NeuroImage 124 (2016) 989–996

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Adolescent neurodevelopment of cognitive control and risk-taking in negative family contexts☆

Ethan M. McCormick [a], Yang Qu [a], Eva H. Telzer [a,b,*]

[a] Department of Psychology, University of Illinois, Urbana-Champaign, United States
[b] Beckman Institute for Advanced Science and Technology, University of Illinois, Urbana-Champaign, United States

ARTICLE INFO

Article history:
Received 8 May 2015
Accepted 28 September 2015
Available online 3 October 2015

Keywords:
Family relationships
Risk taking
Adolescence
Cognitive control
fMRI

ABSTRACT

Adolescents have an increased need to regulate their behavior as they gain access to opportunities for risky behavior; however, cognitive control systems necessary for this regulation remain relatively immature. Parents can impact their adolescent child's abilities to regulate their behavior and engagement in risk taking. Since adolescents undergo significant neural change, negative parent–child relationship quality may impede or alter development in prefrontal regions subserving cognitive control. To test this hypothesis, 20 adolescents completed a Go/NoGo task during two fMRI scans occurring 1 year apart. Adolescents reporting greater family conflict and lower family cohesion showed longitudinal increases in risk-taking behavior, which was mediated by longitudinal increases in left VLPFC activation during cognitive control. These results underscore the importance of parent–child relationships during early adolescence, and the neural processes by which cognitive control may be derailed and may lead to increased risk taking.

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Introduction

One of the most important skills adolescents need to successfully develop is cognitive control. While certainly important during childhood, the ability to regulate one's impulses and behavior becomes increasingly crucial as children transition into adolescence, a time when risk-taking behavior increases substantially. Adolescence involves both the biological transition of puberty, characterized by dramatic physical (Wheeler, 1991) and hormonal (Susman et al., 1985) changes, with a shift in social contexts and roles (Nelson et al., 2005). Adolescents rapidly gain access to a number of potentially dangerous activities such as drugs and alcohol use (Kandel and Logan, 1984), driving (U.S. Census Bureau, 2012), and sexual debut (Cavazos-Rehg et al., 2009). Unfortunately, cognitive control abilities, and the prefrontal cortex which subserves them, remain relatively immature into and through adolescence (Luna et al., 2010). As a result, adolescents have difficulties regulating their impulsive behavior, placing them at increased risk for health compromising outcomes such as sexually-transmitted infection (Kaestle et al., 2005), substance abuse (Santor et al., 2000), school failure (Nelson and DeBacker, 2008), and accidents or death (U.S. Census Bureau, 2012). As such, adolescents' cognitive control abilities can have far-reaching implications for health and successful adjustment.

The quality of family relationships may facilitate adolescents' cognitive control abilities. This may occur through parents' modeling of self-regulation (Eisenberg et al., 2005; Morris et al., 2007), protecting adolescents from stress (Power, 2004), and providing support for adolescents' autonomous regulation (Eccles et al., 1997; Morris et al., 2007). Parent–child relationships characterized by conflict and stress reduce opportunities for adolescents to develop effective cognitive skills, which can increase the likelihood of subsequent risk-taking behaviors (McNeely et al., 2002; Telzer et al., 2014a). Indeed, the quality of parent–adolescent relationships influences sexual debut and riskiness (McNeely et al., 2002; Miller, 2002; Clawson and Reese-Weber, 2003), risky driving practices (Michael and Ben-Zur, 2007), and substance use (Borawski et al., 2003; Telzer et al., 2014a) such that hostility and conflict in family relationships puts teens at increased risk for these negative outcomes. Due to the costly consequences of adolescent risk taking (U.S. Census Bureau, 2012), understanding how parents contribute both positively and negatively to adolescent engagement in risky behaviors has important social and health implications.

Parents may influence their adolescents' engagement in risk taking, in part, through the influence of parenting on neural development. Similar to the early postnatal period, adolescence involves an increase in neural plasticity and reorganization (Casey et al., 2005), such that neural systems are particularly sensitive to social influences (Blakemore and Mills, 2014; Knoll et al., 2015). This increase in social salience may make adolescents more susceptible to the impacts of poor parent–

☆ We greatly appreciate the assistance of the Biomedical Imaging Center at the University of Illinois. This research was supported by a grant from the National Science Foundation (SES 1459719; Telzer), the National Institutes of Health (R01DA039923), and generous funds from the Department of Psychology at the University of Illinois.

