Departmental Papers (ASC)   Annenberg School for Communication

Neural Responses to Exclusion Predict Susceptibility to Social Influence

Emily B. Falk
University of Pennsylvania, falk@asc.upenn.edu

Christopher N. Cascio
University of Pennsylvania

Matthew Brook O'Donnell

Joshua Carp

Francis J. Tinney Jr

See next page for additional authors

Recommended Citation

Neural Responses to Exclusion Predict Susceptibility to Social Influence

Abstract

Purpose

Social influence is prominent across the lifespan, but sensitivity to influence is especially high during adolescence and is often associated with increased risk taking. Such risk taking can have dire consequences. For example, in American adolescents, traffic-related crashes are leading causes of nonfatal injury and death. Neural measures may be especially useful in understanding the basic mechanisms of adolescents' vulnerability to peer influence.

Methods

We examined neural responses to social exclusion as potential predictors of risk taking in the presence of peers in recently licensed adolescent drivers. Risk taking was assessed in a driving simulator session occurring approximately 1 week after the neuroimaging session.

Results

Increased activity in neural systems associated with the distress of social exclusion and mentalizing during an exclusion episode predicted increased risk taking in the presence of a peer (controlling for solo risk behavior) during a driving simulator session outside the neuroimaging laboratory 1 week later. These neural measures predicted risky driving behavior above and beyond self-reports of susceptibility to peer pressure and distress during exclusion.

Conclusions

These results address the neural bases of social influence and risk taking; contribute to our understanding of social and emotional function in the adolescent brain; and link neural activity in specific, hypothesized, regions to risk-relevant outcomes beyond the neuroimaging laboratory. Results of this investigation are discussed in terms of the mechanisms underlying risk taking in adolescents and the public health implications for adolescent driving.

Author(s)

Emily B. Falk, Christopher N. Cascio, Matthew Brook O'Donnell, Joshua Carp, Francis J. Tinney Jr, C Raymond Bingham, Jean T. Shope, Marie Claude Ouimet, Anuj K. Pradhan, and Bruce G. Simons-Morton

ELSEVIER

Original article

Neural Responses to Exclusion Predict Susceptibility to Social Influence

Emily B. Falk, Ph.D. [a,*], Christopher N. Cascio, M.A. [a], Matthew Brook O'Donnell, Ph.D. [a],
Joshua Carp, Ph.D. [b], Francis J. Tinney Jr. [c], C. Raymond Bingham, Ph.D. [d], Jean T. Shope, Ph.D. [d],
Marie Claude Ouimet, Ph.D. [e], Anuj K. Pradhan, Ph.D. [d], and Bruce G. Simons-Morton, Ed.D. [f]

[a] Annenberg School for Communication, University of Pennsylvania, Philadelphia, Pennsylvania
[b] Department of Psychology, University of Michigan, Ann Arbor, Michigan
[c] Institute for Social Research, University of Michigan, Ann Arbor, Michigan
[d] University of Michigan Transportation Research Institute (UMTRI), University of Michigan, Ann Arbor, Michigan
[e] Faculty of Medicine and Health Sciences, University of Sherbrooke, Longueuil, Quebec, Canada
[f] Eunice Kennedy Shriver National Institute of Child Health and Human Development, Health Behavior Branch, Bethesda, Maryland

A B S T R A C T

Purpose: Social influence is prominent across the lifespan, but sensitivity to influence is especially high during adolescence and is often associated with increased risk taking. Such risk taking can have dire consequences. For example, in American adolescents, traffic-related crashes are leading causes of nonfatal injury and death. Neural measures may be especially useful in understanding the basic mechanisms of adolescents' vulnerability to peer influence.
Methods: We examined neural responses to social exclusion as potential predictors of risk taking in the presence of peers in recently licensed adolescent drivers. Risk taking was assessed in a driving simulator session occurring approximately 1 week after the neuroimaging session.
Results: Increased activity in neural systems associated with the distress of social exclusion and mentalizing during an exclusion episode predicted increased risk taking in the presence of a peer (controlling for solo risk behavior) during a driving simulator session outside the neuroimaging laboratory 1 week later. These neural measures predicted risky driving behavior above and beyond self-reports of susceptibility to peer pressure and distress during exclusion.
Conclusions: These results address the neural bases of social influence and risk taking; contribute to our understanding of social and emotional function in the adolescent brain; and link neural activity in specific, hypothesized, regions to risk-relevant outcomes beyond the neuroimaging laboratory. Results of this investigation are discussed in terms of the mechanisms underlying risk taking in adolescents and the public health implications for adolescent driving.
© 2014 Society for Adolescent Health and Medicine. Open access under CC BY-NC-ND license.

IMPLICATIONS AND CONTRIBUTION

Adolescents who show the greatest neural reactivity to exclusion may be most susceptible to risk taking in the presence of peers. Neural responses provided information that was not evident from self-reports of susceptibility to peer pressure or participants' distress during exclusion, lending new insight into the mechanisms of peer influence and risk.

Adolescence is a period characterized by heightened responsivity to social influence across domains [1–4], including increased risk taking in the presence of peers [5–7]. This can have dire consequences. For example, in U.S. adolescents, traffic-related crashes are the leading causes of nonfatal injury and death [8]. Evidence is strong that adolescents drive in a more risky fashion compared with adults [9]. Furthermore, adolescents drive in a more risky fashion in the presence of passengers

[10], with fatal crash rates being higher among adolescent drivers carrying young passengers [11,12].

Neural measures may be especially useful in understanding the basic mechanisms of adolescents' vulnerability to peer influence and risk taking. For example, well-established theories suggest that the imbalance between relatively more rapid development of affective processing systems than cognitive control systems leaves adolescents more vulnerable to risk taking than those in other developmental groups [5,6]. However, adolescents are not uniformly susceptible to risk [7,13–15]; recent empirical work and reviews highlight the likely importance of social context and motivational salience [7,14,16,17], among other factors [13], in affecting adolescent responses to social-cognitive and social-affective cues, as well as in resulting risk-taking behavior. However, current understanding of the neural mechanisms of peer influence on risk taking and knowledge of how neural responses to social cues might interact with context and motivation in adolescents are extremely limited (c.f., [18]).

