-out decisions, and explosion events. We modeled pump decisions separately for trials that ended in cash-outs and trials that ended in explosions. Because the number of pumps is artificially constrained on balloons that end in explosions, analyses were only performed with pump decisions on balloons that ended in cash-outs, as done in prior research (Telzer et al., 2015; Lejuez et al., 2002). The jittered intertrial periods were not modeled and served as the implicit baseline for the task. A parametric modulator (PM) was included for each of the three conditions of interest and represents the pump number for a balloon at each pump or cash-out decision. All the PM values were mean-centered by balloon within participants, such that for each balloon, all PM values summed to 0. The PM served to control for differences across pumps within a balloon trial. Contrasts were then computed at the individual level for each condition of interest.

In addition, we examined neural connectivity by conducting psychophysiological interaction (PPI) analyses. We used structurally defined ROIs (Wake Forest University PickAtlas; Maldjian, Laurienti, Kraft, & Burdette, 2003) as the seed regions, including the medial OFC and bilateral VS. These regions have been strongly implicated in reward-related associative learning, being involved in the formation and manipulation of stimulus–reward expectations (Schoenbaum & Roesch, 2005; Kelley, 2004; Gottfried, O'Doherty, & Dolan, 2003) and as such may be involved in developmental processes that support exploration and learning. PPI analyses utilized a generalized form of context-dependent PPI form the automated generalized PPI toolbox in SPM (McLaren, Ries, Xu, & Johnson, 2012). Deconvolved time series were extracted from the medial OFC and VS ROI for each participant to create the physiological variables. Each trial type was then convolved with the canonical HRF to create the psychological regressor. Finally, the physiological variable was multiplied with the time series from the psychological regressors to create the PPI term. This interaction term was then used to identify regions that covary with the seed region in a task-dependent manner. Each participant has a regressor computed that represents the deconvolved BOLD signal, which was included alongside each psychological and PPI term for each event type to create a generalized PPI model.

Random effects, group level analyses were run on all individual subject contrasts using GLMFlex, which corrects for variance–covariance inequality, removes outliers and sudden activation changes in the brain, partitions error terms, and analyzes all voxels containing data (mrtools.mgh.harvard.edu/index.php/GLM_Flex). Because not all participants had sufficient explosion events to model successfully, group level analyses focused on pump and cash-out decisions. Group-level analyses involved whole-brain regressions using age as a continuous covariate. Correction for multiple comparisons was run using a Monte Carlo simulation through the updated version (April 2016) 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 $p < .001$ and a minimum cluster size of 46 voxels for the whole brain, corresponding to $p < .05$, family-wise error corrected.

Finally, mediation analyses tested how age was associated with behavioral indices of task performance via brain activation during the task. Mediation was performed using the PROCESS macro methods outlined by Hayes (2013). All variables of interest were standardized before entering them into mediation models and using 1000 sample bootstrapping, the magnitude and significance of the indirect effect as well as a bias-corrected confidence interval (CI) were calculated. For all mediation models, age was entered as the predictor variable, the brain as the mediator, and behavioral indices as the outcome. Mediators were added into separate models such that each model only contained one behavioral measure or brain region as the mediator.

RESULTS

Behavioral Results

Age-related Increases in Risk and Learning

We ran bivariate correlations between age of participants and behavioral indices of interest (see Table 1 for means, *SD*s, ranges, and correlations between all study variables). Age was associated with more risk behavior (i.e., higher average pumps; $r = .36$, $p = .001$) and learning (i.e., pumping more after a cash-out and less after an explosion;

Table 1. Descriptives and Correlations for Study Variables of Interest

				Correlations				
Variable	*M*	*SD*	*Range*	*1*	*2*	*3*	*4*	*5*
1. Age	14.10	2.76	8.10–17.7	1	.36****	.51****	.21*	.36****
2. Risk behavior	4.50	0.77	2.78–6.21		1	.82****	.63****	.83****
3. Learning	0.87	0.32	0.24–1.32			1	.50****	.74****
4. Total points	81.83	8.32	63–106				1	.12
5. Number of explosions	5.48	2.41	1–12					1

Numbers along the diagonal represent Pearson's correlations.

