Neural correlates of social influence on risk taking and substance use in adolescents

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Key words: substance use; adolescent brain development; fMRI; peer influence; family influence; risk taking

Abstract

Purpose of Review. Adolescents often engage in elevated levels of risk taking that gives rise to substance use. Family and peers constitute the primary contextual risk factors for adolescent substance use. This report reviews how families and peers influence adolescent neurocognitive development to inform their risk taking and subsequent substance use.
Recent Findings. Developmental neuroscience using functional magnetic resonance imaging (fMRI) has identified regions of the brain involved in social cognition, cognitive control, and reward processing that are integrally linked to social influence on adolescent risk taking. These neural mechanisms play a role in how peer and family influence (e.g., physical presence, relationship quality, rejection) translates into adolescent substance use.
Summary. Peers and families can independently, and in tandem, contribute to adolescent substance use, for better or for worse. We propose that future work utilize fMRI to investigate the neural mechanisms involved in different aspects of peer and family influence, and how these contexts uniquely and interactively influence adolescent substance use initiation and escalation across development.

Introduction

Adolescence is a time of heightened vulnerability for risk-taking behavior that gives rise to substance use [1], one of today's most important societal concerns as it contributes to a host of serious immediate and long term outcomes. Indeed, morbidity and mortality rates increase 300% from childhood to adolescence [2] with over 70% of adolescent deaths each year due to risk-taking behaviors such as smoking, alcohol and drug use, car accidents, and unsafe sexual intercourse [3]. These consequences are particularly concerning during adolescence, a critical time for substance use initiation and experimentation. For example, lifetime illicit drug use more than doubles and current drug use more than triples from 8th to 12th grade [4].

Adolescents' decisions to use substances are impacted by a variety of social influences and are integrally linked to various social psychological processes. Family and peers constitute the primary contextual risk factors for adolescent substance use [5]. While peer influence and conformity to risky behaviors increase from childhood to early adolescence, before declining again in early adulthood [6-8], family factors remain a significant source of influence throughout childhood and adolescence [9], despite the salience of parental influences purportedly receding [10-11]. Thus, although peer influence becomes highly salient during adolescence, family relationships continue to shape adolescents' risk-taking behaviors without being supplanted by peers.

The family is the first and primary social group to which individuals belong, and many have argued that the family is the most important factor in the development of problem behaviors, such as substance use [12]. A wide range of familial variables impact adolescent substance use, including the quality of the relationship, parental monitoring, marital discord, parental depression, and parents' own attitudes about and use of drugs [9, 13]. According to social control theories, adolescents who are close to their parents feel obligated to act in non-deviant ways to please their parents, whereas adolescents in conflictual families do not feel obligated to conform to their parents' expectations and will be more likely to experiment with substance use [14]. Thus, the quality of family relationships impacts adolescents' substance use behaviors directly (i.e., actively choosing not to engage in the behavior), and indirectly by adolescents' avoidance of deviant peers thereby decreasing their exposure to substance use opportunities.

As children enter adolescence, they spend increasing amounts of time with their peers without adult supervision and tend to engage in more risky behaviors during unsupervised time in the presence of their peers [15]. Perhaps the strongest predictor of adolescent substance use

is affiliation with deviant peers [13]. When adolescents' friends use drugs, they are more likely to initiate substance use themselves, whereas they are unlikely to use substances if none of their friends do so [14]. According to Sutherland's differential association theory [16], adolescents will learn from and be socialized to use substances if they associate frequently with peers who have favorable attitudes towards drugs. Thus, not only do deviant peers provide greater opportunities to engage in substance use, but they convey valued social norms and attitudes regarding substance use, thereby increasing adolescents' motivation and rationalization to engage in substance use [12]. Of course, there are selection effects too, such that adolescents who use drugs tend to choose friends who also use drugs [14]. Together, significant research highlights adolescence as a period when both peers and parents play an important role in adolescents' initiation and escalation of substance-use behavior.