* Corresponding author at: 603 East Daniel St., Champaign, IL 61820, United States.
E-mail address: ehtelzer@illinois.edu (E.H. Telzer).

http://dx.doi.org/10.1016/j.neuroimage.2015.09.063
1053-8119/Published by Elsevier Inc.

child relationships. Although parent–child relationships during early postnatal development set the stage for future neural trajectories (Schore, 2001; Gee et al., 2013; Tottenham, 2014), little attention has been paid to the effects of parent–child relationships during adolescence. There is evidence to suggest that adolescents' neural activity in some domains (e.g., affective processing) is modulated by parent–child attachment quality (Gee et al., 2013, 2014; Olsavsky et al., 2013) and structural differences emerge across adolescence in affective and prefrontal regions as a function of earlier positive parent–child interactions (Whittle et al., 2014). However, relatively little is known about the effects of parents on adolescent neural networks involved specifically in cognitive control, which is a significant limitation given that parents play a significant role in the development of youths' basic executive functioning (Deater-Deckard, 2014). Moreover, most neuroimaging studies have explored the effects of family relationships on neural processing using cross-sectional (i.e., single-time point) approaches (but see Qu, Fuligni, Gálvan, & Telzer, 2015). Because adolescence is a time of significant neural changes (Paus, 2005; Lenroot et al., 2007), these snapshots of neural activity may miss how parenting can have effects on adolescents' neural trajectories over time. In particular, by comparing individuals to their own baseline measurements, longitudinal approaches can offer insights into contextual factors which influence ongoing development. Compared with traditional cross-sectional approaches, longitudinal methodologies allow us to examine how specific factors (e.g., family relationships) are associated with changes in developmental trajectories at the individual level and offer a more-precise estimate of how these factors mediate changes in ongoing developmental processes (Maxwell and Cole, 2007). Additionally, longitudinal approaches have increased power to detect changes of interest relative to cross-sectional approaches by examining within-person change and has the benefit of controlling for differences that exist between cohorts compared in cross-sectional analyses (Louis et al., 1986).

To address these gaps in our understanding, we examined the impact of family relationship quality on longitudinal changes in risk taking across early adolescence, as well as the neural processes that may underlie this link. We examined both neural and behavioral changes as adolescents transitioned from the 8th to the 9th grades, a developmental transition marked by increases in risk-taking behaviors such as substance use (Bryant et al., 2003) and sexual initiation (Santelli et al., 2004). Additionally, adolescent–parent relationships during this period are often characterized by increased conflict as adolescents attempt to negotiate increased independence (McGue et al., 2005). Finally, on the neural level, large-scale developmental changes continue through this period, suggesting that neural systems remain plastic and especially sensitive to social and environmental input (Crone and Dahl, 2012; Blakemore and Mills, 2014). Importantly, neural regions in the lateral prefrontal cortex (PFC), which subserve cognitive control, are among the last to reach maturity, continuing to develop throughout the teenage years (Lenroot et al., 2007; Shaw et al., 2008). This prolonged maturation provides an extended window for environmental factors to exert influence on the development of the lateral PFC and associated cognitive control abilities (Nelson & Guyer, 2011) with downstream consequences for adolescents' engagement in risk-taking behavior (Casey et al., 2008). In the current study, we focused on the ventrolateral prefrontal cortex (VLPFC), a region involved in behavioral inhibition and cognitive control (Swick et al., 2008; Souza et al., 2009; Levy and Wagner, 2011). Moreover, recent studies have shown that longitudinal increases in VLPFC activation during a risk-taking task predict longitudinal increases in risk taking behaviors (Qu, Galvan, Fuligni, Lieberman, & Telzer, 2015), and changes in positive family interactions are associated with longitudinal decreases in lateral PFC activation among older adolescents (Qu, Fuligni, Gálvan, & Telzer, 2015). Following this prior work, and building off the extant literature linking family relationships with cognitive control abilities, we hypothesized that negative family relationships would be associated with longitudinal increases in VLPFC activation during a cognitive control task, and these neural changes would explain the link between negative family relationships and increased risk taking. We utilized a longitudinal design, which allowed us to examine how the quality of family relationships predicts individual trajectories in risk taking via changes in neural processing.

Methods

Participants

Twenty (13 male) healthy adolescents participated in the current study. Participants were studied at two time-points, once during 8th grade and again during 9th grade. All adolescents were 14 at Time 1 (T1: M_{age} = 14.39 years, SD = .34) and 15 at Time 2 (T2: M_{age} = 15.20, SD = .31). Three additional adolescents participated, but are not included in the current study (one participant moved excessively (>2.0 mm) and two did not complete self-report measures at T1). Participants provided written consent and assent in accordance with the policies of the University of Illinois' Institutional Review Board.