*$p < .1$, **$p < .05$, ***$p < .01$, ****$p < .005$.

$r = .51, p < .001$). Moreover, increased learning was associated with higher points earned ($r = .50, p < .001$), suggesting the utility of learning and its downstream influence on how participants acquire adaptive outcomes.

Learning Explains Age Differences in Risk-taking Behavior

Next, we examined whether older participants' increased learning explained the link between age and risk taking during the BART. In other words, does the propensity of older youth to learn more from the task environment explain why they tend to take more risks across the task? As shown in Figure 2, we found a significant indirect effect such that the degree of learning exhibited by participants mediates the relationship between age and risk behavior during the task, suggesting that older adolescents' greater risk-taking behaviors is explained, in part, because they are learning from the parameters of the task to a greater extent.

We also examined whether this increased learning and the increased propensity to take risks benefits participants (i.e., they would earn more points) or whether the associated costs of increased explosions would offset their higher rates of pumping (i.e., they would earn fewer points). We found that participants' risk behavior mediates the relationship between learning and the total number of points that participants earned (Figure 2). In other words, participants who show heightened levels of learning are more likely to earn more points because they engage in greater amounts of risk behaviors. Together, results demonstrate that older participants' show increases in learning and risk taking across the task and that these behavioral patterns serve an adaptive function with respect to resource acquisition.

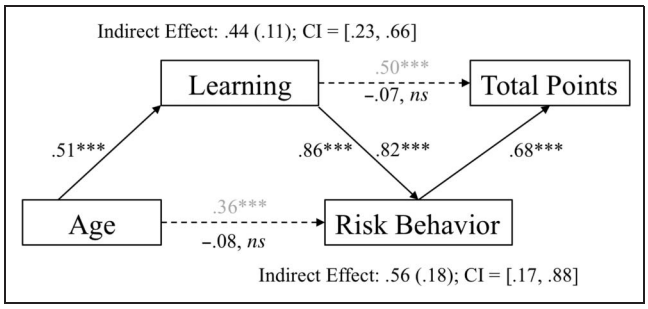

Figure 2. Learning mediates the link between age and risk behavior, which is associated with more total points. Direct effects are indicated by the coefficients (grayed-out) above the dashed lines. For the path from Learning to Risk Behavior, coefficients to the left are for the first model and coefficients to the right are for the second. For indirect effects, coefficients are standardized with the *SD* in parentheses, and all other coefficients are standardized. *$p < .05$, **$p < .01$, **$p < .001$.

fMRI Results

Age-related Differences in Risk- and Reward-related Neural Activity

We examined the effects of age on our conditions of interest by entering Age as a continuous regressor in whole-brain regression analyses (for main effects without Age, see Table 2). Areas showing age-related increases in risk-related activity (i.e., during pumps) included regions of the mid-cingulate cortex (MCC) and bilateral calcarine gyrus, with an additional cluster in the right superior frontal gyrus (SFG) nearing threshold ($k = 42$). For reward-related activity (i.e., during cash-outs), we found age-related increases in the VS and medial OFC. No regions showed significant age-related decreases during risk or reward (Table 3; Figure 3).

Links between Age-related Neural Activation and Learning

Next, we examined whether regions showing age-related increases in activation were associated with learning. To do so, we extracted parameter estimates of signal intensity from the regions that showed significant age effects and performed mediation analyses to examine whether activity in these regions explained the link between age and learning. Correlation analyses indicated that all regions showing age-related increases in activation were related to learning (Table 4). However, mediation analyses indicate that reward-related activity in the medial OFC was the only region to significantly explain the link

