



# Valuation of peers' safe choices is associated with substance-naïveté in adolescents

Dongil Chung<sup>a,b,1</sup>, Mark A. Orloff<sup>a,c,1</sup> , Nina Lauharatanahirun<sup>a,d,2</sup>, Pearl H. Chiu<sup>a,c,d,3</sup> , and Brooks King-Casas<sup>a,c,d,3</sup>

<sup>a</sup>Fralin Biomedical Research Institute at VTC, Virginia Tech, Roanoke, VA 24016; <sup>b</sup>Department of Biomedical Engineering, Ulsan National Institute of Science and Technology, Ulsan 44919, South Korea; <sup>c</sup>Translational Biology, Medicine, and Health Graduate Program, Virginia Tech, Roanoke, VA 24016; and <sup>d</sup>Department of Psychology, Virginia Tech, Blacksburg, VA 24061

Edited by Eva H. Telzer, University of North Carolina, Chapel Hill, NC, and accepted by Editorial Board Member Susan A. Gelman September 28, 2020 (received for review December 4, 2019)

**Social influences on decision-making are particularly pronounced during adolescence and have both protective and detrimental effects. To evaluate how responsiveness to social signals may be linked to substance use in adolescents, we used functional neuroimaging and a gambling task in which adolescents who have and have not used substances (substance-exposed and substance-naïve, respectively) made choices alone and after observing peers' decisions. Using quantitative model-based analyses, we identify behavioral and neural evidence that observing others' safe choices increases the subjective value and selection of safe options for substance-naïve relative to substance-exposed adolescents. Moreover, the effects of observing others' risky choices do not vary by substance exposure. These results provide neurobehavioral evidence for a role of positive peers (here, those who make safer choices) in guiding adolescent real-world risky decision-making.**

adolescent | peer influence | decision-making | social influence | substance use

Adolescence is marked by an increased desire for social acceptance and sensitivity to social influences that can both promote and compromise health-related behaviors (1, 2). During this developmental period, substance use and other health-risk behaviors proliferate (3, 4), particularly among those who associate with peers who engage in those behaviors and less so for adolescents with less deviant peers (5–10). These data suggest that social influence can be bidirectional (1, 11–14), contributing to riskier or safer choices, and that adolescents' responsiveness to riskier or safer peers contributes to engagement in health-risk or health-promoting behaviors (15). To examine this possibility, we used a neurocomputational approach to examine how responsiveness to bidirectional social influence is associated with adolescents' experience with real-world substance use. Our results provide neural and behavioral evidence for a role of positive social peers in influencing decision-making in adolescents.

To examine how peers' choices impact adolescents' decision-making about risky options, we used functional neuroimaging and a gambling task in which adolescents made a series of choices between two gambles (one "safer" with smaller payoff variance and one "riskier" with greater payoff variance), both alone (Solo trials) and after observing others' choices (Info trials) (11). We recruited participants from 15 to 17 y old, as this age range is 1) associated with a developmental period of heightened neural sensitivity to appetitive (16) and social (17, 18) stimuli and is 2) a peak period of substance use initiation among adolescents later admitted for treatment (19). More generally, substance exposure during adolescence is a strong predictor of future health-risk behaviors and poor outcomes including risky sexual behaviors and substance use disorders, as well as diminished educational attainment, criminal activity, and development of psychopathology, after accounting for other predictors including history of conduct problems (19–26).

Adolescents were instructed in groups of up to six members, and informed that on Info trials, the choices of two other

randomly selected players from the group would be revealed (see Table 1 and *Materials and Methods* for group composition details). Group members did not know each other, and adolescents reported group members to be within less than a year of their own age (perceived peer age [mean ± SD]: 0.64 ± 2.05 y from self). Seventy-eight adolescents (ages 15 to 17; *n* = 41 female) were included in the behavioral portion of this study; a subset of these adolescents (*n* = 31) performed the task during functional neuroimaging. During the task, all adolescents played in the third-player position, such that on Info trials, two other players' choices were presented before adolescents made their own decisions (Fig. 1A; also see *SI Appendix*, Fig. S1). Adolescents' choices were thus measured under variants of social influence: safe influence, in which the two presented choices of others were the safer gamble, risky influence, in which the two others' presented choices were the riskier gamble, and mixed influence, in which the two others' presented choices comprised one safer and one riskier gamble. Info and Solo trials were intermixed. Participants were paid at the end of the task based on the outcome of a gamble randomly drawn from among their choices,

## Significance

During adolescence, substance use and other health-risk behaviors emerge, particularly among those who associate with peers engaging in such behaviors and less so for adolescents with less deviant peers. Here, we provide behavioral and neural evidence for a beneficial role of safer peers, rather than a detrimental influence of risky peers, in guiding adolescents' choices and substance use. The extent to which adolescents value peers' safe choices predicted substance-naïveté even after controlling for other factors associated with substance use, while valuation of peers' risky choices was unrelated to substance use. Whereas previous studies have largely examined associations between negative peers and increased health-risk behaviors, our data support a significant role of positive social peers for favorably influencing health-risk behaviors.

Author contributions: D.C., N.L., P.H.C., and B.K.-C. designed research; D.C., M.A.O., and N.L. performed research; D.C. and M.A.O. analyzed data; and D.C., M.A.O., N.L., P.H.C., and B.K.-C. discussed the data and wrote the paper.

The authors declare no competing interest.

This article is a PNAS Direct Submission. E.H.T. is a guest editor invited by the Editorial Board.

This open access article is distributed under [Creative Commons Attribution-NonCommercial-NoDerivatives License 4.0 \(CC BY-NC-ND\)](https://creativecommons.org/licenses/by-nc-nd/4.0/).

<sup>1</sup>D.C. and M.A.O. contributed equally to this work.

<sup>2</sup>Present address: Department of Biomedical Engineering and Department of Biobehavioral Health, Pennsylvania State University, University Park, PA 16802.

<sup>3</sup>To whom correspondence may be addressed. Email: [chiup@vtc.vt.edu](mailto:chiup@vtc.vt.edu) or [bkcasas@vtc.vt.edu](mailto:bkcasas@vtc.vt.edu).

This article contains supporting information online at <https://www.pnas.org/lookup/suppl/doi:10.1073/pnas.191911117/-DCSupplemental>.

**Table 1. Participant characteristics**

	Substance-naïve (n = 46)	Substance-exposed (n = 32)
Male/female participants <sup>†</sup>	25/21	12/20
Age <sup>†</sup>	15.76 ± 0.79	16.09 ± 0.78
Race (% white) <sup>†,‡</sup>	71.74	87.50
Family income level <sup>†,§</sup>	5.24 ± 0.99	4.48 ± 1.42
Highest parental education <sup>†,¶</sup>	4.35 ± 1.21	4.03 ± 1.22
Peers' perceived age <sup>†,#</sup>	16.28 ± 1.88	17.28 ± 3.05
BIS attention <sup>**</sup>	10.04 ± 2.80	12.09 ± 2.87
BIS motor <sup>***</sup>	19.41 ± 3.23	23.28 ± 4.62
BIS nonplanning <sup>*</sup>	21.96 ± 4.53	24.19 ± 4.82
ARQ antisocial	3.89 ± 2.45	4.06 ± 2.38
ARQ rebellious <sup>***</sup>	1.41 ± 1.28	4.97 ± 3.40
ARQ reckless	0.98 ± 1.48	1.34 ± 1.43
ARQ thrill seeking	6.54 ± 3.16	6.31 ± 3.91
YSR ADHD	54.35 ± 5.73	55.50 ± 6.62
YSR CD	53.46 ± 5.12	54.28 ± 6.11
YSR ODD	52.70 ± 4.02	54.53 ± 6.15
% used each drug (alc/mar/tob) <sup>  </sup>	—	90.63%/50.00%/31.25%
% binge drinking <sup>††</sup> in past 6 mo <sup>††</sup>	—	34.48%
% using marijuana ≥ 3× per month <sup>††</sup>	—	25.00%
% using tobacco ≥ 3× per month <sup>††</sup>	—	10.00%
Frequency of alcohol use <sup>**,\$§</sup>	—	1.71 ± 0.80
Frequency of marijuana use <sup>**,\$§</sup>	—	2.31 ± 1.20
Frequency of tobacco use <sup>**,\$§</sup>	—	1.36 ± 0.67
No. of substances used	—	1.88 ± 1.01
Age at earliest substance use	—	14.02 ± 1.87

\* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ . Means ± SDs are reported.