One form of sensitivity to social cues is the cognitive ability to consider and make sense of the contents of other people's minds (termed mentalizing). Primary brain regions associated with mentalizing include the dorsomedial prefrontal cortex (DMPFC) [19], right temporal parietal junction (rTPJ) [20,21], and posterior cingulate cortex (PCC) [22,23]. Individual differences in the sensitivity of this system may also be associated with broader susceptibility to social influence.

A second form of sensitivity to social cues includes affective responses to being accepted or rejected by others [7]. Prior research has demonstrated that heightened reward activity in the presence of peers is associated with risk taking in adolescents [24]. Conversely, sensitivity to "social pain" [25] has not been examined as a predictor of susceptibility to risky influence and is the focus of the present investigation. Social pain is associated with increased neural activity in the anterior insula (AI) and subgenual anterior cingulate cortex (subACC) in adolescents [26], as well as in the dorsal anterior cingulate cortex (dACC) in adults [25,27,28]. Activity within this system is thought to signal that one may not be in line with the group, among other things, and is associated with restoring normative behavior [29,30]. The social pain system may serve to promote learning that keeps individuals in harmony with the group [26,31].

Hypotheses

Consistent with theories that focus on social monitoring during exclusion as a means of remaining or being included as part of the group [32–34], to the extent that individuals are more sensitive to social cues and experience greater physiological reactivity to exclusion, they might be more inclined to behave in ways that preemptively avoid exclusion and promote bonding during social interactions [35]. Given that risk taking in adolescence can be a means of gaining social acceptance [36], those who are more responsive to acute social threats might pre-emptively adjust their behavior to fit in with group norms across situations. More specifically, we hypothesized that increased activity in neural systems associated with social-cognitive sensitivity (mentalizing: DMPFC [19], rTPJ [20,21], and PCC [22,23]) as well as social-affective sensitivity ("social pain": AI, subACC [26]) during exclusion would be associated with differences in risk taking in the presence of peers in a separate simulated driving session, controlling for solo risk-

taking behavior. It is also possible that the dACC might play a role, given prior findings regarding social pain in adults [25,27,28].

To test these hypotheses, we conducted a two-appointment study in which neural responses were recorded using functional magnetic resonance imaging (fMRI) during social exclusion in an especially at-risk group for fatal crash: recently licensed male adolescents [11,37]. Individual differences in neural sensitivity to exclusion during the initial fMRI scan were then used to predict individual differences in risk taking in the presence of peers, controlling for solo risk behavior, in a separate driving simulator session approximately 1 week later (Figure 1). Driving simulation is consistently associated with a number of real-world driving behaviors [38] and is a safe method for investigating the effect of peer influences on risky driving behavior while maintaining a high degree of both experimental control and external validity [39].

Materials and Methods

Participants

Thirty-six neurotypical adolescent males aged 16–17 years ($M = 16.8$, SD = .47) were recruited through the Michigan state driver registry database (driver history record) as part of a larger study on peer influences on adolescent driving [40]. These participants successfully completed both an fMRI session and a separate driving simulator appointment approximately 1 week later (Figure 1; see Supplementary Data for full recruitment and simulator details). Within the 4–9 months prior to the scan, all participants had obtained a Level 2 (intermediate) Michigan driver license allowing them to drive independently but with passenger and night driving restrictions. In addition, participants met standard MRI safety criteria (Supplementary Data). Legal guardians provided written informed consent, and adolescents provided written assent.

Procedure

Session 1: fMRI. At their first (fMRI) session, participants were introduced to two gender-matched peer confederates. Participants were told that they would be playing some computer games on their own, as well as in a group with the other "participants." Research assistants then took them to separate private rooms where the real participant completed the online prescan questionnaires and was given further instructions about the fMRI tasks; the confederates were not involved further in study procedures. Participants next completed a series of tasks within the fMRI scanner, including a game called Cyberball. Cyberball has been validated in a number of behavioral and neuroimaging studies as a reliable way of simulating the experience of social exclusion [27,41]. A fair game of Cyberball was always played first, in which the participant and two virtual players received the ball equally often. This was followed by an unfair game, in which the participant and virtual players started out receiving the ball equally often, but where the participant was left out after a few throws, simulating exclusion. Order of the rounds was held constant to preserve the psychological experience across participants (see Supplementary Data). After the scan, participants completed a second set of questionnaires, including all the questionnaires that are the focus of this investigation.

Figure 1. Overview of study protocol. First, participants completed an fMRI scanning session in which neural activity was recorded during an exclusion episode. Next, approximately 1 week later, driving behavior was recorded in a driving simulator, while participants drove alone and in the presence of a peer (confederate).

Figure 2. Driving simulator. During the driving session, participants drove in a fixed-base, partial cab driving simulator.

Self-reports. Participants completed online self-report assessments of Susceptibility to Peer Pressure (SPP) [42,43], resistance to peer influence (RPI) [44], and distress during the exclusion episode (the need threat scale [NTS]) [45] using Qualtrics. Higher SPP scores indicate increased SPP. Higher RPI scores indicate greater ability to resist peer influence. Higher NTS scores indicate greater need satisfaction, and lower scores indicate greater threat or distress.

Session 2: driving simulation. Approximately 1 week after the fMRI session, participants completed a driving simulator appointment. Within this session, participants drove a practice drive to habituate to a state-of-the-art fixed-base driving simulator (Figure 2; Supplementary Data). All participants then drove alone and in the presence of a young, male confederate, whose behavior was manipulated to portray one of the two risk levels (risk accepting or not risk accepting) as part of a larger study on susceptibility to normative pressures in the driving context [40].[1]

Participants were randomly assigned to conditions blindly by an investigator who was not involved in any of the fMRI or simulator testing within a 2 × 2 × 2 counterbalanced block design composed of conditions: confederate passenger behavior (high

risk vs. low risk) × drive order (drove alone or with the confederate passenger first) × order of scenarios presented within each drive (order A or order B). The solo and passenger drives each consisted of the same number of intersections and yellow light scenarios.