Adolescent Brain Development

Emerging evidence from developmental neuroscience suggests that risk-taking behavior increases during adolescence partly due to changes in the brain's neural circuitry. In the last decade, there has been an explosion of research examining the neurodevelopment of the human adolescent brain. These studies revolutionized our understanding of adolescent decision making and challenged traditional views that the rise in risky behavior during adolescence occurs because adolescents are irrational or deficient in their decision-making capacities [17]. Rather, adolescents demonstrate unique sensitivity to social and affective contexts, perhaps as a result of the remodeling of the brain during puberty [18-19]. Based on developmental cognitive neuroscience research, we now know that the teenage brain is rapidly changing and adapting to its environment. The dramatic changes in brain development that occur in adolescence can be both an opportunity for learning, skill acquisition, and social development [20], but also a vulnerability for heightened social influence and an orientation to social rewards that increase susceptibility to drug use.

Several prominent models of adolescent brain development have been proposed to describe adolescent risk-taking behaviors. The *Imbalance Model* [21-22], similar to the *Dual Systems Model* [17], suggests that a maturational imbalance between development of cognitive control and affective reward systems underlies adolescents' increased risk taking. The subcortical network, comprising neural regions associated with the valuation of rewards (e.g., ventral striatum (VS), orbitofrontal cortex (OFC), ventromedial PFC; Figure 1), matures relatively early, leading to increased reward seeking during adolescence, whereas the cortical network, comprising neural regions involved in higher order cognitive control (e.g., ventrolateral and medial prefrontal cortices (VLPFC, mPFC); Figure 1), gradually matures over adolescence and into adulthood. The temporal gap between early development of the subcortical socioemotional system and later development of the cognitive control system is thought to create a developmental window of vulnerability for risk taking in adolescence [17]. In particular, overreliance on subcortical, reward-related regions drives adolescents to seek out rewards in their environment at a developmental period when self-control is still maturing and unable to effectively down-regulate the heightened drive for rewards, resulting in a peak in risk-taking behavior observed in adolescence.

Supporting adolescents' heightened social sensitivity, a growing body of literature has examined neural systems involved in social cognition [23] with a particular focus on the social brain [24]. The *Social Brain* model highlights a set of neural networks that code for the ability to think about others' mental states such as their thoughts and feelings, to use such information to inform one's own behaviors, and to predict what another person will do next during a social interaction [24-26]. Neural regions in the social brain include the medial prefrontal cortex (mPFC), posterior superior temporal sulcus (pSTS), and temporal parietal junction (TPJ) (see Figure 1). Most prior theory and research examining adolescent risk taking has largely focused on affective and cognitive control neural networks (i.e., dual systems model); however,

incorporating the *Social Brain* model into our understanding of adolescent risk taking can provide deeper insight into the complex interactions among social, cognitive, and affective processing that occur during this developmental period. As demonstrated in Figure 1, we highlight the key regions involved in adolescent risk taking and social cognition.

Despite recent calls to pay greater attention to the social context when examining adolescent neurocognitive development [27-28], surprisingly little research has carefully examined how neurobiological development interacts with fundamental social processes during the adolescent period. This is a critical limitation given that adolescents' decision making does not occur in a social vacuum. Adolescents spend most of their day with others–classmates, friends, family, and teachers–and their decisions are usually made under conditions of socio-emotional arousal [29]. In the following sections, we review literature linking two of the most important social relationships–family and peers–to adolescent neurocognition and risk for substance use.

Neural correlates of peer influence on risk taking and substance use

A large body of epidemiological literature describes a sharp rise in risk-taking behaviors, such as smoking, substance use, and risky driving, when adolescents are with their peers [30-32]. In early adolescence, those with a history of problem behaviors often flock together in deviant peer groups, engaging in daily social interactions that further amplify problem behaviors [33-35]. Experimental studies support these real-world findings and show increased risky and impulsive decisions in the laboratory when adolescents are accompanied by their friends (i.e., implicit peer influence) or receive peer feedback (i.e., explicit peer pressure to take risks) [7, 29, 36-38].