<sup>†</sup>Groups did not differ using  $t$  test or Fisher's exact test, as appropriate (all  $P > 0.14$ ).

<sup>‡</sup>Two participants in the substance-naïve group and one participant from the substance-exposed group identified as Hispanic.

<sup>§</sup>Average household annual income, where 1 = <\$20,000, 2 = \$20,000 to 35,000, 3 = \$35,000 to 50,000, 4 = \$50,000 to 75,000, 5 = \$75,000 to 100,000, and 6 = >\$100,000.

<sup>¶</sup>Highest parental education; where 1 = some high school, 2 = high school diploma or GED, 3 = some college or associate's degree, 4 = bachelor's degree, 5 = master's degree, and 6 = MD/ID/PhD.

<sup>#</sup>Age of peers in task instruction group, as perceived by adolescent participants.

<sup>||</sup>Five adolescents reported using an additional substance aside from alcohol, tobacco, or marijuana.

<sup>††</sup>Having more than five drinks at one time.

<sup>\*\*</sup>Among adolescents who reported use of indicated substance.

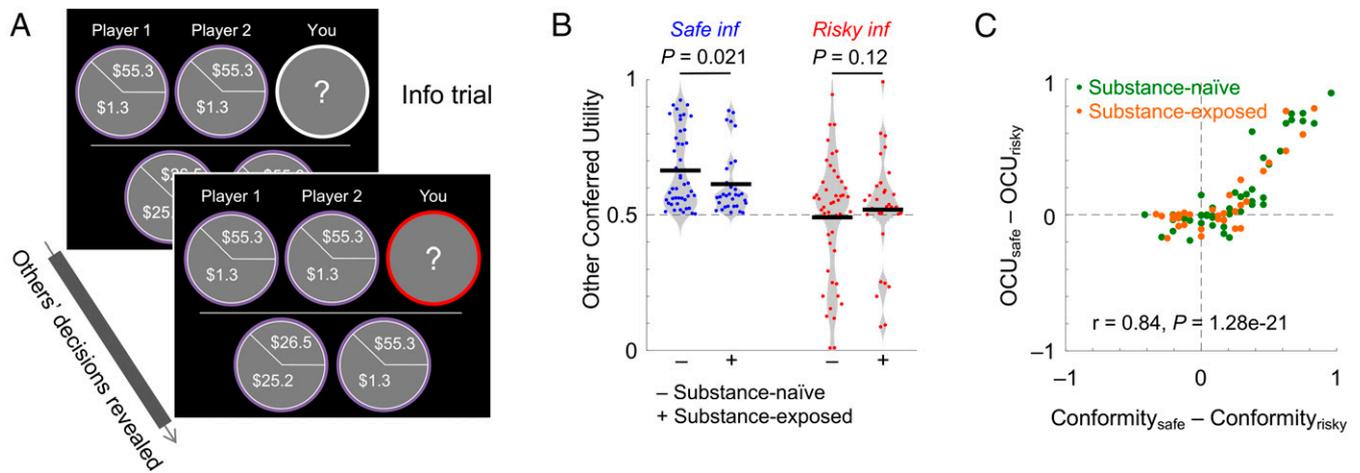
<sup>§§</sup>1 = tried once or twice, 2 = used three to five times, 3 = usually use a few times a month, 4 = usually use a few times a week.

independent from any other players' choices (see *Materials and Methods* for additional procedural details).

Substance exposure was defined based on real-world substance use: adolescents who reported having used alcohol, tobacco, marijuana, or any other illicit drugs (substance-exposed), and adolescents who had never tried these substances (substance-naïve; see *Materials and Methods* and Table 1 for additional participant characteristics). Age, sex, race, family income level, parental education, and perceived age did not differ between substance-exposed and substance-naïve participants (Table 1). The Barratt Impulsiveness Scale (BIS; 27), the Adolescent Risk-Taking Questionnaire (ARQ; 28), and Youth Self Report (YSR; 29) Diagnostic and Statistical Manual of Mental Disorders subscales were used to assess impulsivity, health-risk behaviors, and psychopathology as potential predictors of substance exposure (Table 1), as each has been previously associated with liability for poor outcomes including substance use disorders (30–32). Each of these variables was included as a covariate along with the neural and behavioral social influence variables of interest within a series of stepwise multiple logistic regressions predicting substance exposure (see the logistic regression section of the *Materials and Methods* for details). As described below, a quantitative model adapted from our prior work (11) was used to isolate the impact of observing others' safe and risky choices, respectively, on participants' choices about risky

options; these respective effects were in turn tested for their association with substance exposure among adolescents.

Substantial experimental evidence suggests that human decision-making is guided by the subjective value of information (33) and that decisions about, among, and for others requires the integration of subjective values derived from social and nonsocial information (11) [see Ruff and Fehr (34) for a review]. In the present study, we drew upon this framework and examined adolescents' subjective valuation of riskier and safer choices made by peers and the extent to which this valuation differs between substance-naïve and substance-exposed adolescents. In particular, we adapted previous work showing that during decision-making among social others, others' choices confer utility to those options, represented as other-conferred utilities (OCUs) that reflect utility increases (or decreases) on the options chosen by social others. Critically, these OCUs predict choices under social influence such that individuals who show greater subjective valuation (measured as behavioral and neural substrates of OCU) of social others' choices show greater conformity with those others' choices (35). The basic OCU model is an extension of expected utility models (36) and comprises OCU added to the gamble chosen by others, leading to an OCU-modified utility ( $U_{\text{with OCU}} = U_{\text{Solo}} + \text{OCU}$ ; see *Materials and Methods* for model details). Larger OCUs thus reflect greater subjective valuation of



**Fig. 1.** Substance-naïveté is associated with greater valuation of peers' safe choices. (A) Adolescents made a series of choices between two gambles (one safe and one risky). Per Chung et al. (11), the decisions were made alone (Solo trials) and after observing peers' choices (Info trials). On Info trials, two peers' decisions were revealed prior to the participant's decision. (B) OCU (utilities added to the gambles chosen by peers) under safe peer influence ( $OCU_{safe}$ ), but not risky influence ( $OCU_{risky}$ ) were significantly associated with substance exposure. In addition, Bayesian comparison shows decisive evidence in favor of a logistic regression model that includes  $OCU_{safe}$  over one that includes  $OCU_{risky}$  ( $BF = 9.24$ ), indicating that valuation of others' safe choices is more strongly associated with substance exposure than is valuation of others' risky choices (see Table 2 and Results for model comparison). (C) Differences between  $OCU_{safe}$  and  $OCU_{risky}$  were significantly correlated with model-agnostic conformity choices under safe and risky social influence (i.e., likelihood of making the same choice as peers; Pearson's correlation; substance-naïve:  $r = 0.85, P = 8.3e-14$ ; substance-exposed:  $r = 0.81, P = 2.0e-08$ ). Each point represents an individual participant; group means are indicated in black. Gray shades show the distribution of data points along the y axis.

social others' choices and are associated with higher likelihoods of making the same choices as social others, above and beyond what an individual's risk preference ( $\alpha$ , where  $U_{Solo} = \sum p_i(v_i)^\alpha$  for a gamble that has an  $i^{th}$  outcome  $v_i$  with probability  $p_i$ ) would predict. Here, to examine the impact of bidirectional social influence on adolescents' choices, we included two OCUs to identify the distinct impact of safer and riskier choices, respectively, by peers: The model term  $OCU_{safe}$  reflects subjective valuation of others' safe choices, and  $OCU_{risky}$  reflects subjective valuation of others' risky choices (see Materials and Methods for model description details and SI Appendix for comparison and model parameter recovery details).

## Results

To first evaluate the robustness of the model parameter estimates derived from participants' choices (i.e., true parameter values), we used means and SDs of the group-level distributions to simulate individual choice data for 78 simulated subjects, and re-estimated parameter values from these simulated data (i.e., recovered parameter values). All parameters (inverse temperature  $[\lambda]$ , risk preference  $[\alpha]$ , utility conferred by safe and risky others [ $OCU_{safe}$  and  $OCU_{risky}$ , respectively]) were recoverable and showed high correlations between true and recovered parameter values, including  $OCU_{safe}$  ( $r = 0.82, P = 3.2e-20$ ) and  $OCU_{risky}$  ( $r = 0.82, P = 4.1e-20$ ; see Materials and Methods for other parameter details and SI Appendix for model recovery details).