Data acquisition and analysis

fMRI data. Imaging data were acquired using a 3-T GE Signa MRI scanner (GE Healthcare, Fairfield, CT). One functional run was recorded for each participant (251 volumes) during the Cyberball tasks. Functional data were prepared using a standard preprocessing stream and analyzed using statistical parametric mapping (SPM8) [46]. Data were modeled using the general linear model as implemented in SPM8. Three trial phases were modeled: social inclusion, social exclusion, and visual tracking. Additional details on scanning parameters, processing, and modeling are available in the Supplementary Data.

Regions of interest. Regions of interest (ROIs) were constructed for each of the two a priori hypothesized networks of interest (social pain, Figure 3A; mentalizing, Figure 5A; ROI definitions in Supplementary Data). The social pain network was defined based on prior work examining exclusion in adolescents [26] to include bilateral AI and subACC (Figure 3A). An additional, exploratory, network was constructed to also include the dACC, given prior findings regarding social pain in adults [25,27,28]. The mentalizing network was defined to include DMPFC, rTPJ, and PCC (Figure 5A) [19–23]. Separate ROIs for subregions

---

[1] This manipulation and associated effects from the larger study are described in more extensive detail elsewhere [40] and are not the focus of the current investigation. However, confederate behavior is accounted for in all of our models.
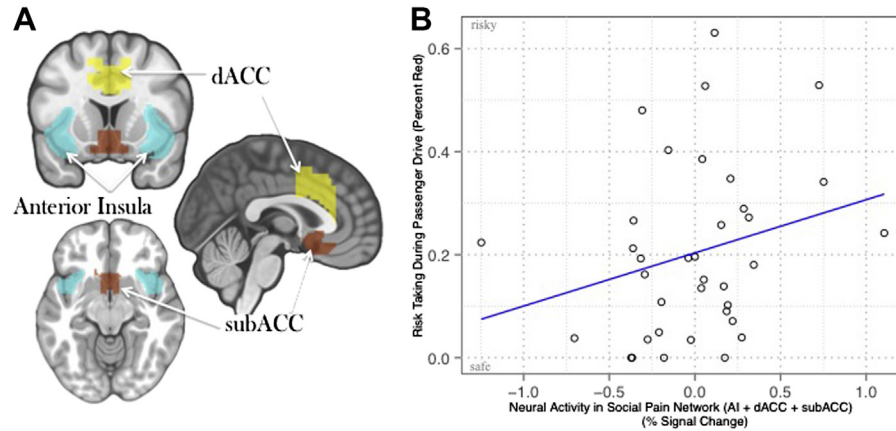
Figure 3. (A) Anatomical ROIs comprising the putative social pain system. Primary results averaged activity in AI and subACC. Additional results also computed including the dACC. (B) Scatter plot of residualized percent signal change for exclusion > inclusion extracted from the combined ROIs during Cyberball predicting risk taking during the passenger drive, controlling for drive order, passenger type, and solo drive behavior.

within each network were also constructed to facilitate secondary follow-up analyses, parsing which regions within the network drive the effects observed. Finally, we identified regions within the broader anatomically defined ROIs that were associated with the main effect of exclusion > inclusion during Cyberball (Figures 4A and 6A; Supplementary Table S5), treated as functionally defined regions of interest (fROIs). The anatomical ROI estimates reflect individual differences in neural activity within a priori hypothesized regions. Supplemental analyses focusing on the fROIs (1) confirm that the task is engaging the regions expected and (2) provide a way of testing whether the subregions that are engaged during exclusion most (on average) predict changes in our dependent measures of risk taking.

Analysis plan

Neural data and driving simulator data were combined using a brain-as-predictor framework [47] in a series of ordinary least squares regressions, implemented in R version 2.15.1. Predictors of interest were estimates of exclusion-related neural activity in the social pain network and the mentalizing network. Parameter estimates from the anatomical and functional ROIs were extracted for each subject using MarsBar from the exclusion > inclusion contrast. Estimates from the functional ROIs were extracted at the cluster level. All extracted parameter estimates were transformed into units of percent signal change by dividing by the grand mean value from the whole run of Cyberball [48,49] and then used as predictors of risky behavior in subsequent models.

Our primary dependent measure of risk taking was the proportion of time during each drive that participants were in the intersection when the traffic signals were red (percent red). The simulation was designed to present dilemma zone (yellow light) situations that would elicit risk-taking behaviors, that is, willingness to run the light. Time spent in the intersection while the light is red is indicative of the driver's willingness to push through lights in a dilemma situation, a measure that is translatable to real-world driving where intersection crashes can be among the most serious [40].

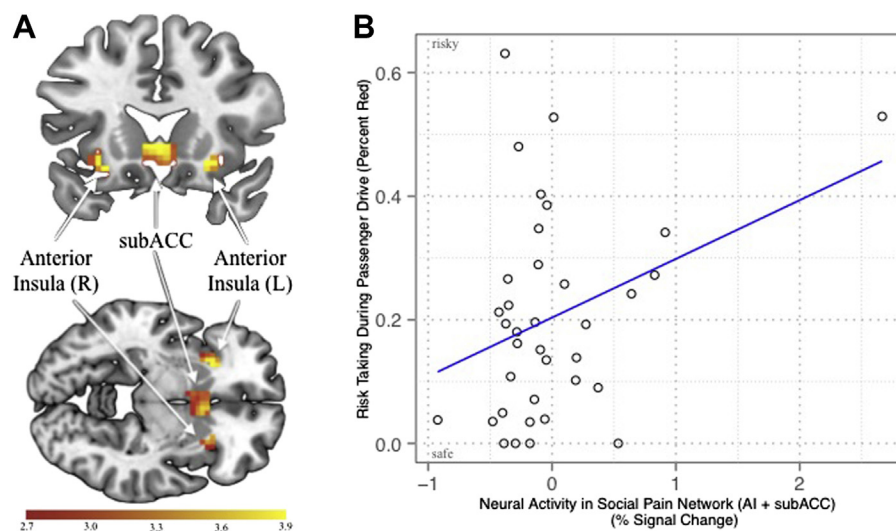**Figure 4.** (A) fROIs were identified within the anatomical ROIs shown in Figure 3, during exclusion compared with inclusion (thresholded at *p* < .005, *k* = 10). (B) Scatter plot of residualized percent signal change for exclusion > inclusion during Cyberball within the identified clusters, predicting risk taking during the passenger drive, controlling for drive order, passenger type, and solo drive behavior.