Self report measures

Family relationship quality

At T1 and T2, participants completed two self-report measures related to family relationship quality. The first asked participants to report on family conflict (Ruiz et al., 1998). Participants completed 10 questions about their relationship with their parents in the last month (e.g., "You and your parents had a serious argument or fight" and "You and your parents yelled or raised your voices at each other"). Participants used a 5-point scale to rate the frequency with which they and their parents engaged in these behaviors (1 = "Almost never" to 5 = "Almost always"). The measure had good reliability (α: T1 = .94, T2 = .93). Participants also reported on their family cohesion (FACES II; Olson et al., 1979). Participants completed 10 questions (e.g., "My mother/father and I feel very close to each other" and "My mother/father and I avoid each other at home") on the same 5-point scale. Questions for the family cohesion score were reverse coded such that a higher score reflected less cohesive family relationships. This measure had good reliability (α: T1 = .91, T2 = .88). The two measures were positively correlated (T1: r = .55, p = .01; T2: r = .45, p = .04), and were combined into a composite family relationship score, with higher scores representing greater levels of family conflict and lower levels of family cohesion.

Adolescent risk taking

In order to examine changes in risk taking, adolescents completed a modified version of the Adolescent Risk-Taking Scale at both T1 and T2 (Alexander et al., 1990; Telzer et al., 2013). Participants responded to 12 questions indicating how often (1 = "Never" to 4 = "Many times") they engaged in a range of risky behaviors (e.g., "I have stolen or shoplifted" or "I have had sex without using protection"). The scale had good reliability at both time points (α: T1 = .76; T2 = .89).

Cognitive control task

At both time points, adolescents performed a Go–NoGo (GNG) task during an fMRI scan. Participants were presented with brief (500 ms) trials which consisted of a single letter and were instructed to respond with a button press as quickly as possible to all letters (Go trials) except for Xs (NoGo trials). X trials occurred 25% of the total number of trials. This high ratio of Go trials reliably causes participants to develop a pre-potent response to perform a button press that must be inhibited during NoGo trials. Trials were separated by a fixation period that varied in length with a gamma distribution (M = 1000 ms). Participants completed four blocks of the task. Each block was composed of 80 trials (60 Go; 20 NoGo), and blocks were separated by a 60 s rest period. Efficacy of cognitive control was measured as successful inhibition of the button press during NoGo trials.

fMRI data acquisition

Imaging data were collected with a 3 T Tesla Siemens Trio MRI scanner. The GNG task involved the acquisition of T1*-weighted magnetization-prepared rapid acquisition gradient echo (MPRAGE) scan which was acquired for registration purposes [TR: 2.3; TE: 2.1; FOV: 256; matrix: 192 × 192; sagittal plane; slice thickness: 1 mm; 160 slices]. Structural scans were acquired with a T2 weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4 s; TE = 63 ms; FOV = 230; matrix = 256 × 256; sagittal plane; slice thickness = 1 mm; 192 slices). MBW and MPRAGE scans were acquired at an oblique axial orientation in order to maximize brain coverage.

fMRI data preprocessing and analysis

Preprocessing and data analysis were conducted using Statistical Parametric Mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK) software package. Scans for T1 and T2 were preprocessed separately. Preprocessing involved spatial realignment to correct for head motion (included participants had no motion in excess of 1 mm between-slice motion), as well as coregistration of all images with the high-resolution T1* MPRAGE structural scan, which was then segmented into gray matter, white matter, and cerebrospinal fluid. Transformation matrices used to normalize the MPRAGE images were applied to the MBW and functional images to transform them into the standard stereotactic space defined by the Montreal Neurological Institute (MNI) and the International Consortium for Brain Mapping. An 8 mm Gaussian kernel, full-width-at-half maximum was used to smooth the normalized functional images to increase the signal-to-noise-ratio. Using the general linear model in SPM8 to perform statistical analyses, each trail was convolved with a canonical hemodynamic response function. High-pass temporal filtering (cutoff = 128 s) was used to remove low-frequency drift across the time series. A restricted maximum likelihood algorithm with an autoregressive model order of 1 was used to estimate serial autocorrelations.

The task was modeled as an event-related design, with a trial duration of 500 ms. For the fixed-effects model, a general linear model (GLM) was created for each regressor of interest to parse the different events, including Go trials, successful NoGo trials, false alarms (i.e., button press on NoGo trials), and misses (i.e., inhibition of button press on Go trial). T1 and T2 regressors were modeled separately. The jittered inter-trial fixation periods and the 1-minute rest period between blocks were not explicitly modeled and therefore served as the implicit baseline. A parametric modulator was included which weighted the trials by block, with trials in the first block being weighted with a 0 and trials in the last block with a 3. Contrasts between T1 and T2 were computed at the individual level in order to examine longitudinal changes in neural reactivity.