Upon verifying the robustness of the OCU parameter estimates, we tested whether substance exposure was significantly associated with the utilities conferred by safe and risky social influence ( $OCU_{safe}$  and  $OCU_{risky}$ ) using stepwise multiple logistic regression (see logistic regression section in Materials and Methods) predicting substance exposure. This analysis showed that  $OCU_{safe}$  (odds-ratio [OR] = 0.0032, 95% CI = [1.7e-05, 0.33],  $P = 0.021$ ), but not  $OCU_{risky}$  (OR = 0.074, 95% CI = [0.0024, 1.72],  $P = 0.12$ ) significantly predicts substance exposure (Fig. 1B and Table 2). Further, Bayesian model comparison between the logistic regression model including  $OCU_{safe}$ , but not  $OCU_{risky}$  to the model including  $OCU_{risky}$ , but not  $OCU_{safe}$  demonstrates substantial evidence in favor of the model with

$OCU_{safe}$  (Bayes factors [BF] = 9.24). These model-based results were paralleled by model-agnostic data (SI Appendix, Figs. S2 and S3) showing increased modulation of decisions when observing peers' safe choices in substance-naïve adolescents and decreased modulation in substance-exposed adolescents. These data provide model-based and model-agnostic evidence showing that during adolescent decision-making, varying responses to peers' safe choices, and not to peers' risky choices, is associated with substance exposure.

As an additional model-agnostic measure of choices under social influence, we computed the proportion of trials in which adolescents chose the same option as others. This conformity metric was calculated separately for choices under safe and risky social influence for each participant and showed a pattern consistent with the model-based OCU parameters, such that adolescents who followed others' safe choices more than risky choices showed larger  $OCU_{safe}$  than  $OCU_{risky}$ , and vice versa (Fig. 1C).

We posited that at least two neurodevelopmental processes may contribute to the varying effects of peers' safe choices as related to substance exposure in adolescents. Specifically, during adolescence, changes occur in both neural valuation systems and neural systems sensitive to other social processes (37, 38) (for review, see (39, 40)); thus, differences in neural instantiation of social valuation and/or nonvaluation social processes may be associated with differing effects of peers on decision-making

**Table 2. Logistic regression of behavioral OCU parameters predicting substance exposure**

Regressor	OR	CI (95%)	P value
Intercept	0.078	[0.0011, 3.86]	0.21
BIS motor	1.40	[1.19, 1.72]	0.00028***
$OCU_{safe}$	0.0032	[1.7e-05, 0.33]	0.021*
$OCU_{risky}$	0.074	[0.0024, 1.72]	0.12

$OCU_{safe}$  is associated with decreased likelihood of substance exposure. No association was found between  $OCU_{risky}$  and substance exposure. \* $P < 0.05$ , \*\*\* $P < 0.001$ .

about risky options. Extant data identifying separable roles of ventromedial prefrontal cortex (vmPFC) in social valuation (41) and dorsomedial prefrontal cortex (dmPFC) in nonvaluation social processing (37, 42, 43) allow us to differentiate between these possibilities. To test these neural hypotheses, a subset of  $n = 31$  adolescents was scanned during task performance, and we performed event-related functional MRI (fMRI) analyses of adolescents' blood-oxygen-level dependent (BOLD) responses at the time at which they viewed others' choices. Region-of-interest (ROI) selection was carried out using a Neurosynth-based meta-analytic approach to identify regions most likely to represent the processes of interest (i.e., social valuation and nonvaluation social processing; see ROI analyses section in *Materials and Methods* for further details).

To evaluate potential neural differences in valuation of peers' safe and risky choices (as identified in the model-based and model-agnostic behavioral data reported above), we examined BOLD responses representing other-conferred utility (see *Materials and Methods* for contrast details) under safe and risky social influence, respectively, in the adolescents who were scanned during task performance. Stepwise multiple logistic regression revealed that social valuation responses in vmPFC were associated with substance exposure only for OCU<sub>safe</sub> and not OCU<sub>risky</sub> (Fig. 2A), after accounting for effects of impulsivity and psychopathology (Table 3). In addition, Bayesian comparison of a regression model excluding OCU<sub>safe</sub> to a model excluding OCU<sub>risky</sub> showed decisive evidence in favor of the model that includes OCU<sub>safe</sub> (BF = 278.66), indicating that neural valuation of peers' safe choices is more strongly associated with substance exposure than is neural valuation of peers' risky choices. As expected, vmPFC activity was also related to subjective value of gamble options as a whole across all participants (*SI Appendix, Fig. S4*).

Next, to evaluate whether nonvaluation social processing is associated with substance exposure, we compared social (Info) trials to Solo trials, in which no social information was available, during the gamble viewing phase. Stepwise multiple logistic regression revealed that BOLD responses in the dmPFC to social versus nonsocial information was not related to substance exposure for either safe or risky peer choices (Fig. 2B and Table 4), indicating that the extent to which adolescents are responsive to nonvaluation social information is not related to substance exposure. For completeness, we compared eight other nonvaluation social processing regions (see ROI analyses section in *Materials and Methods* for details) between groups; none of these additional ROIs evidenced a relationship between neural

**Table 3. Logistic regression of neural response to OCU predicting substance exposure**

Regressor	OR	CI (95%)	P value
Intercept	4.9e-23	[1.4e-50, 2.1e-08]	0.025*
BIS motor	3.31	[1.48, 13.53]	0.026*
YSR ODD	1.69	[1.14, 3.50]	0.047*
OCU <sub>safe</sub> (neural)	0.0086	[3.0e-05, 0.21]	0.027*
OCU <sub>risky</sub> (neural)	0.13	[0.0023, 1.28]	0.16

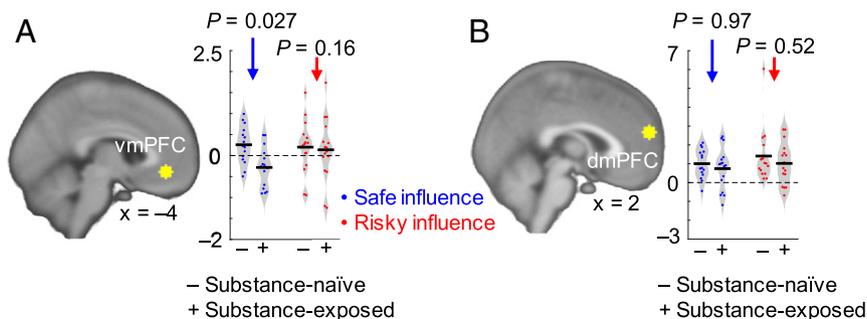
Neural response to OCU<sub>safe</sub> is associated with decreased likelihood of substance exposure. Neural response to OCU<sub>risky</sub> is not associated with substance exposure. \* $P < 0.05$ .

responses to social Info versus Solo trials and substance exposure (*SI Appendix, Table S1*). As expected, dmPFC activity was observed for social versus nonsocial trials as a whole across participants (*SI Appendix, Fig. S5*).

Finally, as previous studies indicate that functional connectivity between socio-affective and valuation neural circuits is sensitive to developmental changes and increases in adolescence (37, 38), we further examined the possibility that the substance-naïve and substance-exposed adolescents differ in such functional connectivity. Specifically, we conducted a psychophysiological interaction (PPI) analysis testing whether the interaction between dmPFC-vmPFC connectivity and the psychological factor of conformity (i.e., decisions to follow others' choices or not) is affected by substance exposure (see *SI Appendix* for PPI design matrix details). The PPI was comparable between substance-naïve and substance-exposed adolescents, providing little support for an association between substance exposure and functional connectivity between socio-affective and valuation circuitry (see *SI Appendix, Fig. S6* for further discussion of PPI analyses).

## Discussion

Here, using a computational model-based approach, we show that substance-naïveté in adolescents is related to increased valuation of peers' safer choices. Furthermore, we evaluate three neurodevelopmental hypotheses linking sensitivity to social influence to increased risky behavior in adolescents. These data provide converging evidence that the varying influence of safe peers on adolescents' choices is attributable to differential valuation of peers' safe choices, rather than valuation of peers' risky choices, general sensitivity to social information, or enhanced connectivity of valuation and socio-affective neural circuitry.