Figure 5. (A) Anatomical ROIs comprising the mentalizing system. Primary results averaged activity in DMPFC, rTPJ, and PCC. (B) Scatter plot of residualized percent signal change for exclusion > inclusion extracted from the combined ROIs during Cyberball predicting risk taking during the passenger drive, controlling for drive order, passenger type, and solo drive behavior.

We operationalized susceptibility to social influence in terms of risk-taking behaviors during the passenger drive, controlling for baseline (solo) risk-taking behavior. We ran separate, successive regression models to examine the relationships between neural activity in our a priori hypothesized mentalizing and social pain networks, respectively, and risk taking during the passenger drive. Parallel models were then run examining self-reports of distress during exclusion and SPP as predictors, respectively. Finally, neural data and self-report data were entered in a series of combined models that examined the effects of each neural network, separately, above and beyond self-report predictors. To control for potential learning effects of repeated exposure to the driving simulator, as well as confederate behavior, all models also included solo drive risk-taking behavior, confederate condition (risk accepting vs. not risk accepting), and drive order (participant first vs. confederate first) as covariates.

Results

Behavioral results

Self-reports. At baseline, participants reported being somewhat unsusceptible to peer influence (SPP: $M = 1.95$, corresponding to an average response indicating that they would "probably not" go along with their friend's risky behavior, SD = .49, Cronbach's $\alpha = .78$) and somewhat more similar to the person who resists peer influence than to someone who is susceptible to peer influence (RPI: $M = 2.93$, SD = .46, Cronbach's $\alpha = .53$). Given that RPI did not exhibit strong reliability and the results of our primary target analyses are similar for both SPP and RPI, we report results throughout the rest of the article focusing on SPP. Consistent with past work examining the effects of Cyberball in adolescents [26], participants reported relatively neutral levels of threat following exclusion (NTS overall score: $M = 3.89$,

Figure 6. (A) fROIs were identified within the anatomical ROIs shown in Figure 5, during exclusion compared with inclusion (thresholded at $p < .005$, $k = 10$). (B) Scatter plot of residualized percent signal change for exclusion > inclusion during Cyberball within the identified clusters, predicting risk taking during the passenger drive, controlling for drive order, passenger type, and solo drive behavior.
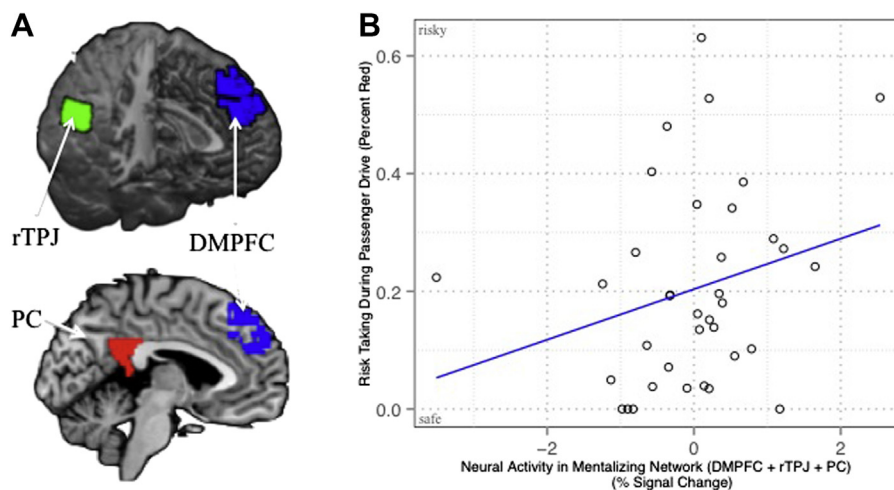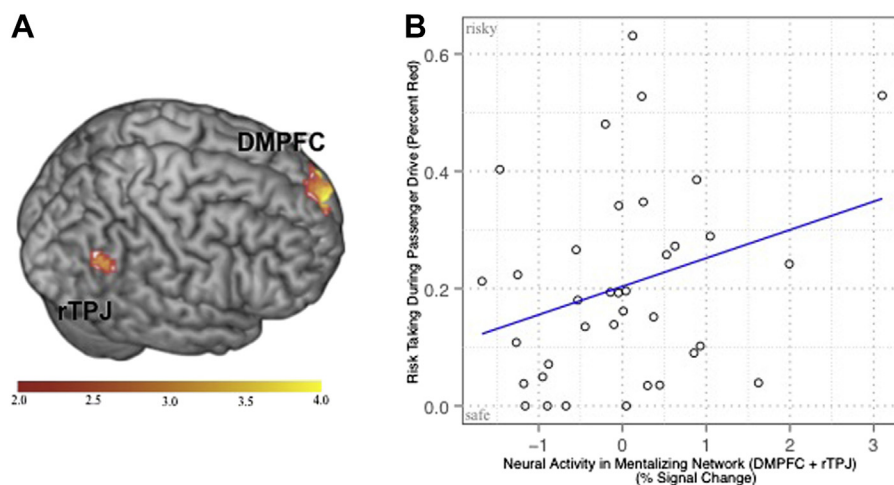
corresponding to an average response between "neither agree nor disagree" and "somewhat disagree", with respect to items measuring felt threat, SD = .97, Cronbach's α = .92).

Risky behavior during the drives. Risk taking during solo and passenger driving was highly correlated with one another (r = .78, $p < .001$). Consistent with prior findings [24], however, we observed a main effect of passenger presence, such that on average, participants drove in a riskier manner during the passenger drive (percent red: $M = .21$, SD = .17) than during the solo drive (percent red: $M = .13$, SD = .14, $t(36) = 4.33$, $p < .001$; higher percentages of time in the intersection when the light is red indicate increased risk taking). We also observed an effect of confederate passenger type, such that participants exposed to the risk-accepting confederate passenger took more risks on average than those exposed to the risk-averse confederate passenger ($t(34) = -2.15$, $p = .04$), although participants took more risks with both types of passengers compared with driving alone ($t(35) = 4.29$, $p < .001$). Confederate type did not interact with our neural predictors to predict risk taking or susceptibility to influence in any of the models that form the primary focus of this article (ps range from .15 to .82).