Random effects and 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 (http://mrtools.mgh.harvard.edu/index.php/GLM_Flex). All group-level analyses in this study focused on successful inhibition trials (NoGo) since our primary goal was to examine neural activation supporting changes in effective cognitive control. To examine how changes in neural activation covaried with self-reported behavior, we entered changes in risk taking (i.e., difference score between risk taking at T2–T1) and T1 family relationship scores as regressors in whole brain regression analyses.

Correction for multiple comparisons was run using a Monte Carlo simulation through 3dClustSim from the AFNI software package (Ward, 2000) using the group-level brain mask. The simulation resulted in a voxel-wise threshold of $p < .005$ and a minimum cluster size of 42 voxels for the whole brain, corresponding to $p < .05$, False Wise Error (FWE) corrected.

Results

Behavioral results

Association between negative family relationship quality and change in risk taking

Our first analyses examined descriptive statistics for all variables as well as correlations across the variables across the two time points. As shown in Table 1, self-reported risk taking did not change from T1 to T2, whereas family relationship quality tended to get worse across time. We ran bivariate correlations to examine how negative family relationship quality was associated with adolescent risk-taking. In order to examine change in risk taking and family relationships, we computed a difference score, which represents risk taking (or family relationships) at T2 minus risk taking (or family relationships) at T1. More negative family relationship quality at T1 was associated higher risk taking at T1 and T2 as well as longitudinal increases in risk taking from T1 to T2. Negative family relationship quality at T2 and changes in family relationship quality (T2–T1) were not significantly correlated with risk-taking behavior (Fig. 1).

Behavioral performance on the Go–NoGo task

To measure performance on the Go–NoGo task, we used participants' false alarm rate: or the proportion of NoGo trials where participants failed to inhibit their button press. T1 and T2 false alarm rates did not significantly differ and were not related to either T1 negative family relationship quality or longitudinal changes in risk taking (Table 1).

Table 1
Descriptive statistics.

Variable of interest	M	SD	Range	t(19)	1	2	3	4	5	6	7	8	9
1. T1 Family relationship	2.30	.51	1.10–4.20		1	.77**	−.39	.59**	.71**	.51*	.30	.22	−.04
2. T2 family relationship	2.50	.69	1.40–4.25			1	.29	.36	.39	.28	.06	.07	.02
3. T2–T1 family relationship	0.20	.48	−.75–1.25	1.9+			1	−.37	−.49+	−.36	−.36	−.23	.09
4. T1 risk taking	1.30	.32	1.00–2.30					1	.80**	.34	.20	.10	−.08
5. T2 risk taking	1.40	.50	1.00–2.80						1	.77**	.28	.12	−.15
6. T2–T1 risk taking	0.10	.18	−.25–.48	1.4						1	.19	.06	−.14
7. T1 false alarm rate	0.09	.04	.01–.14								1	.63**	−.22
8. T2 false alarm rate	0.08	.04	.003–.17									1	.62**
9. T2–T1 false alarm rate	0.00	.04	−.07–.06	0.4									1

Note: Paired-samples t-tests were used to determine differences between T1 and T2 for variables of interest.
+ <.1.
* <.05.
** <.01.

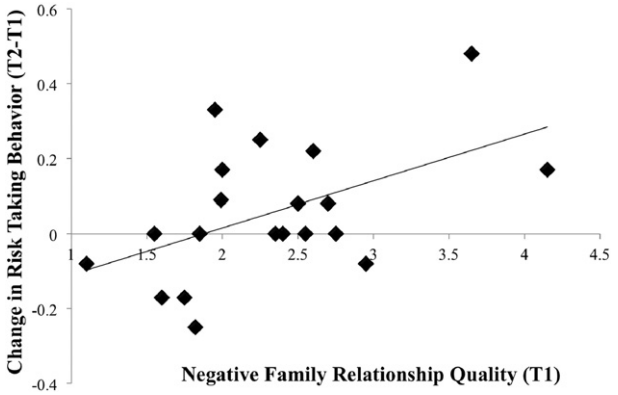

Fig. 1. Negative family relationship at T1 are associated with longitudinal increases in risk taking.

Table 2
Neural regions that correlated with negative family relationship at T1 during successful NoGo and Go trials at T2–T1 (all correlations are positive).