Negative peer influence increases adolescent risk taking by modulating neural processes involved in reward sensitivity, cognitive control, and the social brain. One particularly influential functional magnetic resonance imaging (fMRI) study examined the neural correlates of peer presence (i.e., implicit peer influence) on simulated driving behavior in adolescents, young adults, and adults [36]. Adolescents engaged in significantly more risky driving in the presence of their friends, an effect that was not found in young adults or adults, and this was modulated via increased activity in the ventral striatum with friends present, suggesting that peer presence may make risks more rewarding during adolescence [36]. In another study testing the role of risk-encouraging peers (i.e., explicit peer pressure), adolescents were examined in a driving simulation using electroencephalography (EEG) [39]. Adolescents who reported greater sensation seeking were riskier in the presence of their peers, and this was modulated by reduced mPFC activity when adolescents played in the presence of peers as compared to alone [39]. Collectively, neuroimaging studies highlight that risky behaviors may be more rewarding [36], whereas cognitive control may be compromised [39], in the context of peers during adolescence.

Another way in which peers negatively impact risk-taking behaviors is through peer rejection, a highly prevalent stressor with 41% of adolescents reporting peer exclusion in the past two months [40]. Neuroimaging research has examined how peer exclusion modulates risk-taking behavior and the neural processing of risks. After an acute instance of peer exclusion, adolescents display greater activation in social brain regions (mPFC and TPJ) while making safe decisions, as well as greater activation in the TPJ when making risky decisions [41]. Extending this work to real-life risk taking, another study found that brain activity in social brain regions (dmPFC and TPJ) during an episode of acute peer exclusion predicted increased risk taking in the presence of a peer during a driving simulation one week later [42]. Together, these studies suggest that peer exclusion may increase mentalizing about the beliefs and norms of peers, a process involving the recruitment of social brain regions. Consequently, this process may lead to adolescents engaging in higher rates of risk-taking behavior in order to fit in with expected group norms for peer acceptance.

For some adolescents, peer rejection is not incidental but rather a chronic childhood experience. Building upon previous work, we found that adolescents who were chronically victimized as children showed significantly greater increases in risk taking following acute social exclusion [43]. This increase in risk taking was paralleled by enhanced activation in the same social brain regions (mPFC and TPJ) when making both risky and safe decisions during the driving simulation, compared to a non-victimized control group. Further, brain activation in the social brain mediated victimization group differences in risk taking. Perhaps chronically victimized adolescents heavily focus on peer approval and try to gauge peer opinions to preemptively adjust their behavior, during which these mentalizing regions are recruited [43]. Youth experiencing chronic peer victimization [43] and peer conflict [44] also show greater activation in the VS when making risky choices, suggesting that the expected value of risk taking is heightened in youth who have a history of peer stress. This work provides support that peer rejection leads to increases in risk taking via brain activity in affective regions as well as social cognition regions, reflecting anticipation of social reward (i.e., peer approval) and mentalizing about peer opinions.

Fortunately, recent research suggests that peers are not always associated with negative effects. Whereas peer conflict increases risk-taking behavior via enhanced activation in the VS, high levels of peer support can buffer these effects [44], underscoring the important, protective role of supportive friends in the context of negative peers. Another study showed the positive effects of peers by investigating risky driving in a simulator in the presence of either a risky or a cautious peer. Results showed that activation of the cognitive control network during a response inhibition task predicted safer driving in the presence of a cautious peer compared to a risky peer one week later [45]. These studies provide evidence that supportive peer relationships can buffer potential negative peer effects, processes that may also mitigate substance use in youth. Due to small sample sizes, and the nascence of developmental social neuroscience, little attention has been directed towards examining sex differences in brain response to peer influence. However, some evidence is emerging, suggesting different developmental trajectories between boys and girls in their neural response to peer evaluation [46] as well as winning money for peers [47].

Neural correlates of family influence on risk taking and substance use

Although adolescent development typically encompasses a social reorientation toward peers [17] and spending less time with parents [48], families still remain an important source of influence on adolescent decision making and substance use. For example, adolescents with strong familial values are less likely to use substances such as cigarettes, alcohol, and marijuana [49]. In addition to family values, parental involvement, sibling companionship, as well as overall low levels of family conflict and high family cohesion, have the potential to buffer adolescents from substance use [50-51]. Thus, positive family environments can protect adolescents against the initiation and maintenance of substance use.