**Fig. 2.** Neural valuation of peers' safe choices (OCU<sub>safe</sub>) is associated with substance-naïveté. (A) Social valuation: In vmPFC, responses to OCU<sub>safe</sub> but not to OCU<sub>risky</sub> were significantly associated with substance exposure (OCU<sub>safe</sub>, OR = 0.0086, 95% CI for OR: [3.0e-05, 0.21],  $P = 0.027$ ; OCU<sub>risky</sub>, OR = 0.13, 95% CI for OR: [0.0023, 1.28],  $P = 0.16$ ). In addition, Bayesian comparison shows decisive evidence in favor of a logistic regression model that includes neural responses to OCU<sub>safe</sub> over that including OCU<sub>risky</sub> (BF = 278.66), indicating that neural valuation of others' safe choices is more strongly associated with substance exposure than is neural valuation of others' risky choices (see *Results* for model comparison details). (B) Nonvaluation social processing: In dmPFC, responses to social information were not associated with substance exposure (for Social versus Solo trials; safe info: OR = 0.97, 95% CI for OR: [0.17, 5.30],  $P = 0.97$ ; risky info: OR = 0.61, 95% CI for OR: [0.11, 2.14],  $P = 0.52$ ).

**Table 4. Logistic regression of neural response to nonvaluation social processing predicting substance exposure**

Regressor	OR	CI (95%)	P value
Intercept	3.3e-09	[1.4e-18, 0.0087]	0.031*
BIS motor	1.67	[1.19, 2.83]	0.017*
YSR ODD	1.20	[0.96, 1.66]	0.15
Safe social (neural)	0.97	[0.17, 5.30]	0.97
Risky social (neural)	0.61	[0.11, 2.14]	0.52

Neither safe nor risky nonvaluation social processing is associated with likelihood of substance exposure. \* $P < 0.05$ .

The association between subjective valuation of peers' safe choices and substance exposure provides evidence of a bias in social valuation that plays a role in engagement (or not) in substance use. This has implications for public health and potential early intervention approaches, given the myriad poor outcomes associated with initiating substance use in adolescence (19–26). While previous studies have largely focused on variables contributing to health-risk behaviors (44–48), peer effects that facilitate health-promoting behaviors may be important when aiming to reduce deviant behaviors. More generally, biases in social influence sensitivity may interact with risk preferences and risk assortment, and amplify the effects of peers on adolescents' decision-making. Individuals with similar risk preferences have been found to positively assort (49, 50), and following this, adolescents whose choices are most influenced by safe peers (e.g., substance-naïve adolescents) may also be more likely to interact with individuals who make safe choices. Previous studies have reported that individuals show higher neural and behavioral reward responses and are more generous when rewards are shared with friends or those at close social distance (51, 52). In the present work, we sought to minimize social network effects by recruiting adolescents who did not know each other, and who were thus equidistant from one another with regard to degree of objective social distance. However, substance-naïve adolescents may nevertheless perceive greater closeness to peers who make safe choices; such perceptions may contribute to the greater reward response to others' safe choices in substance-naïve, relative to substance-exposed, adolescents. More generally, the nature of social relationships and composition of social groups are likely to affect the influence of peers on decision-making and should be examined in future research of social effects on health-risk behaviors.

Our stepwise multiple logistic regression approach allowed an examination of the rich dimensionality that may contribute to risk liability for substance use in adolescents. As expected from previous reports (53), impulsivity (for the behavioral and neural OCU regression models) and psychopathology (for the neural OCU model) covaried with adolescent substance exposure. Both behavioral and neural indicators of sensitivity to safe peers were significantly and consistently associated with substance exposure after accounting for impulsivity and psychopathology, indicating that social valuation of safe others, but not risky others, is associated with substance exposure above and beyond other common predictors of health-risk behaviors. We also note that, based on previous reports, we expected more apparent associations between adolescent psychopathology and substance exposure (54). However, aside from the oppositional defiant disorder (ODD) factor in the vmPFC model, psychopathology indices did not explain adolescents' substance use behaviors. This lack of association could be due to our sample excluding medicated adolescents, leading to less psychopathology than previous reports that detected relationships with substance use. That is, the vast majority of adolescents were below clinical threshold (29) for any YSR externalizing disorders. Finally, in our sample, age

was not related to adolescent substance exposure. This is likely due to the restricted age range we examined (15 to 17 y; again chosen based on previous reports that this is a particularly sensitive developmental period (16–18)).

Sensitivity to peers' risky choices was not related to adolescent substance exposure. One possible explanation for this lack of association could be a ceiling effect where substance-exposed adolescents were already making a large proportion of risky decisions outside of viewing peers' choices (i.e., on Solo trials). However, this explanation is unlikely; substance-exposed adolescents made safe choices on 57% of Solo trials on average, and were thus not at ceiling with regard to risky choices (*SI Appendix, Fig. S3*). Another possibility is that for substance-naïve adolescents, following peers' risky choices may occur via factors not assessed by our model. For example, prior data suggest that adolescents who are perceived as more popular engage more often in risky behaviors, such as substance use (55). Thus, in the face of peers' risky choices, substance-naïve adolescents may be additionally influenced by considerations of social status (5, 10, 56). Future work will be important to understand the observed asymmetry in influence of peers' safe and risky choices on adolescent decisions.

It remains to be seen whether the variation in responses to safe versus risky information is specific to the social nature of the information, or whether the effects extend to decision-related information from nonsocial sources. While our previous work in adults demonstrated other-conferred utility to be observed in social contexts only (11), it is nevertheless important that future work evaluate whether influences of risk-related information on adolescent decision-making as related to health-risk behaviors is restricted to social contexts, or whether the influence is also observed in responses to nonsocial information. In particular, implications of this work for information-based intervention strategies in adolescents may be informed by the nature of the information and the context in which it is delivered.

This work broadly points to the importance of positive peers (here, those who make safer decisions) in influencing adolescent choices. The data provide a critical starting point for future studies in adolescents examining whether reactions to positive peers may confer a protective effect against substance exposure or be a risk factor for substance use, or both. These possibilities may be explored with longitudinal and/or neuromodulation studies (e.g., transcranial magnetic stimulation, real-time fMRI neurofeedback) examining links between valuation of peers' choices and real-world outcomes across development. Further, our substance-exposed adolescents comprise individuals with a range of substance use behaviors from those who have tried a substance once to those who reported frequent use of multiple substances (adolescents range from not using alcohol, tobacco, or marijuana at all to using alcohol or tobacco a few times a month, and marijuana a few times a week). Future studies should examine whether frequency, quantity, or length of substance use, or other use characteristics, may be related to sensitivity to peers' choices in adolescence.

Our neuroimaging data provide additional insight about the processes via which peers influence adolescent decision-making. The vmPFC plays a key role in representing social and nonsocial subjective values (11, 57–61), and here, adolescents' substance-naïveté was associated with vmPFC encoding of the subjective value of others' safe, and not risky, choices. The specificity of the neural findings to valuation within vmPFC (and not general nonvaluation social processing regions or connectivity among valuation and socio-affective regions) further supports biases in other-conferred utility that guide participants' choices under social influence (11). These patterns are consistent with the idea that substance-naïve adolescents follow others' safe choices more than substance-exposed adolescents do, because they value others' safe choices more. In sum, these data contribute to

understanding how peers influence decision-making and find that valuation of peers' positive (here, safer) choices, and not peers' riskier choices is associated with substance exposure. More broadly, this work points to the importance of positive peers in influencing adolescent real-world behaviors.

## Materials and Methods

**Participants.** Ninety-one community adolescents (male/female = 45/46, age =  $15.90 \pm 0.80$ ) participated in the current study. All adolescents provided written informed assent, and legal guardians provided written informed consent for the adolescents' participation in a research protocol approved by the Institutional Review Board of Virginia Tech. Participants' substance use history was assessed with the Youth Risk Behavior Survey (YRBS) (62). Exclusion criteria included current psychiatric medications, previous head injuries resulting in loss of consciousness, neurological disorders, and MRI contraindications. A priori data analytic exclusion criteria included behavioral patterns that indicated lack of attention or understanding of the task (i.e., choosing the option with a larger high payoff value less often when it was more likely to return the high payoff), individuals for whom individual-level models did not arrive at a unique solution using exploratory maximum likelihood estimation (MLE) (see Computational model), MRI-related artifact, and excessive movement during the functional scanning run ( $>5$  mm in the x, y, or z direction); these factors would hinder a meaningful analysis of the social, decision-making, and neural effects of interest.