Region	BA	x	y	z	t	k
NoGo T2 > NoGo T1						
L inferior frontal gyrus	10	−33	53	−5	4.07	101
L middle frontal gyrus	46	−24	47	10	4.39	59
R postcentral gyrus[a]	6	54	−10	25	4.40	207
R middle temporal gyrus[a]	21	51	−43	4	3.90	
L lingual gyrus	19	−24	−85	−5	5.39	107
L cerebellum[b]		−12	−58	−38	4.02	80
R cerebellum[b]		9	−61	−38	3.51	
Go T2 > Go T1						
L orbitofrontal gyrus	11	−36	47	−14	4.80	78
R orbitofrontal gyrus	11	48	47	4	4.26	73
Posterior cingulate[c]	23	3	−31	31	6.40	562
R precentral gyrus[c]	4	24	−31	67	4.47	
L postcentral gyrus	1	−48	−22	37	5.70	73
L superior temporal gyrus	22	−54	−34	22	5.63	98
L cerebellum		−30	−34	−32	6.04	233

Note: L and R refer to left and right hemispheres; 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; and x, y, and z refer to MNI coordinates.

fMRI results

Neural changes associated with negative family relationship quality

Our first fMRI analyses examined how family relationship quality at T1 was associated with changes in neural activation during cognitive control. In whole-brain regression analyses, we regressed T1 family relationship quality onto changes in brain activation during successful NoGo trials (NoGo T2 > NoGo T1). More negative family relationship quality at T1 was related to longitudinal increases in activation in the left VLPFC over time (Fig. 2, Table 2). Negative family relationship quality at T1 was not associated with longitudinal decreases in neural activation from T1 to T2. We conducted similar whole-brain regression analyses using negative family relationship quality at T2 as well as change in family relationship quality (T2–T1). Neither of these regressions yielded significant clusters of activation.

We similarly regressed T1 family relationship quality onto changes in brain activation during successful Go trials (Go T2 > Go T1). Negative family relationship quality at T1 was positively associated with longitudinal increases in activation from T1 to T2 in the bilateral orbitofrontal gyri, posterior cingulate, and postcentral gyrus (Table 2). To test for overlapping regions of activation during the Go T2 > Go T1 and NoGo T2 > NoGo T1 regressions, we created a mask of active regions in the Go T2 > Go T1 regression and applied it to the regression of T1 negative family relationships on NoGo T2 > NoGo T1. When the mask is applied, there are no significant regions of activation in the NoGo T2 > NoGo T1 regressions, suggesting that effects in the Go and NoGo conditions are distinct.

Neural changes associated with increases in risk taking

Next, to examine whether changes in neural activation were related to changes in self-reported risk-taking behaviors, we ran whole-brain regression analyses in which we regressed changes in risk taking (T2–T1) onto changes in brain activation during successful NoGo trials (NoGo T2 > NoGo T1). Increases in risk taking from T1 to T2 were associated with longitudinal increases in activation in the bilateral VLPFC (Table 3). Notably, the left VLPFC cluster is nearly identical to the cluster found for negative family relationship quality. Using the MarsBar toolbox extension in SPM (Brett et al., 2002), cluster overlap was determined by creating masks of the VLPFC clusters related to negative family relationships at T1 and changes in risk-taking separately, and then combining them into a new mask which only contained regions of overlap present in both original clusters. We found that the clusters indeed share a common region of activation in the left VLPFC that includes 101 voxels (Fig. 3). The two regressions shared no other significant clusters of activation. Finally, we regressed change in risk-taking

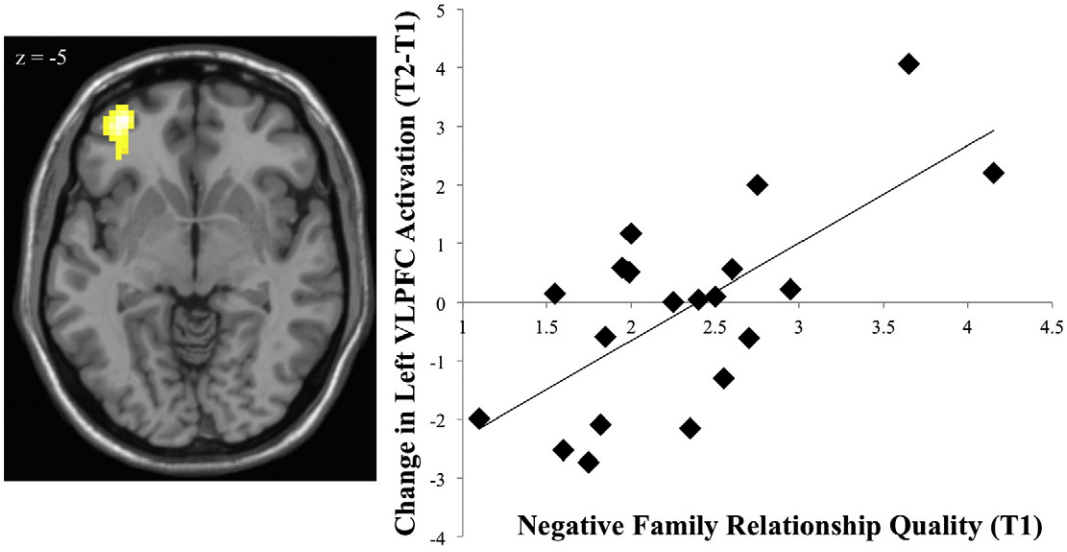

Fig. 2. Negative family relationship at T1 are associated with longitudinal increases in activation in the left VLPFC.