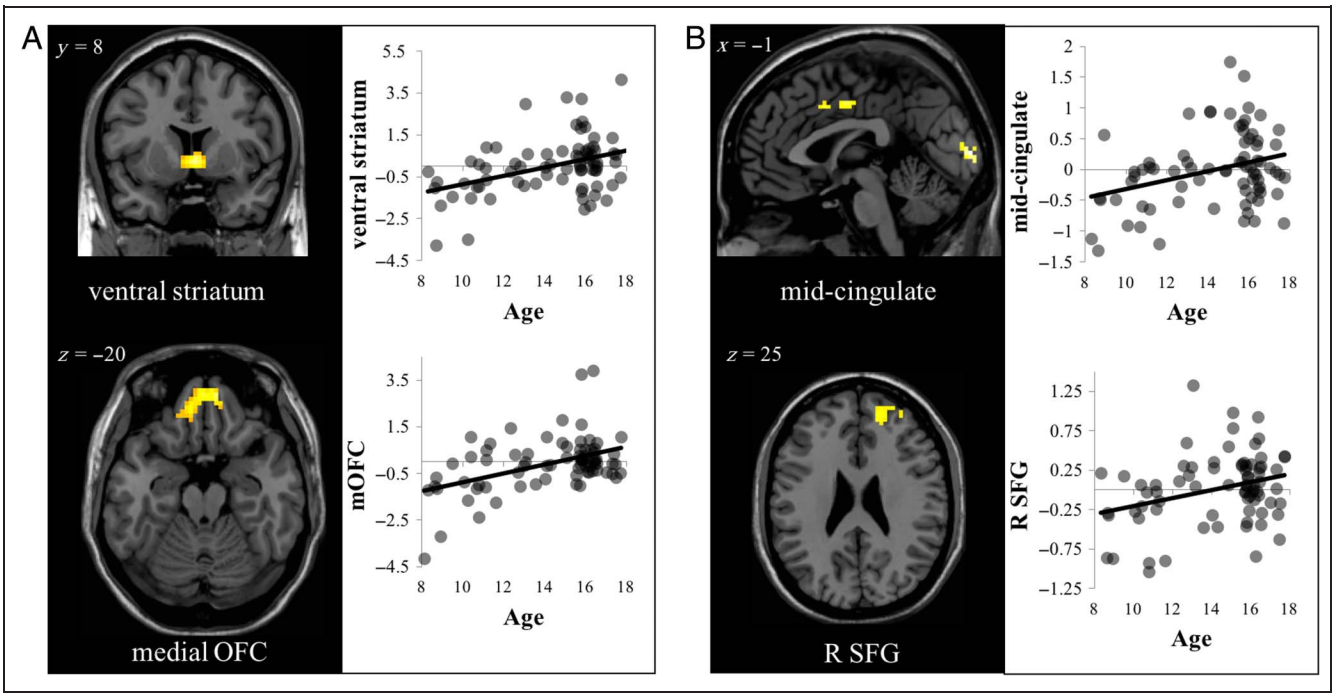

Figure 3. (A) During reward (e.g., cash-outs), we found age-related increases in VS and medial OFC activation. (B) During risk (e.g., pumps), we found age-related increases in MCC and R SFG activation.

between age and learning (indirect effect: $B = .11$, $SE = .05$, 95% CI [.03, .23]). These findings suggest that developmental differences in these regions support increased learning within the task environment observed in older adolescents.

Functional Connectivity

Age-related Changes in Connectivity during Risk and Reward

Next, we ran PPI analyses using our medial OFC and VS seed regions (for main effects without Age, see Table 2). We entered Age as a regressor in whole-brain PPI analyses. We found that the medial OFC shows age-related increases in functional connectivity with the medial PFC (mPFC) during risk and with the mPFC and posterior cingulate cortex (PCC) during reward (Table 3; Figure 4). There were no regions that showed age-related decreases in connectivity with the medial OFC during either condition. We found no regions that showed age-related change in VS connectivity.

Links between Age-related Neural Connectivity and Learning

Finally, we examined whether age-related differences in OFC–mPFC connectivity explain age-related differences in learning. Correlation analyses indicate that all regions showing age-related increases in OFC connectivity during both risk and reward were related to increased learning (Table 4); however, only age-related increases in OFC–mPFC functional connectivity during reward significantly explain the link between age and learning (indirect effect: $B = .12$, $SE = .05$, 95% CI [.04, .25]).