The meaningfulness of families is manifested as a neurobiological endophenotype [52], suggesting that the family context may wire adolescents to respond to their social environments in specific ways. For example, adolescents with strong familial values show suppressed activation in the VS during risk taking as well as greater activation in the PFC during cognitive control [53]. Importantly, suppressed VS activation is associated with less risk taking and heightened PFC activation with better decision-making skills [53]. Thus, adolescents who place greater value on familial values may be less likely to take risks, such as substance use, because they find risks less rewarding and are better able to avoid the impulse to engage in such behaviors. Relationship quality between parents and adolescents also influences adolescent decisions to take risks, such that greater closeness with parents across adolescence predicts longitudinal declines in the VS during risk taking [50]. Importantly, declines in the VS mediate links between parent-adolescent relationship quality and later declines in risk taking,

suggesting that parent relationship quality contributes to less risk taking through deactivation in the VS in response to rewards. These studies provide support that families, particularly parents, can positively influence adolescents to not engage in substance use through the brain by maintaining positive relationships and transmitting family values.

Although parents tend to reduce their supervision of their children as youth enter adolescence, parental monitoring remains a key influence on adolescents' decisions to avoid risk taking. For example, in the presence of their mother, adolescents make less risky choices compared to when alone, and they recruit the VS less during risky decision making [54], a phenomenon specific to mothers and not other adults [55]. In addition, the presence of mothers is associated with greater activation in the lateral PFC and a social brain region, the mPFC, as well as greater coupling between the VS and PFC during safe decision-making [54]. These results indicate that parental influence has the potential to reorient neural circuitry toward more controlled and safe decision-making and highlight the potential of families to provide a protective and positive influence in preventing adolescent substance use.

Unfortunately, not every family exerts a positive influence on adolescent development, which can lead to risk taking and subsequent substance use [56]. For example, adolescents who experience a lack of parental structure and monitoring exhibit an increase in delinquency and substance use across adolescence [57]. The salience of negative family influences on adolescent substance use has led to recent investigations of whether the brain plays a role in these associations. Indeed, high family conflict predicts increases in risk-taking behaviors across the adolescent years [58]. In addition, longitudinal increases in PFC activation during a cognitive control task mediates this association, suggesting that hostile family relationships heighten risk-taking behavior via enhanced activation in brain regions associated with cognitive control during adolescence. Increases in PFC activation might reflect the need for greater PFC activation to perform tasks involving cognitive control among youth who experience high family conflict. Parental depression has also been linked to adolescent risk taking [59]. Adolescents with a parent who feels depressed exhibit longitudinal increases in risk taking, which is explained by increases in the VS and DLPFC during risk taking, suggesting that the lack of parental positive affect may predispose adolescents towards seeking external rewards in their environment. These findings suggest that negative family relationships can change adolescent neural circuitry in response to rewards, and subsequently adolescent susceptibility to risk taking and substance use.

We can also understand the negative link between the family context and adolescent substance use by measuring the cumulative effects of family stressors, rather than just one stressor [56]. Family life stress, including events such as family member death, difficult financial circumstances, and arguments between family members is associated with less mPFC activity during anticipation and receipt of rewards, which is also related to greater alcohol dependence during adolescence [60]. In addition, decreased mPFC activity mediates the link between stressful family life events and later alcohol use. This study suggests that an accumulation of family stressors can lead to less neural reactivity in cognitive control regions in rewarding environments, as well greater risk toward substance use. Collectively, these findings provide support that stressors within the family, such as conflict, parental depression, financial hardship, and death of a family member can contribute to increased substance use during adolescence, particularly through the responsivity of the reward and cognitive control networks of the brain. As with the peer research, little attention has been directed towards examining sex differences in brain response to family influence.

Simultaneous influence of peers and family on risk taking and substance use

Most prior research has examined how peer and family contexts independently influence adolescent substance use without taking into account their interrelated and simultaneous effects. Family and peer contexts do not occur in isolation; families influence who adolescents

choose to spend time with (i.e., selection of friends), friends can influence the familial attitudes that adolescents value, and antisocial peer conformity can provoke arguments at home [5, 8-9]. Thus, social influence on adolescent substance use does not occur along a linear, independent path, but instead family and peer influence interact dynamically as part of an extended social context. Research examining the simultaneous influence of peers and family is scarce, but emerging evidence suggests an important, interacting role of each source of influence that changes from childhood to adolescence.