Table 3
Neural regions that correlated with changes in risk taking (T2–T1) during successful NoGo trials at T2–T1 (all correlations are positive).

Region	BA	x	y	z	t	k
NoGo T2 > NoGo T1						
L inferior frontal gyrus	10	−39	53	−2	6.59	286
L insula		−36	14	−2	4.06	42
R insula		36	14	−2	5.16	226
R superior medial gyrus	8	3	29	52	4.15	80
Cerebellum		0	−52	−41	6.52	227
Go T2 > Go T1						
R ACC[a]	32	9	35	7	6.12	121
R superior frontal gyrus	46	21	56	7	4.50	177

Note: L and R refer to left and right hemispheres; 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; and x, y, and z refer to MNI coordinates.

onto the contrast comparing change in Go trials (Go T2 > Go T1). This analysis revealed significant clusters of activation in the right DLPFC, and rostral ACC (Table 3), suggesting that VLPFC activation is an effect unique to trials involving successful inhibition.

Neural changes mediate the relationship between negative family relationships and increases in risk taking

Next, we examined whether changes in neural activation in the VLPFC mediate the association between negative family relationship quality at T1 and changes in self-reported risk-taking. We extracted parameter estimates of signal intensity from the cluster of activation which showed an overlap in the two analyses described above and ran mediation analyses using the methods outlined by Hayes (2013). We calculated the magnitude and the significance of the indirect effect, in which bootstrapping was performed with 1000 samples and a bias-corrected confidence interval (CI) was created for the indirect effect. We standardized family relationship quality and risk-taking, and controlled for T1 risk taking in the mediation analyses in order to account for associations between starting levels of risk-taking and longitudinal changes in risk taking. We found a significant mediation, such that the link between greater negative family relationship quality and longitudinal increases in risk-taking behavior is mediated by increases in left VLPFC activation (indirect effect: $B = .51$, $SE = .25$; 95% CI [.07, 1.03]). Moreover, with the inclusion of neural changes in the model, the direct path from negative family relationships to increases in risk taking is no longer significant (Fig. 4).

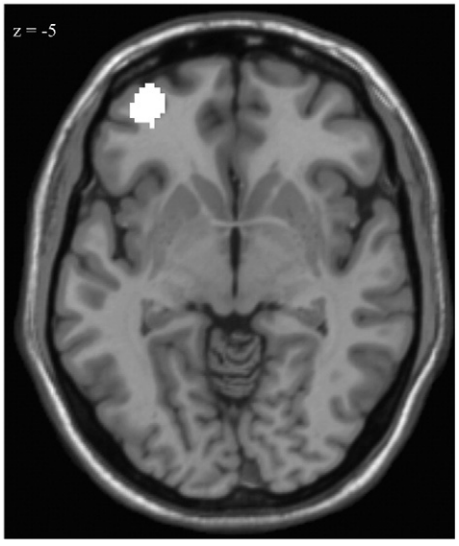

Fig. 3. Overlapping voxels for regressions with negative family relationship quality (T1) and changes in risk taking (T2–T1) onto changes in neural activation during cognitive control.

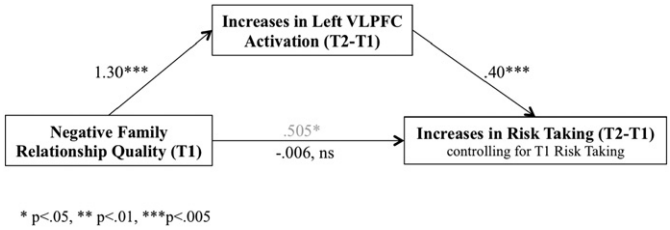

Fig. 4. The relationship between negative family relationship quality at T1 and longitudinal increases in risk taking is mediated by increases in left VLPFC activation.

Finally, we tested whether longitudinal changes in VLPFC activation serves as a unique mediator above and beyond the effects of either T1 or T2 VLPFC activation. Moreover, we wanted to test whether either starting point (T1) or ending point (T2) VLPFC activation contributed to the indirect effect of negative family relationships to increased risk taking. To this end, we extracted parameter estimates of signal intensity from the left VLPFC cluster at T1 and T2 separately. We ran the same mediation analysis described above with the inclusion of these as additional mediators. Results indicate that changes in left VLPFC activation remain a significant mediator of the relationship between T1 negative family relationship quality and increases in risk taking (indirect effect: $B = .51$, $SE = .28$; 95% CI [.03, 1.13]). Left VLPFC activation at T1 (indirect effect: $B = -.002$, $SE = .08$; 95% CI [−.17, .10]) and T2 (indirect effect: $B = .01$, $SE = .05$; 95% CI [−.05, .17]) were not significant mediators of the relationship between negative family relationship quality at T1 and longitudinal changes in risk taking, suggesting that family relationships relate to increased risk taking via longitudinal changes in VLPFC activation during cognitive control, and that the starting point (i.e., T1 activation) or ending point (i.e., T2 activation) is not driving this effect.