After exclusions, the analyzed behavioral data included 78 adolescents: 46 adolescents who had never tried alcohol, tobacco, marijuana, or any other drugs (substance-naïve; male/female = 25/21, age =  $15.76 \pm 0.79$ ) and 32 adolescents who had tried one or more of these substances (substance-exposed; male/female = 12/20, age =  $16.09 \pm 0.78$ ). Substance-naïve and substance-exposed adolescents were of comparable age (Fisher's exact,  $P = 0.17$ ), gender (Fisher's exact,  $P = 0.17$ ), family income level (Fisher's exact,  $P = 0.14$ ), parental education (Fisher's exact,  $P = 0.52$ ), perception of social others' age ( $t(37) = 1.24$ ,  $P = 0.22$ , see *SI Appendix* for additional exclusion criteria for this analysis), and racial distribution (Fisher's exact,  $P = 0.16$ ). See Table 1 for additional participant characteristics. Of these 78 adolescents, 31 participated in functional neuroimaging as described below.

**Experimental Procedures.** Following our previous work (11), we used a decision-making task in which adolescents made a series of choices between one safer and one riskier gamble; as detailed below, decisions were made either alone or after viewing the choices of peers. Each pair of gambles had the same chance of winning high and low payoffs, and the payoff probabilities were shown proportional to the size of the pie pieces (Fig. 1A). As per Holt and Laury (63), the safer (safe) gamble always had a smaller variance between high and low payoffs compared to the riskier (risky) gamble. Decisions were made either after observing peers' choices or alone (11). Specifically, participants were instructed that on some indicated trials, choices made by a subset of the group would be shown (Info trials), and that on other trials, decisions made by others would not be revealed (Solo trials).

Instructions were presented to groups of four, five, or six, and participants were told that two other players' choices would be selected to be presented on a subset of trials. To enhance the social nature of the group-instruction environment, 26 additional adolescents and young adults were recruited (unbeknownst to participants) to be present only during the instruction phase; these individuals did not participate in the study beyond being present for the task instructions (no effects of peers' perceived age on influence were observed; see details in *SI Appendix*). Participants were instructed that the order of decision-making would be presented on the computer screen or in the scanner at the beginning of the task, and that position would remain the same throughout the task. All adolescents were assigned to the third-player position, such that they would observe two other players' choices on the Info trials. After the instructions were completed, all participants were given the opportunity to ask questions about the task and a brief quiz was administered to assess task comprehension; any incorrect quiz answers were addressed at that time. Participants were instructed that they would be escorted one at a time to separate rooms for the task. Adolescents were escorted first to the scanner suite or behavioral testing room, and then the instruction-only participants were called one at a time, compensated, and released.

To investigate the influence of observing others' choices on decision-making about uncertain options, four different trial types, based on the combination of the two peers' choices that were presented, were intermixed and pseudorandomly presented. Specifically, 1) Solo trials were defined as trials in which participants made their choices alone, without information

about peers' choices, 2) Info: 'safe, safe' were trials in which the two peers' displayed choices were the safe gamble, 3) Info: 'risky, risky' were trials in which the two peers' displayed choices were the risky gamble, and 4) Info: 'mix' were trials in which the two peers' displayed choices comprised one safe and one risky gamble. Each participant made 96 choices (four lottery menus  $\times$  six payoff probabilities  $\times$  four trial types), where four lottery menus were randomly selected from eight unique lottery menus adapted from Holt and Laury (63) and the six probabilities of high payoff were 40%, 50%, 60%, 70%, 80%, and 90%. See Chung et al. (11) for the entire set of eight menus and task development details.

We have previously shown that the task used herein captures a social process (as opposed to a more general information/affective effect). Specifically, in Chung et al. (11), we implemented a separate behavioral experiment explicitly instructing participants ( $n = 30$ ) that Info trials were computer-generated choices wherein, prior to the participant's decision, two computers would randomly pick among the options, and these two options would be presented (Computer Info trials). The visual aspects and trial structure of the original social game were maintained, and no effect of Computer Info selections on participants' choices was observed (*SI Appendix, Fig. S7*).

Participants were paid at the end of the study. To avoid potential effects of gamble outcomes in decision-making, only the outcome of a single lottery selected from all choices made by the participant was carried out. It was emphasized before the task that other players' choices would not affect participants' payoff, and that participants' payoff solely depended on their own behavioral choices.

**Questionnaire Measures.** The BIS (27), the ARQ (28), and the YSR (29) were administered, as these measures have previously been shown to be associated with substance use behaviors (30–32). We specifically used subscales from these measures in a series of multiple stepwise logistic regression models to examine potential alternative predictors of substance exposure (aside from our task-derived behavioral measures of sensitivity to peer influence and neural measures of social valuation and nonvaluation social processing). Particularly, we focused on the second order subscales of the BIS (attention, motor, and nonplanning), the three ARQ subscales not querying substance use (antisocial, reckless, and thrill seeking), and the externalizing YSR Diagnostic and Statistical Manual of Mental Disorders (DSM) subscales (attention deficit hyperactivity disorder [ADHD], ODD, and conduct disorder [CD]) (Table 1).

**Logistic Regression.** We analyzed the relationships between our sensitivity to social influence measures and substance exposure using a stepwise multiple logistic regression framework. This approach allows us to examine these effects in the context of potential confounds and alternative explanations for substance exposure. We constructed three separate stepwise logistic regression models to predict substance exposure based on our primary hypothesized predictors of interest: 1) behavioral OCU parameters (OCU<sub>safe</sub> and OCU<sub>risky</sub>), 2) neural representation of social valuation in vmPFC (represented by responses to OCU<sub>safe</sub> and OCU<sub>risky</sub>), and 3) neural response to nonvaluation social processing in dmPFC (represented by responses on social vs. solo trials). Prior to running these models, the dimensionality of potential other predictors was reduced using an initial logistic regression predicting substance exposure with: age, sex, parental income, BIS (attention, motor, and nonplanning subscales), YSR DSM subscales associated with disinhibition (54) (ADHD and ODD), and the ARQ subscales (antisocial, reckless, and thrill-seeking subscales). The CD subscale of YSR was not included due to collinearity with the ODD subscale ( $r > 0.7$ ), and the rebellious subscale of the ARQ was excluded as it directly queries substance use. Covariates for each initial model were selected using alpha-to-remove of 0.2 ( $P > 0.2$ ) in backward stepwise regression to reduce the dimensionality of predictors. Then, separately for each model including social influence variables of interest, we added the social influence variables using a stepwise method to further refine the relevant model by removing the least-significant covariates until all covariates had  $P < 0.15$  (64). The final behavioral OCU model predicting substance exposure included the BIS motor subscale as a covariate. Final covariates for both neural models predicting substance exposure included the BIS motor subscale and the YSR ODD subscale. We obtained BFs to directly compare logistic regression models by first using the brms package to estimate the models in a Bayesian framework (65) and bayestestR to calculate the BF using these estimates (66).  $BF > 1$  indicates evidence for a particular model as compared to an alternative model, where the larger the BF, the more evidence for that model.

**Social Influence Model: Two-OCU Model.** To investigate the extent to which observing risky, safe, and mixed choices of peers affects adolescents' decision-making about risky options, we constructed an extension of our previously reported OCU model (11). The OCU model is an expected utility model (36)

that includes the addition of a subjective valuation of peers' choices. Here, we introduced two separate OCUs, each of which corresponds to an additional utility to the safe ( $OCU_{safe}$ ) and risky ( $OCU_{risky}$ ) gamble chosen by peers. As per Chung et al. (11), the OCU-modified utility on info trials ( $U_{with\ OCU}$ ) of each option and the probability of selecting the safe gamble were computed as follows:

$$U_{with\ OCU: safe} = P_{high} \times V_{high-payoff: safe}^{\alpha} + (1 - P_{high}) \times V_{low-payoff: safe}^{\alpha} + \delta('safe, safe') \times OCU_{safe} \quad [1]$$

$$U_{with\ OCU: risky} = P_{high} \times V_{high-payoff: risky}^{\alpha} + (1 - P_{high}) \times V_{low-payoff: risky}^{\alpha} + \delta('risky, risky') \times OCU_{risky} \quad [2]$$

$$P(\text{choosing safe gamble}) = [1 + \exp(-\lambda \times (U_{with\ OCU: safe} - U_{with\ OCU: risky}))]^{-1} \quad [3]$$

where  $U_{with\ OCU: safe}$  (or  $U_{with\ OCU: risky}$ ) is the OCU-modified utility of the safe (or risky) gamble,  $P_{high}$  is the probability of earning the high payoff,  $V$  represents a payoff for each gamble,  $\lambda$  indicates sensitivity to the difference between the utilities of the paired options,  $\alpha$  is a risk preference, and indicator  $\delta(\cdot) = 1$  if the others chose the indicated options on that trial (0, otherwise). The estimated risk preference  $\alpha$  indicates whether a participant is risk neutral ( $\alpha = 1$ ), risk seeking ( $\alpha > 1$ ), or risk averse ( $0 < \alpha < 1$ ).