DISCUSSION

A major focus of research on neural development during adolescence has been the neural mechanisms that support changes in risk taking and sensation seeking (Casey, 2015). However, much of the theoretical and empirical work on adolescent neural development has highlighted aspects of adolescent neural circuitry, which are deficient or ineffective, while ignoring potentially adaptive roles for developing neural circuits (see Telzer, 2016; Casey, 2015). In contrast, we focused on aspects of adolescent neurodevelopment that might support learning and, in turn, adaptive outcomes. Our findings highlight adolescence as a period of behavioral and neural flexibility, which leads to increases learning within risky contexts. Additionally, this flexibility can drive behaviors that extract adaptive outcomes from these contexts, suggesting that a more-nuanced view of adolescence is warranted. Instead of characterizing still-developing neural systems as deficient, developmentally appropriate neural circuitry can play an adaptive role in adolescent behavior.

Consistent with prior research, we found that participants showed age-related increases in risk taking. Supporting the theory that increased exploration of the environment, even at potential risk, supports adaptive behavior, we found that age-related increases in learning (i.e., participants' successfully exploring the task parameters and changing their behavior in response to

Table 2. Neural Regions Showing Significant Activation during Risk and Reward in the Main Effects and PPI Analyses

Anatomical Region	+/−	*BA*	*x*	*y*	*z*	*t*	*k*
Main Effect							
Risk							
L insula	+		−30	20	7	8.82	479
R insula	+		33	23	7	5.94	219
ACC	+	24/32	3	26	31	8.00	710
L MFG	+	9	−33	53	25	5.06	120
R MFG	+	9/46	36	44	34	4.86	53
L postcentral gyrus	+		−63	−22	25	6.15	258
L IFG (pars triangularis)	−	45	−36	11	28	−5.67	171
R IFG (pars triangularis)	−	45	48	29	22	−4.38	155
PCC	−	23/31	6	−46	34	−5.13	438
Reward							
L insula[a]	+		−30	17	1	11.30	32470
R insula[a]	+		33	23	−2	11.07	
ACC[a]	+	24/32	3	29	31	10.83	
R VS[a]	+		21	11	−2	8.42	
L VS[a]	+		−18	14	−5	7.92	
R MFG[a]	+	9	33	−70	31	7.42	
L MFG[a]	+	9/46	−45	41	22	6.75	
R lateral OFC[a]	+	11	21	41	−20	6.76	
L lateral OFC	+	11	−27	50	−14	6.61	93
Medial OFC	−	11	−6	56	−8	−4.56	65
PPI (Medial OFC Seed)							
Risk							
Ventromedial PFC	+	10/11	0	50	−17	17.20	44147
PCC	+	23/31	0	−49	37	12.61	
R amygdala	+		21	−7	−17	7.57	
L amygdala	+		−21	−10	−17	8.51	
L SFG	+	8/9	−18	35	43	9.56	
R SFG	+	8	24	32	46	8.30	
L VS	+		−9	14	−8	8.06	
Reward							
Ventromedial PFC[b]	+	10/11	−3	50	−17	14.30	49598
VS[b]	+		0	8	−8	5.90	
Superior mPFC[b]	+	9/10	−9	44	46	9.38	
PCC[b]	+	23/31	−3	−49	25	12.92	
R IFG[b]	+	45	51	32	−8	8.86	
L IFG[b]	+	45	−51	29	−2	8.33	
PPI (VS Seed)							
Risk							
R caudate[c]	+		0	−10	10	14.38	56455
L caudate[c]	+		−12	−7	13	12.02	
L amygdala[c]	+		−18	−1	−14	11.48	
R amygdala[c]	+		18	5	−14	10.34	
dACC[c]	+	24/32	−3	32	28	11.52	
PCC[c]	+	23/31	3	−27	25	11.77	
Reward							
R caudate[d]	+		6	−10	10	11.78	49598
L putamen[d]	+		−18	11	7	11.41	
R putamen[d]	+		21	15	−5	11.35	
L amygdala[d]	+		−18	−1	−14	9.34	
PCC[d]	+	23/31	0	−40	22	10.13	
dACC[d]	+	24/32	3	32	31	9.62	