In one of the earliest studies to examine the differential effects of peers and parents, children from the 3rd to 12th grades rated their attitudes about antisocial, prosocial, and neutral behaviors in the contexts of peer and parent conformity [8]. Peer conformity to antisocial behaviors showed a curvilinear pattern, increasing from childhood, peaking in 9th grade and declining thereafter. Parent conformity to prosocial and neutral behaviors, on the other hand, was high in childhood, but showed declines across adolescence. In a more recent experimental study, 8-60 year olds rated their own perceptions of risky behaviors after viewing the perceptions of other teenagers and adults (albeit not their parents) [7]. All age groups showed greater influence towards adults except for young adolescents (12-14 years), who were more influenced by teenagers. These studies suggest that early adolescence is a window of vulnerability for negative peer influence.

In one of the only neuroimaging studies to jointly examine peer and family influence [61], adolescents were shown their parent and peers' attitudes on neutral stimuli and rated their own attitudes. Although adolescents showed significant shifts in their attitudes towards both parents and peers, parent influence effects were significantly greater than that of peers. At the neural level, no differences were found between parent and peer influence. Both types of influence were similarly associated with increased activation in regions of the social brain (TPJ, DMPFC), self-control (VLPFC), and reward processing (VMPFC), suggesting that social influence involves mental state reasoning as well as inhibition of one's own antecedent attitudes. In the only study to date to examine sex differences in neural responses to both family and peers, youth from 9 to 26 years were scanned as they won money for their friend or their mother [47]. For girls, there was an age-related increase in ventral striatum activation when winning for friends relative to their mother, whereas boys' ventral striatum responses to winning for mother and friend did not differ with age. Thus, girls' prosocial motivations for friends become more salient in adolescence and adulthood, which can have significant implications for peer influence effects in adolescent girls. Together, these studies suggest that early adolescence may be a sensitive period when peer influence may have its largest effect. The family context appears to lay the foundation for neurobiological susceptibility to influence, which is maintained across the adolescent years, whereas peers play a unique role during early adolescent development [27].

Future Directions

Social influences from peers and family are among the most potent predictors of adolescents' initiation and escalation of substance use behaviors. As reviewed here, emerging evidence has identified key neurobiological processes by which parents and peers influence substance use and risk taking via changes in the brain's functional system. Despite these exciting advances, additional research in this area is key to better understand the simultaneous influence of family and peer contexts on adolescent substance use.

Developmental timing. To better understand the etiology of adolescent substance use, it is essential to examine the relative timing of family and peer influence effects [9]. Indeed, prior research has found that family relationships influence younger adolescents' smoking whereas peer relationships influence older adolescents' smoking [62]. Other studies have found that both family and peers influence substance use across adolescence [9, 63]. Interestingly, the form and function of family influence may shift across adolescence. Whereas in early adolescence, parental influence occurs via behavioral control (e.g., monitoring), the transition to high school

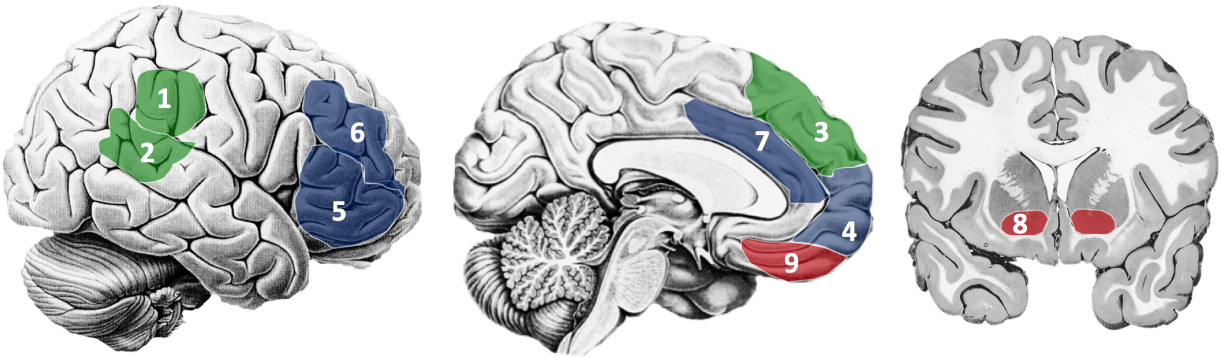
marks a time when relationship quality (e.g., trust, communication) influences adolescent substance use [9]. Thus, the relative contribution of family and peer influence may wax and wane across development as well as change in their function. Future research should therefore utilize longitudinal methods to examine family and peer influence on substance use at multiple time points across childhood and adolescence [9]. Moreover, research is needed that examines the effects of pubertal maturation as well as sex differences in response to family and peer influence, given initial evidence for divergent developmental trajectories in neural responses to parent and peer vicarious rewards among boys and girls [47].