Longitudinal changes in activation during NoGo trials

As a final analysis, we examined longitudinal changes in neural activation during NoGo trials (NoGo T2 > NoGo T1) to assess normative changes in neural response during cognitive control across the year. A whole brain t-test showed no significant differences in neural activation from T1 to T2; however, using the VLPFC cluster defined previously, we extracted cluster parameters from T1 and T2 VLPFC separately and calculated the intraclass correlation (ICC) between T1 and T2 activation in the VLPFC. The ICC = .24, which indicates that individuals showed very low stability in neural activation between years (Hallgren, 2012), suggesting that while there is no significant average difference in neural activation across the year, there are significant individual differences in neural trajectories over this period.

Discussion

Family relationships during early adolescence help set the foundation for later neural and behavioral development (McNeely et al., 2002; Miller, 2002; Telzer et al., 2014a). Building on this previous research, we found that adolescents reporting more negative family relationships, characterized by high conflict and low cohesion, were more likely to show longitudinal increases in risk taking across early adolescence. Negative family relationships at T1 were associated with longitudinal changes in VLPFC activation, and these neural changes served a mediating role in the relationship between negative family contexts and longitudinal changes in risk-taking behavior. Furthermore, changes in VLPFC activation mediated this relationship even when controlling for T1 risk taking, as well as T1 and T2 VLPFC activation, suggesting that neurodevelopmental trajectories have a unique importance for risk taking behavior during adolescence.

Our results highlight the associations between negative family contexts in early adolescence and changes in VLPFC activation and risk

taking behavior. Early strife appears to be uniquely predictive of these developmental changes, as neither T2 nor changes in family relationships were associated with risk taking or neural changes. Additionally, risk taking at T1 was not predictive of T2 or T2–T1 family relationships. This lack of a reciprocal effect suggests a potential causal direction: that negative family relationship quality in early adolescence sets the stage for later changes in risk taking behavior. Interestingly, adolescents' performance on the Go–NoGo task was stable from T1 to T2. The lack of longitudinal behavioral differences suggests that the changes seen in VLPFC activation are driven by differences in *how* adolescents are completing the task and not by differences in teens' abilities to succeed in the task. Furthermore, our results highlight the importance of longitudinal approaches in examining adolescent brain–behavior relationships. In our mediation analyses, changes in VLPFC activation served as a mediator between negative family relationships and changes in risk taking, while T1 and T2 VLPFC activities were not significant mediators of this relationship. This suggests that negative family contexts are not only related to patterns of neural change, but that these patterns of change have importance in predicting behavioral changes beyond single-time point measurements. Cross-sectional approaches would be unable to detect this relationship, demonstrating the need for longitudinal studies examining neural change and their behavioral correlates over adolescence.

Our findings fit well with previous research that has examined the effects of parent–child relationship quality on adolescent risk taking (Miller, 2002; Borawski et al., 2003; Telzer et al., 2013; 2014a, Qu, Fuligni, Gálvan, & Telzer, 2015). Generally, positive relationships characterized by high levels of warmth, connectedness, and communication facilitate the delay of risk taking onset as well as decreases in the frequency of engagement (Boyer, 2006). However, previous research has mainly focused on external explanations for this link (but see Qu, Fuligni, Gálvan, & Telzer, 2015), such as how and when parents communicate with their children about risky behavior (Clawson and Reese-Weber, 2003; Huebner and Howell, 2003) or the level of control and monitoring parents maintain over adolescents (DiClemente et al., 2001; Borawski et al., 2003). Exploring neural mechanisms underlying behavioral relationships allows us to take a mechanistic and process-oriented approach by which we can explain a range of environmental effects by their differential impacts on common neural circuitry. This research strategy has the added benefit of synthesizing findings and methodologies from current social developmental and neurobiological research, allowing us to relate neurodevelopmental processes to changes in real-world behavior. Combining these advantages with within-subject variation through longitudinal neuroimaging allows us to make inferences based not only on a snapshot of neural activity, but also how changes in neural systems across adolescence can impact behavior. Longitudinal fMRI studies are especially critical during adolescences, as the major changes that occur in neural systems during this developmental period may make single-time point measurements particularly misleading without contextual information (Rogol et al., 2000).