Self-report predictors of risky influence. Self-reports of SPP were significantly positively associated with increased risk-taking behavior during the passenger drive ($\beta = .23$, $t(32) = 2.12$, $p = .042$), controlling for solo risk-taking behavior, confederate condition, and drive order. Self-reports of distress during exclusion were uncorrelated with risk-taking behavior during the passenger drive ($\beta = .05$, $t(32) = .48$, $p = .638$).

Neural results

Neural activity in the social pain network previously observed in adolescents for the exclusion > inclusion contrast (AI + subACC) was significantly positively associated with risky behavior in the driving simulator (Table 1A, Figure 3B); those who showed greater activity in the social pain network during exclusion > inclusion drove in a riskier manner in the presence of a peer, controlling for solo drive behavior. Parallel results were observed treating the change score from solo to passenger drive as the dependent measure. In addition, measures of neural activity in the social pain network remained significantly positively associated with risk taking during the passenger drive, controlling for SPP (Table 1B) and distress during the exclusion episode (Table 1C). Parallel results were observed in our fROIs (Tables 1D–1F) and in the extended social pain network that included AI, subACC, and dACC [25,27,28]. However, the results were strongest in the AI and subACC regions that were also most strongly associated with the main effect of the task (Supplementary Table S1; see also, supplemental whole brain search for regions associated with risk taking in passenger, compared with solo drives, Supplementary Table S6).

We next examined activity in the mentalizing network during exclusion versus inclusion as a predictor of later susceptibility to risky influence in the driving simulator (Table 2A). Consistent with the idea that broader sensitivity to social cues is associated with susceptibility to influence, neural activity in the mentalizing network (DMPFC + rTPJ + PCC; Figure 5A) was associated with risk taking during the passenger drive (Table 2A; Figure 5B). Parallel results were observed treating the change score from solo to passenger drive as the dependent measure. In addition,

neural activity in the mentalizing network remained significantly positively associated with risk taking during the passenger drive, controlling for SPP and distress during the exclusion episode (Tables 2B and 2C; see Supplementary Table S2 for subregion analyses), and using fROIs (Tables 2D–2F).

Discussion

We examined neural and self-report predictors of susceptibility to risky social influence in a sample of recently licensed adolescent drivers. Consistent with past work [24], the mere presence of a peer was enough to promote risk, whether or not explicit risky norms were communicated (although the effect was strongest with passengers who promote risk-accepting norms [40]). Furthermore, increased activity in both social-cognitive (mentalizing) and social-affective (social pain) systems during exclusion predicted increased risky behavior in the presence of a peer (confederate), controlling for solo risk-taking behavior and the confederate's behavior. The brain undergoes development of both affective and social-cognitive systems across adolescence [5,15,16,50–53], and adolescence is thought to be a period of reorienting "social and motivational tendencies, which in turn influence behavior and emotion in adolescence depending upon interactions with social context" [54]. Our data suggest that individual differences in the responsiveness of these systems may relate to adolescent behavior in the presence of peers, controlling for their solo risk behavior.

Social psychologists have suggested that one function of human susceptibility to social influence is to promote bonding and group harmony [55], and humans have developed strong biological alarm systems to prevent social isolation [25,27,56–60]. Indeed, social monitoring during exclusion may serve as a means of remaining connected with the group [32–34]. More generally, these results may be interpreted within the framework of rewards and punishments that are known to drive human and animal behavior and learning [56,61–65]. Both real and anticipated rewards reinforce behaviors, whereas real and anticipated punishments inhibit behaviors that are known to produce punishment [66–70], with variation in individual differences regarding sensitivity to rewards and punishments in motivating behavior [61–63]. Our data suggest that such differences may also translate into susceptibility to behavior change in the presence of peers and indicate specific neural processes that may underlie such effects; adolescents whose brains are more responsive to social cues during exclusion may be more likely to change their behavior in the presence of peers (perhaps to avoid social exclusion or promote affiliation), regardless of whether peers explicitly encourage risk or not.

Neural activity explains variance in risky behavior that is not explained by self-reports

Activity in both the social pain and mentalizing networks remained significant predictors of risky driving in the presence of a peer, not only when controlling for individual differences in solo risk behavior and confederate behavior but also when controlling for self-reports of SPP [42] and distress during Cyberball. Although self-reported SPP predicted risk taking in the presence of a passenger during the driving simulator session, when neural activity and self-report were simultaneously modeled, the neural activity predicted variance in the behavioral outcome, above and beyond self-report. This

Table 1
Relationship between neural activity in the social pain network during Cyberball exclusion > inclusion and risk taking (percent red) in the presence of peers in the driving simulator session

1A: Effect of neural activity in the social pain network (AI and subACC), controlling for passenger type, drive order, and solo risk-taking behavior (as measured by performance in the solo drive at the simulator); 1B: Study of the same relationships as in Table 1A but controls for self-reported SPP; 1C: Study of the same relationships as in Table 1A but controls for distress during the exclusion episode (Cyberball), as measured by the NTS; 1D: Study of the same relationships as in Table 1A but replaces the anatomical ROI with an fROI defined based on exclusion > inclusion within hypothesized anatomical regions; 1E: Study of the same relationships as in Table 1D but controls for self-reported SPP; 1F: Study of the same relationships as in Table 1D but controls for distress during the exclusion episode (Cyberball), as measured by the NTS.