Overlapping influence of family and peer contexts. Despite emerging evidence highlighting the interplay between family and peer contexts, the processes by which family and peers influence adolescent substance use are far from clear. It is possible that family influence precedes peer influence on substance use. That is, the family environment provides the foundation for peer influence effects to occur. For instance, parents who endorse substance use will be more likely to have youth who associate with deviant peers, and such deviant peer association provides the context for youth to access substance use. Indeed, there is evidence that more effective parental monitoring and stronger relationship quality is associated with less deviant peer association, which, in turn, predicts less substance use [64-65]. It is also possible that family contexts interact with peer contexts. For instance, all youth may be vulnerable to peer influence because they value peer acceptance and spend considerable time with friends, but spending time with peers may only lead to substance use behaviors when youth have emotionally volatile families or a lack of family support [66]. Thus, families may exacerbate or buffer peer influence effects. Others have argued that parent and peer influence occur independently [67], or that the strength of each social context is situation and domain dependent [68]. Thus, future research should unpack how parent and peer contexts dynamically operate together to inform substance use. Finally, other important social relationships should be examined, including romantic relationship partners, siblings, and teachers. These social relationships have differential influences on adolescent substance use across development, and their unique contributions should be examined independently, and alongside parents and peers.

Conclusions

In this review, we highlight the important role of social contexts, particularly peers and family, in shaping adolescent substance-use behaviors. Recent affective neuroscience research has developed novel paradigms to identify regions of the brain associated with adolescent risk taking within a social influence. Sensitivity in neural regions involved in social cognition, cognitive control, and reward processing underlie how social influence affects adolescents' decisions to take risks and engage in substance use. Specifically, we show that peers and families play critical roles in decisions related to substance use throughout adolescent development. However, the growing body of research shows that the form and function of influence from both peers and families likely changes across adolescence. For instance, relationship quality of both peers and families significantly interacts with neural mechanisms to predict concurrent and later adolescent risk-taking behaviors; however, dynamic characteristics of these relationships, such as perceived monitoring, trust, victimization, and rejection, may vary in their magnitude of influence depending on adolescents' age, sex, and pubertal stage. Given the complexity of social influences across adolescence, we propose a great need for future research to examine the influence of peers and families both independently, and in tandem, to better understand how these social contexts give rise to, or prevent, adolescent engagement in substance use. Adolescent substance use is a serious public health concern, and identifying mechanisms by which substance use occurs can inform the development of effective interventions.

Figure 1. Brain regions involved in social cognition (green), cognitive control (blue), and reward processing (red).



Social Cognition
(1) Temporal Parietal Junction (TPJ)
(2) Posterior Superior Temporal Sulcus (pSTS)
(3) Dorsomedial PFC (DMPFC)
(4) Medial PFC (MPFC)

Cognitive Control
(4) Medial Prefrontal Cortex
(5) Ventrolateral PFC (VLPFC)
(6) Dorsolateral PFC (DLPFC)
(7) Anterior Cingulate Cortex (ACC)

Reward Processing
(8) Ventral Striatum
(9) Orbitofrontal cortex (OFC)/ Ventromedial PFC (VMPFC)

Disclosures
This article does not contain any studies with human or animal subjects performed by any of the authors. The authors declare no conflicts of interest.

Acknowledgments
Preparation of this manuscript was supported by the National Institutes of Health (R01DA039923 to Telzer) and the National Science Foundation (SES 1459719).

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