L and R refer to left and right hemispheres; + and − refer to positive or negative activation; BA refers to Brodmann's 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 mm^3. Superscripts (e.g., a, b, etc.) indicate that peak voxels are part of a contiguous cluster. dACC = dorsal ACC, MFG = middle frontal gyrus, IFG = inferior frontal gyrus.

feedback) explained age-related increases in the tendency to take more risks during adolescence, and greater risk taking was linked to greater acquisition of points. Although previous work has suggested the potential utility of risk taking during adolescence (Spear, 2000), the current literature generally discusses and tests how increased risk taking during adolescence is impulsive and irrational behavior driven by increases in sensitivity to motivational stimuli (see Casey, 2015; Steinberg et al., 2008). Results in this study suggest that risk taking may emerge, in part, from an increased ability to flexibly learn from the environment during adolescence. Such learning from environmental feedback likely plays an adaptive role in adolescent skill acquisition, establishment of new social networks, and identity formation.

Context is an important determinate of whether a propensity to take risks is adaptive or maladaptive (Humphreys et al., 2013). In an uncertain environment, increased risk taking may expose adolescents to opportunities for learning, which in turn may help the individual to increase the likelihood of attaining adaptive outcomes. When we consider the evolutionary history of adolescence, it is likely that there are trade-offs on a population level for a developmental period marked by increased risk, where the risk of exposure to detrimental outcomes is weighted against the opportunities for food and mate resources that exploration promotes (Spear, 2000). This study suggests that, as children transition into adolescence, they are more willing to

Table 3. Neural Regions Showing Age-related Increases during Risk and Reward in Activation and PPI Analyses

Anatomical Region	*+/−*	*BA*	*x*	*y*	*z*	*t*	*k*
Activation							
Risk							
R SFG[†]	+	9	15	47	25	3.63	42
MCC	+	31	0	−10	43	3.79	67
R motor cortex	+	4	24	−25	61	4.12	82
Calcarine gyrus	+	17	0	−99	7	4.45	50
Reward							
R VS	+		3	8	−2	4.64	50
R medial OFC[e]	+	11	9	53	−20	4.46	76
L medial OFC[e]	+	11	−9	41	−17	4.00	
R cerebelum	+		24	−70	−38	3.89	55
PPI (Medial OFC Seed)							
Risk							
Superior mPFC	+	9/10	−3	56	10	4.31	299
Reward							
Superior mPFC[f]	+	9/10	−6	59	10	5.85	891
rACC[f]	+	32	6	32	−2	5.40	
PCC	+	23/31	3	−49	28	4.02	112

L and R refer to left and right hemispheres; + and − refer to positive or negative association; BA refers to Brodmann's 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 mm^3. Superscripts (e.g., a, b, etc.) indicate that peak voxels are part of a contiguous cluster. rACC = rostral ACC.

[†]Cluster is subthreshold.

Table 4. Associations between Neural Regions Showing Age-related Increases in Activation and Behavioral Learning

Neural Regions	*Age*	*Risk Behavior*	*Learning*
Activation			
Risk			
MCC	.40****	.20*	.35****
R SFG[a] (k = 42)	.39****	.28**	.31****
Reward			
Medial OFC	.46****	.41****	.42****
VS	.46****	.18	.28**
PPI (Medial OFC Seed)			
Risk			
mPFC	.45****	.25**	.30***
Reward			
mPFC	.52****	.33****	.35****
PCC	.42****	.27**	.28**

*p < .1, **p < .05, ***p < .01, ****p < .001.

[a]Cluster is subthreshold.

engage in these trade-offs between risk taking and learning than are younger children, behavior that may result in adaptive outcomes.