Table 1A

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.028	.035	.085	.796	.432
Passenger type	.091	.036	.276	2.546	.016
Solo drive	.779	.128	.671	6.07	<.001
Cyber (AI and subACC)	.119	.038	.314	3.102	.004
			N = 36	Model R square = .718	

Table 1B

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.044	.036	.135	1.225	.23
Passenger type	.081	.036	.246	2.269	.031
Solo drive	.78	.126	.672	6.19	<.001
Cyber (AI and subACC)	.104	.039	.275	2.678	.012
SPP	.05	.034	.153	1.467	.153
			N = 36	Model R square = .737	

Table 1C

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.033	.037	.099	.894	.379
Passenger type	.096	.037	.289	2.567	.016
Solo drive	.781	.132	.67	5.93	<.001
Cyber (AI and subACC)	.118	.039	.312	2.992	.006
NTS	.007	.016	.04	.399	.693
			N = 36	Model R square = .721	

Table 1D

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.034	.033	.105	1.029	.311
Passenger type	.082	.033	.251	2.516	.017
Solo drive	.739	.123	.637	5.997	<.001
Cyber fROI (AI and subACC)	.095	.025	.351	3.74	.001
			N = 36	Model R square = .746	

Table 1E

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.05	.034	.151	1.442	.16
Passenger type	.075	.033	.227	2.291	.029
Solo drive	.743	.121	.64	6.141	<.001
Cyber fROI (AI and subACC)	.086	.026	.317	3.348	.002
SPP	.048	.032	.145	1.477	.15
			N = 36	Model R square = .763	

Table 1F

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.039	.035	.118	1.116	.273
Passenger type	.088	.035	.264	2.536	.017
Solo drive	.741	.127	.635	5.854	<.001
Cyber fROI (AI and subACC)	.095	.026	.35	3.614	.001
NTS	.004	.016	.027	.28	.782
			N = 36	Model R square = .748	

AI = anterior insula; fROI = functionally defined region of interest; NTS = need threat scale; SE = standard error; SPP = susceptibility to peer pressure; subACC = subgenual anterior cingulate cortex.

Table 2
Relationship between neural activity in the mentalizing network during exclusion > inclusion in Cyberball and risk taking (percent red) in the presence of peers in the driving simulator session

2A: Effect of neural activity in the mentalizing network (DMPFC, rTPJ, and PCC), controlling for passenger type, drive order, and solo risk-taking behavior (as measured by performance in the solo drive at the simulator); 2B: Study of the same relationships as in Table 2A but controls for self-reported SPP; 2C: Study of the same relationships as in Table 2A but controls for distress during the exclusion episode (Cyberball), as measured by the NTS; 2D: Study of the same relationships as in Table 2A but replaces the anatomical ROI with an fROI defined based on exclusion > inclusion within hypothesized anatomical regions; 2E: Study of the same relationships as in Table 2D but controls for self-reported SPP; 2F: Study of the same relationships as in Table 2D but controls for distress during the exclusion episode (Cyberball), as measured by the NTS.

Table 2A

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.005	.037	.017	.148	.884
Passenger type	.075	.036	.229	2.11	.043
Solo drive	.822	.132	.708	6.229	<.001
Cyber (DMPFC, rTPJ, and PCC)	.079	.03	.274	2.629	.013
			N = 36	Model R square = .698	

Table 2B

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.027	.038	.082	.706	.486
Passenger type	.066	.035	.202	1.888	.069
Solo drive	.817	.129	.704	6.354	<.001
Cyber (DMPFC, rTPJ, and PCC)	.069	.03	.237	2.287	.029
SPP	.057	.035	.172	1.628	.114
			N = 36	Model R square = .723	

Table 2C

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.011	.038	.034	.292	.773
Passenger type	.082	.038	.248	2.191	.037
Solo drive	.825	.135	.708	6.106	<.001
Cyber (DMPFC, rTPJ, and PCC)	.079	.031	.274	2.572	.015
NTS	.01	.017	.063	.607	.549
			N = 36	Model R square = .703	

Table 2D

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	−.007	.036	−.02	−.185	.854
Passenger type	.068	.034	.208	2.025	.052
Solo drive	.828	.127	.714	6.507	<.001
Cyber fROI (DMPFC and rTPJ)	.072	.023	.314	3.124	.004
			N = 36	Model R square = .719	

Table 2E

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.01	.04	.031	.26	.797
Passenger type	.062	.034	.19	1.818	.079
Solo drive	.824	.127	.71	6.479	<.001
Cyber fROI (DMPFC and rTPJ)	.062	.025	.269	2.456	.02
SPP	.038	.037	.115	1.03	.311
			N = 36	Model R square = .729	

Table 2F

Variable	Unstandardized coefficients		Standardized coefficients		Significance
	B	SE	Beta	t	
Drive order	.001	.037	.002	.014	.989
Passenger type	.079	.035	.239	2.242	.033
Solo drive	.832	.129	.714	6.469	<.001
Cyber fROI (DMPFC and rTPJ)	.075	.023	.327	3.204	.003
NTS	.014	.016	.085	.853	.401
			N = 36	Model R square = .730	

DMPFC = dorsomedial prefrontal cortex; fROI = functionally defined region of interest; NTS = need threat scale; PCC = posterior cingulate cortex; rTPJ = right temporal parietal junction; SE = standard error; SPP = susceptibility to peer pressure.

suggests that the neural measure of "social pain" may index processes and capture information that participants are either not consciously aware of or unwilling to report (e.g., due to self-presentation concerns that are especially salient during adolescence).

Strengths and limitations

The combination of highly controlled neuroimaging paradigms with outcome data from a social interaction in a driving simulator allowed us a high degree of control over external confounds while still maintaining a high degree of internal validity and external validity. However, limitations present opportunities for future research. First, although recruited broadly from our target population via the state driver history record, the final sample is not a random subset of the population, given that participants were all volunteer responders. Second, our chief findings are correlational; it is possible that individuals who later took more risks in the presence of peers also showed increased responsiveness in the social pain and mentalizing networks due to a third variable or that susceptibility to risky peer influence sensitizes the brain systems in question. Prior work [26] reported that neural activity in the brain's social pain network (including dACC, subgenual ACC, and AI), as well as DMPFC during exclusion > inclusion, was associated with interpersonal competence scores in adolescents, which may also be associated with social influences [71]. Third, driving simulators provide external validity with respect to relative risk behavior under different conditions while controlling for a number of confounding variables in actual driving and ethically manipulating crash risk [38,39]. However, future research that combines parallel neural measures with instrumented vehicles on real roads would provide additional information about the relationship between our neural variables and naturalistic driving. Finally, we focused on recently licensed male adolescents, given that they are the highest risk group for fatal crash, especially in the presence of other male adolescents [11,37]. Future research on samples that include both men and women, as well as younger and older participants, and other risk behaviors will inform the boundary conditions on the effects observed. It is also possible that more robust effects of SPP would be observed if peer pressure were more directly applied, suggesting a key avenue for future research.