For parameter comparison analyses to distinguish effects of OCUs on decision-making between trial types (Info: 'safe, safe' vs. Info: 'risky, risky') and between adolescent groups, we normalized each estimated OCU to a scale between 0 and 1:

$$OCU_{normalized} = [1 + \exp(-\lambda \times OCU_{raw})]^{-1} \quad [4]$$

where  $\lambda$  is that obtained from Eq. 3,  $OCU_{normalized} = 0.5$  indicates no influence from observing peers' choices,  $0.5 < OCU_{normalized} \leq 1$  indicates higher likelihood of choosing the same gamble as peers, and  $0 \leq OCU_{normalized} < 0.5$  indicates higher likelihood of choosing the gamble different from peers.

Model recovery and model comparison details are provided in the [SI Appendix](#).

**Parameter Estimation.** For model parameter estimation, all 96 trials per participant were used. We adopted a hierarchical Bayesian model structure of the population, such that all of the participants' model parameters ( $\lambda$ ,  $\alpha$ ,  $OCU_{safe}$ , and  $OCU_{risky}$ ) were taken as random effects, assumed as samples from common group-level parameter distributions (67, 68). For all parameters, the group-level distributions were Gaussian with free group-level mean ( $\mu$ ), SD ( $\sigma$ ), and a standard normal distribution ( $Normal(0, 1)$ ) following noncentered parameterization (69). For  $\lambda$  and  $\alpha$ , we applied an inverse probit transformation and multiplied the transformed value by a constant (50 for  $\lambda$  and 2 for  $\alpha$ ) to constrain the parameters between 0 and the multiplied constant. We estimated the hyperparameters (parameters of the group-level distributions;  $[\mu_{\lambda}, \sigma_{\lambda}, \mu_{\alpha}, \sigma_{\alpha}, \mu_{OCU: safe}, \sigma_{OCU: safe}, \mu_{OCU: risky}, \sigma_{OCU: risky}]$ ) using uninformative priors: the prior means  $\sim Normal(0, 10)$  and the prior SDs  $\sim Cauchy(0, 2.5)$  with lower bound of zero.

All observed behavioral choices from adolescents in each group were used for estimating the joint distribution of the parameters of the model. We used Markov chain Monte Carlo (MCMC) sampling with the No-U-Turn variant of the Hamiltonian Monte Carlo technique implemented in Stan (70) and its interface to R (69). A total of four chains were run where each chain had 5,000 samples drawn, discarding the first 2,000 samples for burn-in. We visually inspected the chains for convergence and good mixing, and confirmed all values of the potential scale reduction factor were less than 1.05 for all variables (71).

**fMRI Acquisition and Preprocessing.** Functional and structural brain scans were acquired on a 3.0-T Siemens Trio scanner. High-resolution T1 weighted structural images were acquired using the magnetization-prepared rapid gradient-echo sequence (Siemens) with the following parameters: repetition time (TR) = 1,200 ms, echo time (TE) = 2.66 ms, slices = 192, field of view = 245 × 245 mm, and voxel size: 1 × 1 × 1 mm<sup>3</sup>. Echo planar images were collected throughout the task procedure to measure BOLD signal. Scans were angled 30° from the anterior commissure–posterior commissure line. Scanner parameters were as follows: TR = 2,000 ms, TE = 30 ms, slices = 34, slice thickness = 4 mm, flip angle = 90°, voxel size: 3.4 × 3.4 × 4 mm<sup>3</sup>.

Preprocessing analyses included slice timing correction, motion correction, coregistration, normalization to the Montreal Neurological Institute template, and spatial smoothing using a 6-mm Gaussian kernel and were performed with Statistical Parametric Mapping (SPM) 12 (<https://www.fil.ion.ucl.ac.uk/spm/>). Functional images were resampled to 3 × 3 × 3 mm<sup>3</sup> voxels during normalization. Autocorrelation of the hemodynamic responses was modeled as a first-order autoregressive process and a high-pass filter of 1/128 Hz was applied to all scans.

**General Linear Model (GLM) Analyses.** We performed event-related fMRI analyses of adolescents' BOLD responses at the time at which they viewed other players' decisions. Two separate design matrices (DM1, DM2) were used to specify the extent to which each type of social information affected the utility signal (Fig. 2A). A separate regressor was included to censor each volume where framewise displacement (FD) was greater than 0.9 mm (72). Mean FD (after censoring) is 0.15 ± 0.082, range: 0.079 to 0.44 (substance-naïve: mean FD: 0.17 ± 0.090, range: 0.079 to 0.44; substance-exposed: mean FD: 0.14 ± 0.074, range: 0.084 to 0.37). The number of censored volumes per subject was 7.74 ± 15.64, range: 0 to 69 (substance-naïve: 9.07 ± 14.39, range: 0 to 52; substance-exposed: 6.50 ± 17.12, range: 0 to 69). Neither mean FD ( $P = 0.46$ ) nor number of censored volumes ( $P = 0.66$ ) differed with substance exposure.

In DM1, the task-related regressors were arranged as follows to assess neural signatures of  $OCU_{safe}$ :

- 1) *Player1Cue*: on Info trials, onset of the cue indicating Player 1's choices. At the same time, a new pair of gambles was revealed.
- 2) *InfoViewP1P2\_othersRisky*: on [Info: 'risky, risky'] trials, simultaneous revelation of both other players' decisions.
- 3) *InfoViewP1P2\_othersSafe\_conform*: on [Info: 'safe, safe'] trials, simultaneous revelation of both other players' decisions when the participant chose the SAME option after observing others' choices.
- 4) *InfoViewP1P2\_othersSafe\_notconform*: on [Info: 'safe, safe'] trials, simultaneous revelation of both other players' decisions when the participant chose the DIFFERENT option after observing others' choices.
- 5) *InfoViewP1P2\_othersMix\_conformMatch*: on [Info: 'mix'] trials, simultaneous revelation of both other players' decisions. Only the particular trials where the same pairs of gambles as on the trials included in the third regressor *InfoViewP1P2\_othersSafe\_conform* were modeled.
- 6) *InfoViewP1P2\_othersMix\_notconformMatch*: on [Info: 'mix'] trials, simultaneous revelation of both other players' decisions. Only the particular trials where the same pairs of gambles as on the trials included in the fourth regressor *InfoViewP1P2\_othersSafe\_notconform* were modeled.
- 7) *SoloViewGambles\_conformMatch*: on Solo trials, revelation of new pair of gambles. Only the particular trials where the same pairs of gambles as on the trials included in the third regressor *InfoViewP1P2\_othersSafe\_conform* were modeled.
- 8) *SoloViewGambles\_notconformMatch*: on Solo trials, revelation of new pair of gambles. Only the particular trials where the same pairs of gambles as on the trials included in the fourth regressor *InfoViewP1P2\_othersSafe\_notconform* were modeled.
- 9) *Keypress*: all trial key presses during the decision period.
- 10) *Review*: reviewing gamble choice that was made.

All regressors in DM1, except the regressors *Player1Cue*, *Keypress*, and *Review*, were modeled as 6-s events. *Review* was modeled as a 2-s event.

To identify neural responses specific to individuals'  $OCU_{safe}$ , we calculated a contrast between Info: 'safe, safe' trials and the "gamble-matched trials" (the same pairs of gambles as on the Info: 'safe, safe' trials, where participants conformed [or not]) that consisted of Solo and Info: 'mix' trials as follows:

$$conformMatch = 0.5(SoloViewGambles_{conformMatch}) + 0.5(InfoViewP1P2_{othersMix_{conformMatch}}) \quad [5]$$

$$notconformMatch = 0.5(SoloViewGambles_{notconformMatch}) + 0.5(InfoViewP1P2_{othersMix_{notconformMatch}}) \quad [6]$$

$$OCU_{safe} = [InfoViewP1P2_{othersSafe_{conformMatch}} + [notconformMatch - InfoViewP1P2_{othersSafe_{notconform}}]] \quad [7]$$

Given our expectation that the brain encodes utilities between the chosen and unchosen gambles and the definition of OCU-modified utilities (Eqs. 1 and 2), we separately modeled trials in which participants did and did not conform with the two other participants' displayed choices. By doing so, the

subjective neural responses to gamble information are cancelled, so that the contrast only leaves  $OCU_{safe}$  (Fig. 2A).