At the neural level, we found that age-related increases in both motivational and regulatory neural systems supported flexible learning. Motivational regions included the VS and OFC. The VS, a region with a high density of dopaminergic neurons, has been classically implicated in reward anticipation and reactivity and shows heightened activation during adolescence (Galvan et al., 2005, 2006; see Telzer, 2016). The OFC's role in reward processing involves assigning and updating the relative reward value of actions and stimuli (Gottfried et al., 2003; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001). This study's findings of age-related increases in these two regions during reward acquisition fit well with previous research. Furthermore, we found that OFC reward-related activity explained links between age and increases in learning, which supports previous research implicating the OFC in reward-related learning (Schoenbaum & Roesch, 2005). Reward-related activity in the OFC may help adolescents track the motivational salience of points in the task as well as integrate reward (i.e., cash-outs) and punishment (i.e., explosion) feedback from the

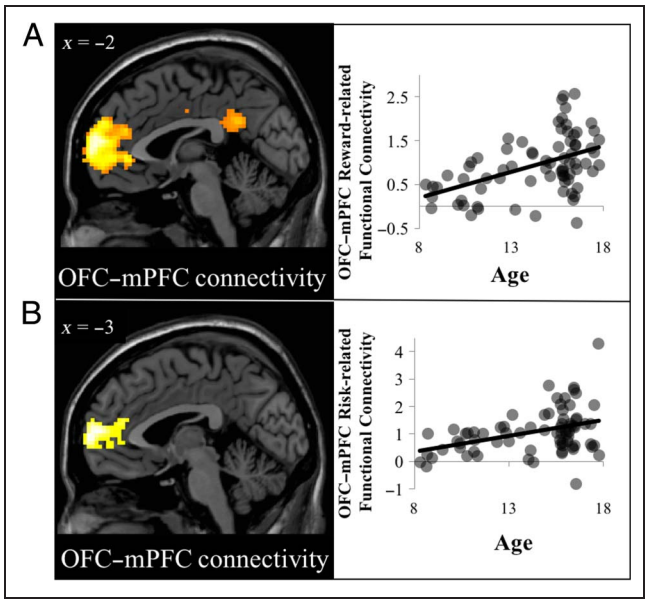

Figure 4. We found age-related increases in (A) both mPFC and PCC functional connectivity with OFC during reward and (B) mPFC functional connectivity with OFC during risk.

task into their cost–benefit representations for future risk taking.

We also found age-related increases in regulatory regions during risk decisions, including the MCC, which has been implicated in action selection (Shackman et al., 2011; Vogt, 2005). Developmental increases in reward-related activation in the OFC may reflect greater valuation of reward which drives changes in future behavior, whereas increases in regulatory and action selection regions may support increases in goal-directed behavior enactment. Changes in these neural systems support both learning and risk taking by increasing attention to certain stimuli, weighting information gained in rewarding contexts more so than children. This weighted information is in turn used to a greater degree to direct behavior during this period of development. However, an overweighting of reward-related information likely also is responsible for adolescents sometimes pursuing rewarding contexts without complete regard for the potential negative consequences. These results highlight the importance of two types of neural systems in supporting flexible learning and reflect a growing understanding that complex behaviors are not supported by the development of single brain regions, but rather a system of regions that play particular computational roles in the service of behavior.

Finally, to examine connectivity of circuits that may be important for learning, we examined how age-related changes in functional connectivity between motivational and regulatory regions support flexible behavior. We found age-related increases in functional connectivity between the medial OFC and the mPFC during both risk and reward conditions. Medial regions of the OFC show both structural (Öngür & Price, 2000) and positive functional (Kahnt et al., 2012) connectivity to regions of the mPFC, which has been implicated in associative learning and response adaptation (Euston, Gruber, & McNaughton, 2012) and are sensitive to risk conditions (Van Leijenhorst et al., 2010). Both regions have been implicated in risk-taking behavior (Van Duijvenvoorde et al., 2014; Chein, Albert, O'Brien, Uckert, & Steinberg, 2011) and show positive functional connectivity during risky decision-making in adults (Cohen, Heller, & Ranganath, 2005). Although the development of these circuits across adolescence has not been reported, resting state functional connectivity between OFC and mPFC regions has been shown to differ between individuals with drug addiction and controls (Janes, Nickerson, & Kaufman, 2012). These findings suggest that the development of OFC–mPFC circuitry plays an important role in risk-taking behavior.