Summary

In sum, adolescents who demonstrated greatest activity in social-cognitive (mentalizing) and social-affective (social pain) brain systems during exclusion showed greatest susceptibility to risk taking in the presence of peers, suggesting potential mechanisms of peer influence on risk taking in adolescents. Furthermore, neural activity forecasted variability in driving risk with a peer that was not explained by self-report alone. Given that traffic-related crashes are a leading cause of adolescent mortality and nonfatal injury, these data also contribute understanding that might eventually inform programs and policies to reduce injuries and fatalities in adolescents.

Supplementary Data

Supplementary data related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jadohealth.2013.12.035

References

[1] Steinberg L, Morris AS. Adolescent development. Annu Rev Psychol 2001; 52: p. 83–110.
[2] Juvonen J, Galvan A. Peer influence in involuntary social groups: Lessons from research on bullying. In: Prinstein MJ, Dodge KA, eds. Peer influence processes among youth. New York: Guilford Press; 2008:225–44.
[3] Brown BB, Larson J. Peer relationships in adolescence. In: Lerner R, Steinberg L, eds. Handbook of adolescent psychology. Hoboken, NJ: John Wiley & Sons; 2009: p. 74–103.
[4] Somerville LH. The teenage brain: Sensitivity to social evaluation. Curr Dir Psychol Sci 2013;22:129–35.
[5] Steinberg L. A social neuroscience perspective on adolescent risk-taking. Dev Rev 2008;28:78–106.
[6] Casey BJ, Getz S, Galvan A. The adolescent brain. Dev Rev 2008;28:62–77.
[7] Crone EA, Dahl RE. Understanding adolescence as a period of social-affective engagement and goal flexibility. Nat Rev Neurosci 2012;13: 636–50.
[8] NHTSA. Traffic safety facts: Young drivers, 2008 data, N.H.T.S.A. US DOT, Editor. Washington DC: NCSA Publication; 2008.
[9] Simons-Morton BG, Ouimet MC, Zhang Z, et al. Crash and risky driving involvement among novice adolescent drivers and their parents. Am J Public Health 2011;101:2362–7.
[10] Simons-Morton B, Lerner N, Singer J. The observed effects of teenage passengers on the risky driving behavior of teenage drivers. Accid Anal Prev 2005;37:973–82.
[11] Ouimet MC, Simons-Morton BG, Zador PL, et al. Using the U.S. National Household Travel Survey to estimate the impact of passenger characteristics on young drivers' relative risk of fatal crash involvement. Accid Anal Prev 2010;42:689–94.
[12] Williams AF, Teft BC. Characteristics of teens-with-teens fatal crashes in the United States, 2005–2010. J Saf Res 2013. advance online.
[13] Romer D. Adolescent risk taking, impulsivity, and brain development: Implications for prevention. Dev Psychobiol 2010;52:263–76.
[14] Pfeifer JH, Allen NB. Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. Trends Cogn Sci 2012;16:322–9.
[15] Pfeifer JH, Blakemore SJ. Adolescent social cognitive and affective neuroscience: Past, present, and future. Soc Cogn Affect Neurosci 2012;7:1–10.
[16] Sebastian C, Viding E, Williams KD, Blakemore SJ. Social brain development and the affective consequences of ostracism in adolescence. Brain Cogn 2010;72:134–45.
[17] Blakemore SJ, Mills KL. Is adolescence a sensitive period for sociocultural processing? Annu Rev Psychol 2013.

[18] Peake SJ, Dishion TJ, Stormshak EA, et al. Risk-taking and social exclusion in adolescence: Neural mechanisms underlying peer influences on decision-making. Neuroimage 2013;82:23–34.

[19] Lieberman MD. Social cognitive neuroscience. In: Fiske S, Gilbert D, Lindzey G, eds. Handbook of social psychology. New York, NY: McGraw-Hill; 2010:143–93.

[20] Saxe, R., The right temporo-parietal junction: A specific brain region for thinking about thoughts, in Handbook of theory of mind, A. Leslie and T. German, Editors. 2010.

[21] Saxe R, Wexler A. Making sense of another mind: The role of the right temporo-parietal junction. Neuropsychologia 2005;43:1391–9.

[22] Spunt RP, Satpute AB, Lieberman MD. Identifying the what, why, and how of an observed action: An fMRI study of mentalizing and mechanizing during action observation. J Cogn Neurosci 2010;12:63–74.

[23] Fletcher PC, Happé F, Frith U, et al. Other minds in the brain: A functional imaging study of "theory of mind" in story comprehension. Cognition 1995;57:109–28.

[24] Chein J, Albert D, O'Brien L, et al. Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. Developmental Sci 2010; 14:F1–10.

[25] Eisenberger NI, Lieberman MD, Williams KD. Does rejection hurt? An fMRI study of social exclusion. Science 2003;302:290–2.

[26] Masten CL, Eisenberger NI, Borofsky LA, et al. Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. Social Cogn Affective Neurosci 2009;4:143–57.

[27] Eisenberger NI. The neural bases of social pain: Evidence for shared representations with physical pain. Psychosom Med 2012;74: 126–35.

[28] Kross E, Berman MG, Mischel W, et al. Social rejection shares somatosensory representations with physical pain. Proc Natl Acad Sci U S A 2011;108: 6270–5.

[29] Berns GS, Capra CM, Moore S, Noussair C. Neural mechanisms of the influence of popularity on adolescent ratings of music. Neuroimage 2010; 49:2687–96.

[30] Klucharev V, Hytönen K, Rijpkema M, et al. Reinforcement learning signal predicts social conformity. Neuron 2009;61:140–51.

[31] Masten CL, Telzer EH, Fuligni AJ, et al. Time spent with friends in adolescence relates to less neural sensitivity to later peer rejection. Social Cogn Affective Neurosci 2012;7:106–14.

[32] Leary MR, Haupt AL, Strausser KS, Chokel JT. Calibrating the sociometer: The relationship between interpersonal appraisals and state self-esteem. J Personal Social Psychol 1998;74:1290–9.

[33] Pickett CL, Gardner WL. The social monitoring system: Enhanced sensitivity to social clues as an adaptive response to social exclusion. In: Williams KD, Forgas JP, Hippel WV, eds. The Social Outcast: Ostracism, Social Exclusion, Rejection, and Bullying. New York: Psychology Press; 2005.