Neural responses to  $OCU_{risky}$  were assessed in DM2 in the same way as DM1; Info: 'safe, safe' trials and their gamble-matched trials were interchanged with Info: 'risky, risky' trials and the corresponding gamble-matched trials (the same pairs of gambles as on the Info: 'risky, risky' trials, where participants conformed [or not]) (Fig. 2A).

To examine neural substrates of general sensitivity to social information, we defined two different contrasts based on DM1 and DM2. Specifically, neural sensitivity to safe social information (Fig. 2B) was examined contrasting BOLD responses to regressors as follows:

$$\{([InfoViewP1P2\_othersSafe\_conform + InfoViewP1P2\_othersSafe\_notconform] - [SoloViewGambles.conformMatch + SoloViewGambles\_notconformMatch])\}$$

[8]

For neural sensitivity to risky social information (Fig. 2B), we used an equivalent contrast defined within DM2 replacing Info: 'safe, safe' trials with 'Info: 'risky, risky' trials.

At the first level, contrast images were generated for each participant that reflected whole-brain activity correlated with utility differences,  $OCU_{safe}$ ,  $OCU_{risky}$ , safe social information, and risky social information as described above. At the second level, stepwise multiple logistic regression was used to relate neural responses to  $OCU_{safe}$  (or  $OCU_{risky}$ ) to substance exposure (Fig. 2A). The association of neural nonvaluation safe (or risky) social processing to substance exposure was also assessed using logistic regression (Fig. 2B). The main finding that neural responses to  $OCU_{safe}$  but not  $OCU_{risky}$  are associated with substance exposure is robust to potential differences in trial numbers across subjects (SI Appendix, Fig. S8). Neurosynth term-based decoding (73) shows that the activation maps derived from the specified contrasts are consistent with the hypothesized processes (SI Appendix, Fig. S9).

**ROI Analyses.** Separate ROIs associated with 1) social valuation and 2) nonvaluation social processing were identified through the use of Neurosynth term-based meta-analyses.

**Social valuation:** First, metamaps of value and social were generated through Neurosynth term-based meta-analyses (73), false discovery rate corrected at  $P < 0.01$  with cluster size  $> 100$  voxels. Valuation regions falling within social regions were identified as the intersection of the value and social metamaps (social  $\cap$  value; social valuation; SI Appendix, Fig. S10). A single significant value cluster was identified within this intersection. This valuation cluster was maximal within vmPFC ( $x = -4, y = 40, z = -8$ ), and a sphere of 6 mm radius around this peak defined the social valuation ROI. To test whether substance exposure is associated with the neural correlates of social valuation, we extracted mean beta estimates within this ROI from the  $OCU_{safe}$  and  $OCU_{risky}$  contrasts, as illustrated in Fig. 2A.

**Nonvaluation social processing:** A nonvaluation social processing map was constructed as the social metamap excluding voxels common to the value metamap (social  $\cap$  value; SI Appendix, Fig. S11). The largest cluster falling within this subtraction was maximal within dmPFC ( $x = 2, y = 56, z = 20$ ), and a sphere of 6 mm radius around this peak defined the nonvaluation social processing ROI illustrated in Fig. 2B. To test whether substance exposure is associated with neural processing of social information unrelated to valuation, we extracted beta estimates from 1) the contrast of Safe trials–Solo trials and 2) the contrast of Risky trials–Solo trials, as illustrated in Fig. 2B, where Safe and Risky trials present information from social peers and Solo trials do not. The results of additional ROIs associated with nonvaluation social processing are presented in SI Appendix, Table S1 and are consistent with the results presented in Fig. 2B.

**Data Availability.** Analytic scripts are available on Virginia Tech GitLab (<https://code.vt.edu/maorloff/adolescent-peer-influence>); unthresholded first-level images are available on Neurovault (<https://neurovault.org/collections/8727/>); anonymized raw fMRI images are available on OpenNeuro (<http://doi.org/10.18112/openneuro.ds003096.v1.0.0>).

**ACKNOWLEDGMENTS.** This work was supported in part by the NIH (MH091872 to P.H.C.; DA036017, MH115221, MH122948 to B.K.-C.; DA042274 to P.H.C. and D.C.) and UNIST internal funding (1.180031.01 to D.C.). We thank Alexandra Hanlon and the statistical team at the Virginia Tech Center for Biostatistics and Health Data Science, Brennan Delattre and Jacob Lee for their research support, and Jae Shin for his technical assistance.

1. J. van Hoorn, A. J. Fuligni, E. A. Crone, A. Galván, Peer influence effects on risk-taking and prosocial decision-making in adolescence: Insights from neuroimaging studies. *Curr. Opin. Behav. Sci.* **10**, 59–64 (2016).
2. W. A. Brechwald, M. J. Prinstein, Beyond homophily: A decade of advances in understanding peer influence processes. *J. Res. Adolesc.* **21**, 166–179 (2011).
3. L. Steinberg, A social neuroscience perspective on adolescent risk-taking. *Dev. Rev.* **28**, 78–106 (2008).
4. B. J. Casey, R. M. Jones, T. A. Hare, The adolescent brain. *Ann. N. Y. Acad. Sci.* **1124**, 111–126 (2008).
5. J. A. Ford, Social learning theory and nonmedical prescription drug use among adolescents. *Sociol. Spectr.* **28**, 299–316 (2008).
6. J. Chein, D. Albert, L. O'Brien, K. Uckert, L. Steinberg, Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Dev. Sci.* **14**, F1–F10 (2011).
7. M. Z. Levitt, R. L. Selman, J. B. Richmond, The psychosocial foundations of early adolescents' high-risk behavior: Implications for research and practice. *J. Res. Adolesc.* **1**, 349–378 (1991).
8. T. J. Dishion, D. W. Andrews, Preventing escalation in problem behaviors with high-risk young adolescents: Immediate and 1-year outcomes. *J. Consult. Clin. Psychol.* **63**, 538–548 (1995).
9. R. Ramirez, A. Hinman, S. Sterling, C. Weisner, C. Campbell, Peer influences on adolescent alcohol and other drug use outcomes. *J. Nurs. Scholarsh.* **44**, 36–44 (2012).
10. R. L. Akers, *Deviant Behavior: A Social Learning Approach* (Wadsworth Publishing Company, Belmont, CA, 1985).
11. D. Chung, G. I. Christopoulos, B. King-Casas, S. B. Ball, P. H. Chiu, Social signals of safety and risk confer utility and have asymmetric effects on observers' choices. *Nat. Neurosci.* **18**, 912–916 (2015).
12. I. H. Lee, Market crashes and informational avalanches. *Rev. Econ. Stud.* **65**, 741–759 (1998).
13. E. van de Waal, C. Borgeaud, A. Whiten, Potent social learning and conformity shape a wild primate's foraging decisions. *Science* **340**, 483–485 (2013).
14. B. R. Braams, J. Y. Davidow, L. H. Somerville, Developmental patterns of change in the influence of safe and risky peer choices on risky decision-making. *Dev. Sci.* **22**, e12717 (2019).
15. L. Steinberg, A. Fletcher, N. Darling, Parental monitoring and peer influences on adolescent substance use. *Pediatrics* **93**, 1060–1064 (1994).
16. A. Galvan, Adolescent development of the reward system. *Front. Hum. Neurosci.* **4**, 6 (2010).
17. S.-J. Blakemore, The social brain in adolescence. *Nat. Rev. Neurosci.* **9**, 267–277 (2008).
18. E. A. Crone, R. E. Dahl, Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nat. Rev. Neurosci.* **13**, 636–650 (2012).
19. A. Strashny, "Age of substance use initiation among treatment admissions aged 18 to 30: The CBHSQ Report" (Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration, Rockville, MD, 2014).
20. T. D. Ritchwood, H. Ford, J. DeCoster, M. Sutton, J. E. Lochman, Risky sexual behavior and substance use among adolescents: A meta-analysis. *Child. Youth Serv. Rev.* **52**, 74–88 (2015).
21. K. M. King, B. T. Meehan, R. S. Trim, L. Chassin, Marker or mediator? The effects of adolescent substance use on young adult educational attainment. *Addiction* **101**, 1730–1740 (2006).
22. E. J. D'Amico, M. O. Edelen, J. N. V. Miles, A. R. Morral, The longitudinal association between substance use and delinquency among high-risk youth. *Drug Alcohol Depend.* **93**, 85–92 (2008).
23. J. D. Grant *et al.*, Adolescent alcohol use is a risk factor for adult alcohol and drug dependence: Evidence from a twin design. *Psychol. Med.* **36**, 109–118 (2006).
24. D. Fuhrmann, L. J. Knoll, S. J. Blakemore, Adolescence as a sensitive period of brain development. *Trends Cogn. Sci.* **19**, 558–566 (2015).
25. S. Hansell, H. R. White, Adolescent drug use, psychological distress, and physical symptoms. *J. Health Soc. Behav.* **32**, 288–301 (1991).
26. C. L. Odgers *et al.*, Is it important to prevent early exposure to drugs and alcohol among adolescents? *Psychol. Sci.* **19**, 1037–1044 (2008).
27. J. H. Patton, M. S. Stanford, E. S. Barratt, Factor structure of the Barratt impulsiveness scale. *J. Clin. Psychol.* **51**, 768–774 (1995).
28. E. Gullone, S. Moore, S. Moss, C. Boyd, The adolescent risk-taking questionnaire: Development and psychometric evaluation. *J. Adolesc. Res.* **15**, 231–250 (2000).
29. T. M. Achenbach, L. A. Rescorla, *Manual for the ASEBA Preschool Forms and Profiles* (University of Vermont, Research Center for Children, Youth, Burlington, VT, 2000), vol. 30.
30. B. Reynolds *et al.*, Laboratory and self-report assessments of impulsive behavior in adolescent daily smokers and nonsmokers. *Exp. Clin. Psychopharmacol* **15**, 264–271 (2007).
31. J. Mazanov, D. G. Byrne, Modelling change in adolescent smoking behaviour: Stability of predictors across analytic models. *Br. J. Health Psychol.* **13**, 361–379 (2008).
32. M. Ernst *et al.*, Behavioral predictors of substance-use initiation in adolescents with and without attention-deficit/hyperactivity disorder. *Pediatrics* **117**, 2030–2039 (2006).
33. A. Rangel, C. Camerer, P. R. Montague, A framework for studying the neurobiology of value-based decision making. *Nat. Rev. Neurosci.* **9**, 545–556 (2008).
34. C. C. Ruff, E. Fehr, The neurobiology of rewards and values in social decision making. *Nat. Rev. Neurosci.* **15**, 549–562 (2014).