Age-related increases in OFC–mPFC connectivity provide a mechanism for age-related increases in learning, suggesting that increased OFC–mPFC functional connectivity reflects a more integrated motivational regulatory system, with greater intercommunication between regions involved in reward processing and regions involved in action updating and selection. This supports previous findings that still-developing top–down regulation of the mPFC is associated with adaptive outcomes (Gee et al., 2013) and that, similar to other forms of physiological development (e.g., pubertal and reproductive timing), acceleration of neural development likely will involve trade-offs, which curtail extended learning and plasticity (Ellis et al., 2009). This study further suggests that the development of learning depends not only on localized activational increases but also on how neural regions interact, which underscores the importance of circuit-based understandings of neurodevelopmental processes (Casey, 2015). Mapping the functional significance of system level neurodevelopmental changes for adolescent behavior is an important future step for the examination of the neurobiological mechanisms driving the increases in risk taking and learning that characterize adolescence. When studying complex processes, such as risky decision-making, both localized and circuit-based changes should be considered as possible supporting mechanisms for behavior changes seen across development (Casey, Galván, & Somerville, 2016). Although research localizing function to particular brain regions has greatly contributed to our understanding of neural function, the brain operates as an integrated circuit, and studying developmental changes in individual regions may have a finite utility.

Although the results reported in the current study suggest an exciting new perspective on adolescent risk- and reward-related neural development, several compelling questions remain to be explored. We examined developmental trends in risk- and reward-relatedneural processes and functional connectivity in a large sample of 8- to 17-year-olds. Because of the constraints of participant behavior, we were unable to examine developmental trends in neural sensitivity to explosion events, and future research should aim to close this gap in our understanding of developmental trends in neural sensitivity to loss. Another constraint for interpreting the reported results arises from the complexity of the BART and the variety of strategies participants could conceivably employ during the task (for a review of some possibilities, see Wallsten, Pleskac, & Lejuez, 2005). As such, it is difficult to describe any one behavioral pattern as optimal in its own right, and therefore, we relied on strong associations with the total points participants earned to characterize higher learning as more optimal behavior. Additionally, previous research has suggested that developmentally significant changes in these neural systems likely continue further through the end of adolescence and into young adulthood (Braams, van Duijvenvoorde, Peper, & Crone, 2015; Van Leijenhorst et al., 2010). Future research should seek to extend the results reported in the current study by including participants through the transition from adolescence to young adulthood. Such a study could inform whether the developmental effects we report here continue to increase linearly into young adulthood or whether learning and exploration plateau or even

diminish. Finally, the cross-sectional nature of the current study limits our ability to draw any conclusions about individual differences in the neural and behavioral developmental trajectories we examined. Future research should examine how the processes of exploration, risk taking, and learning change within individuals over time. Longitudinal examination of these behavioral and neurodevelopmental processes can help to confirm and extend our understanding of how individual differences in these trajectories contribute to differences in adaptive and maladaptive outcomes across adolescence.

In summary, our findings support a new perspective of the behavioral and neurobiological changes that characterize adolescence. Development of motivational and regulatory neural circuitry supports adolescents' learning, which contributes to increases in risk taking. However, in contrast with much of the literature on adolescent development concerning risk behavior, we found that risky decisions emerge in part through adolescents' increased propensity for flexible learning, which suggests an adaptive role for still-developing neural circuitry. These results complement findings in nonhuman models, which suggest that adolescent animals (Vigilant et al., 2015; Johnson & Wilbrecht, 2011) show unique behavioral patterns that support flexibility in service of adaptive goals. This adaptive role for developing neural circuitry also supports previous suggestions that accelerated development may actually be detrimental and linked to negative outcomes (Ellis et al., 2009). Instead of a one-to-one correspondence between maturity and function, normative development may rely on neural and behavioral states that happen in a particular, developmentally appropriate fashion. Our findings underscore the importance of paying greater attention to the potentially adaptive roles that still-developing neural circuitry can have for adolescent behavior and the contexts in which these propensities for seeking learning opportunities may be appropriately channeled.

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Reprint requests should be sent to Eva H. Telzer, 235 E. Cameron Avenue, Chaperl Hill, NC 27514, or via email: ehtelzer@unc.edu.

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