[34] Leary MR, Tambor ES, Terdal SK, Downs DL. Self-Esteem as an interpersonal monitor – The sociometer hypothesis. J Personal Social Psychol 1995;68: 518–30.

[35] Falk EB, Way BM, Jasinska AJ. An imaging genetics approach to understanding social influence. Front hum neurosci 2012;6:1–13.

[36] Allen JP, Porter MR, McFarland FC, et al. The two faces of adolescents' success with peers: Adolescent popularity, social adaptation, and deviant behavior. Child Development 2005;76:747–60.

[37] Williams AF. Teenage drivers: Patterns of risk. J Saf Res 2003;34:5–15.

[38] Mullen N, Charlton J, Devlin A, Bédard M. Simulator validity: Behaviors observed on the simulator and on the road. In: Fisher DL, Rizzo M, Caird J, Lee JD, eds. Handbook of Driving Simulation for Engineering, Medicine and Psychology. Boca Raton, FL: CRC Press; 2011:13.1–13.18.

[39] Caird JK, Horrey WJ. Twelve practical and useful questions about driving simulation. In: Fisher DL, Rizzo M, Caird J, Lee JD, eds. Handbook of Driving Simulation for Engineering, Medicine and Psychology. Boca Raton, FL: CRC Press; 2011:5.1-5.16.

[40] Simons-Morton BG, Bingham CR, Falk EB, et al. Experimental effects of injunctive norms on simulated risky driving among teenage males [published online ahead of print January 27, 2014]. Health Psychol http://dx.doi.org/10.1037/a0034837.

[41] Williams KD. Ostracism. Annu Rev Psychol 2007;58:425–52.

[42] Dielman TE, Campanelli PC, Shope JT, Butchart AT. Susceptibility to peer pressure, self-esteem, and health locus of control as correlates of adolescent substance abuse. Health Educ Q 1987;14:207–21.

[43] Simons-Morton BG, Ouimet MC, Chen R, et al. Peer influence predicts speeding prevalence among teenage drivers. J Saf Res 2012;43:397–403.

[44] Steinberg L, Monahan KC. Age differences in resistance to peer influence. Dev Psychol 2007;43:1531–43.

[45] van Beest I, Williams KD. When inclusion costs and ostracism pays, ostracism still hurts. J Personal Social Psychol 2006;91:918–28.

[46] SPM8: Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK.

[47] Berkman ET, Falk EB. Beyond brain mapping: Using the brain to predict real-world outcomes. Curr Dir Psychol Sci 2013;22:45–55.

[48] Brett M, Anton J-L, Valabregue R, Poline J-B. Region of interest analysis using an SPM toolbox, in The 8th International Conference on functional mapping of the human brain 2002: Sendai, Japan.

[49] Poldrack RA, Mumford JA, Nichols TE. Handbook of functional MRI data analysis. Cambridge University Press; 2011.

[50] Blakemore SJ. Development of the social brain in adolescence. J R Soc Med 2012;105:111–6.

[51] Blakemore SJ. The social brain in adolescence. Nat Rev Neurosci 2008;9: 267–77.

[52] Nelson EE, Leibenluft E, McClure EB, Pine DS. The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. Psychol Med 2005;35:163–74.

[53] Casey BJ, Jones RM, Hare TA. The adolescent brain. Ann N Y Acad Sci 2008; 1124:111–26.

[54] Forbes EE, Dahl RE. Pubertal development and behavior: Hormonal activation of social and motivational tendencies. Brain Cogn 2010;72:66–72.

[55] Cialdini RB, Goldstein NJ. Social influence: Compliance and conformity. Annu Rev Psychol 2004;55:591–621.

[56] Panksepp J, Herman B, Conner R, et al. The biology of social attachments: Opiates alleviate separation distress. Biol Psychiatry 1978;13:607–18.

[57] Cacioppo JT, Hawkley LC, Crawford LE, et al. Loneliness and health: Potential mechanisms. Psychosom Med 2002;64:407–17.

[58] Hawkley LC, Burleson MH, Berntson GG, Cacioppo JT. Loneliness in everyday life: Cardiovascular activity, psychosocial context, and health behaviors. J Pers Soc Psychol 2003;85:105–20.

[59] Hawkley LC, Thisted RA, Masi CM, Cacioppo JT. Loneliness predicts increased blood pressure: 5-year cross-lagged analyses in middle-aged and older adults. Psychol Aging 2010;25:132–41.

[60] Peters E, Riksen-Walraven JM, Cillessen AH, de Weerth C. Peer rejection and HPA activity in middle childhood: Friendship makes a difference. Child Dev 2011;82:1906–20.

[61] Carver C, White T. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scale. J Personal Social Psychol 1994;67:319–33.

[62] Cacioppo JT, Gardner WL, Berntson GG. Beyond bipolar conceptualizations and measures: The case of attitudes and evaluative space. Pers Soc Psychol Rev 1997;1:3–25.

[63] Gray JA. Brain systems that mediate both emotion and cognition. Cogn Emot 1990;4:269–88.

[64] Sobotka SS, Davidson RJ, Senulis JA. Anterior brain electrical asymmetries in response to reward and punishment. Electroencephalogr Clin Neurophysiol 1992;83:236–47.

[65] Skinner BF. Operant behavior. Am Psychol 1963;18:503–15.

[66] Knutson B, Greer SM. Anticipatory affect: Neural correlates and consequences for choice. Philos Trans R Soc Lond B Biol Sci 2008;363:3771–86.

[67] Knutson B, Rick S, Wimmer GE, et al. Neural predictors of purchases. Neuron 2007;53:147–56.

[68] Adcock RA, Thangavel A, Whitfield-Gabrieli S, et al. Reward-motivated learning: Mesolimbic activation precedes memory formation. Neuron 2006;50:507–17.

[69] Berridge KC. From prediction error to incentive salience: Mesolimbic computation of reward motivation. Eur J Neurosci 2012;35:1124-1143.

[70] Gray JA. The psychology of fear and stress. 2nd edition. , New York, NY: Cambridge University Press; 1987.

[71] Dekovic M, Gerris JRM. Developmental analysis of social cognitive and behavioral differences between popular and rejected children. J Appl Dev Psychol 1994;15:367–86.