We took an important approach by examining longitudinal changes in brain activation to test how family relationship quality may be associated with adolescent neural development as well as risk taking outcomes. We found that higher levels of negativity in adolescents' relationship with their parents was associated with longitudinal increases in activation in the VLPFC across early adolescence, and the increases in activation were associated with increases in risk taking. While the effects of parenting have been explored in terms of behavioral outcomes in adolescents (Miller, 2002; Borawski et al., 2003), this study provides novel evidence of a neural mechanism linking poor family relationship quality to greater adolescent risk-taking behaviors. The increased plasticity and flexibility which adolescent neural systems display are likely an adaptive feature of this developmental period, enabling learning and the ability to adapt more-readily to environmental contexts (Crone and Dahl, 2012). Indeed, we have previously found that positive parent–child relationships later in adolescence are associated with decreases in risk taking via changes in neural processing (Qu, Fuligni, Gálvan, & Telzer, 2015). The link between negative parent–child relationships and neural and behavioral outcomes in the current study suggests that the plasticity and flexibility associated with adolescence also confer unique vulnerabilities to negative social contexts, which put adolescents at risk for negative health and achievement outcomes. Understanding adolescents' vulnerability to negative social relationships can help explain why stress at particular developmental stages can have lasting influences on neurodevelopment.

The association between negative parent–child relationships during adolescence and the neural and behavioral outcomes reported here may be driven by a number of factors. Chronic stress related to negative parent–child relationships may impair adolescents' ability to deploy cognitive control resources effectively (Liston et al., 2009; Mueller et al., 2010). Additionally, many of the cognitive control skills adolescents acquire are learned from parental modeling (Morris et al., 2007). On the neural level, stress is known to impair normative development in regions subserving cognitive control through hormones released by the HPA-axis (Lupien et al., 2009; Casey et al., 2010), and the neural signature of regulation in parents predicts adolescents own abilities to regulate, suggesting that adolescents are picking up on cues from their parents' regulatory abilities and incorporating them in their own neuroregulatory strategies (Telzer et al., 2014b). Adolescents in families characterized by high levels of conflict and low levels of cohesion may have compounded difficulties in developing effective cognitive control as they have both more familial stress and fewer opportunities to learn positive regulation strategies from their parents, both of which negatively impact adaptive neurodevelopment of regulatory regions.

Conclusions drawn from the current study should be viewed in light of some limitations. First, the approach taken in the current study does not allow for the complete dissociation of development in negative family contexts from normative VLPFC activation changes during development. Since there is likely to be an increase in negativity between parents and adolescents as a natural part of this developmental transition, it would be informative to know whether parents had long-standing negative relationships with their children, or if this conflict is more recent. There may be differences between the effects of chronic or more-recent negativity on adolescents' neural development and risk taking. Future research should address this by following younger children as they enter adolescence. This approach will not only help disentangle the differential effects of long-standing conflict versus more-normative conflict that emerges as children enter adolescence, but will also allow researchers to examine how the emergence of negative family relationships influences on-going neural trajectories as compared with individuals who do not experience negative family contexts over the same period of development. Finally, while the pattern of results reported here is suggestive, definitive conclusions regarding the direction of effect between family relationship quality and the neural and behavioral outcomes remain unknown. Adolescents' risk-taking behaviors prior to 14 likely contribute to parent–child conflict at 14, which may indicate that risk-taking and family relationship quality reinforce one another over time. Moreover the influences of family relationships at age 14 on risk taking may potentially differ as adolescents move into more mature forms of risk taking, such as illicit substance use. Although overall risk taking behaviors in the current sample were relatively low, the fact that family relationships early in adolescence are predictive of even comparatively minor increases in risk-taking behavior points to the strong role of family context in the development of cognitive control and risk taking. Finally, different antecedent factors which contribute to negative family environments may have unique and dissociable effects on neuroregulatory development. It is also possible that negative family contexts and changes in risk taking are related to other underlying child and parent factors such as difficult or reactive temperament which engender both greater conflict and risk-taking. While the current study focused on individual differences in negative family contexts, without consideration for the causes of such strife,

additional information may be gleaned from considering different pathways that can lead to negative family contexts (e.g., difficult child temperament vs. parent substance use). Future studies should aim to address the causal direction of this relationship and attempt to disentangle unique effects of the different manners in which family strife can emerge.

In conclusion, we found that family relationships characterized by high levels of conflict and low levels of cohesion were related to increases in risk taking across early adolescence. Furthermore, we found that higher levels of negative family relationships predicted longitudinal neural changes in the VLPFC that mediated the impact of negative family relationships on trajectories of adolescent risk taking. These findings offer a first look at the importance of family relationships on neural development during adolescence and underscore how high levels of tension between parents and adolescents can have real-world implications for long-term adolescent health.

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