35. R. Otten, C. J. Mun, T. J. Dishion, The social exigencies of the gateway progression to the use of illicit drugs from adolescence into adulthood. *Addict. Behav.* **73**, 144–150 (2017).
36. D. Bernoulli, Exposition of a new theory on the measurement of risk. *Econometrica* **22**, 23–36 (1954).
37. L. H. Somerville *et al.*, The medial prefrontal cortex and the emergence of self-conscious emotion in adolescence. *Psychol. Sci.* **24**, 1554–1562 (2013).
38. P. Shaw *et al.*, Neurodevelopmental trajectories of the human cerebral cortex. *J. Neurosci.* **28**, 3586–3594 (2008).
39. L. H. Somerville, Special issue on the teenage brain: Sensitivity to social evaluation. *Curr. Dir. Psychol. Sci.* **22**, 121–127 (2013).
40. S.-J. Blakemore, K. L. Mills, Is adolescence a sensitive period for sociocultural processing? *Annu. Rev. Psychol.* **65**, 187–207 (2014).
41. D. J. Levy, P. W. Glimcher, The root of all value: A neural common currency for choice. *Curr. Opin. Neurobiol.* **22**, 1027–1038 (2012).
42. G. I. Christopoulos, B. King-Casas, With you or against you: Social orientation dependent learning signals guide actions made for others. *Neuroimage* **104**, 326–335 (2015).
43. D. Schiller, J. B. Freeman, J. P. Mitchell, J. S. Uleman, E. A. Phelps, A neural mechanism of first impressions. *Nat. Neurosci.* **12**, 508–514 (2009).
44. N. E. Blankenstein, E. A. Crone, W. van den Bos, A. C. van Duijvenvoorde, Dealing with uncertainty: Testing risk-and ambiguity-attitude across adolescence. *Dev. Neuro-psychol.* **41**, 77–92 (2016).
45. M. Gardner, L. Steinberg, Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Dev. Psychol.* **41**, 625–635 (2005).
46. S. J. Segalowitz *et al.*, Adolescent peer interaction and trait surgency weaken medial prefrontal cortex responses to failure. *Soc. Cogn. Affect. Neurosci.* **7**, 115–124 (2012).
47. A. R. Smith, J. Chein, L. Steinberg, Peers increase adolescent risk taking even when the probabilities of negative outcomes are known. *Dev. Psychol.* **50**, 1564–1568 (2014).
48. A. Weigard, J. Chein, D. Albert, A. Smith, L. Steinberg, Effects of anonymous peer observation on adolescents' preference for immediate rewards. *Dev. Sci.* **17**, 71–78 (2014).
49. O. Attanasio, A. Barr, J. C. Cardenas, G. Genicot, C. Meghir, Risk pooling, risk preferences, and social networks. *Am. Econ. J. Appl. Econ.* **4**, 134–167 (2012).
50. R. M. Raafat, N. Chater, C. Frith, Herding in humans. *Trends Cogn. Sci.* **13**, 420–428 (2009).
51. D. S. Fareri, M. A. Niznikiewicz, V. K. Lee, M. R. Delgado, Social network modulation of reward-related signals. *J. Neurosci.* **32**, 9045–9052 (2012).
52. T. Strombach *et al.*, Social discounting involves modulation of neural value signals by temporoparietal junction. *Proc. Natl. Acad. Sci. U.S.A.* **112**, 1619–1624 (2015).
53. M. S. Stanford *et al.*, Fifty years of the Barratt impulsiveness scale: An update and review. *Pers. Individ. Dif.* **47**, 385–395 (2009).
54. J. M. Bjork, D. A. Pardini, Who are those “risk-taking adolescents”? Individual differences in developmental neuroimaging research. *Dev. Cogn. Neurosci.* **11**, 56–64 (2015).
55. J. S. Tucker *et al.*, Substance use among middle school students: Associations with self-rated and peer-nominated popularity. *J. Adolesc.* **34**, 513–519 (2011).
56. K. Izuma, R. Adolphs, Social manipulation of preference in the human brain. *Neuron* **78**, 563–573 (2013).
57. V. S. Chib, A. Rangel, S. Shimojo, J. P. O'Doherty, Evidence for a common representation of decision values for dissimilar goods in human ventromedial prefrontal cortex. *J. Neurosci.* **29**, 12315–12320 (2009).
58. D. J. Levy, P. W. Glimcher, Comparing apples and oranges: Using reward-specific and reward-general subjective value representation in the brain. *J. Neurosci.* **31**, 14693–14707 (2011).
59. K. E. Sip, D. V. Smith, A. J. Porcelli, K. Kar, M. R. Delgado, Social closeness and feedback modulate susceptibility to the framing effect. *Soc. Neurosci.* **10**, 35–45 (2015).
60. L. H. Somerville, W. M. Kelley, T. F. Heatherton, Self-esteem modulates medial prefrontal cortical responses to evaluative social feedback. *Cereb. Cortex* **20**, 3005–3013 (2010).
61. J. Zaki, J. Schirmer, J. P. Mitchell, Social influence modulates the neural computation of value. *Psychol. Sci.* **22**, 894–900 (2011).
62. The Centers for Disease Control and Prevention, Youth risk behavior survey. <https://www.cdc.gov/healthyyouth/data/yrbs/questionnaires.htm>. Accessed 2 April 2016.
63. C. A. Holt, S. K. Laury, Risk aversion and incentive effects. *Am. Econ. Rev.* **92**, 1644–1655 (2002).
64. P. Royston, W. Sauerbrei, *Multivariable Model-building: A Pragmatic Approach to Regression Analysis Based on Fractional Polynomials for Modelling Continuous Variables* (John Wiley & Sons, 2008), vol. 777.
65. P.-C. Bürkner, brms: An R package for Bayesian multilevel models using stan. *J. Stat. Softw.* **26**, 26206 (2017).
66. D. Makowski, M. Ben-Shachar, D. Lüdtke, bayestestR: Describing effects and their uncertainty, existence and significance within the Bayesian framework. *J. Open Source Softw.* **4**, 1541 (2019).
67. W.-Y. Ahn, N. Haines, L. Zhang, Revealing neurocomputational mechanisms of reinforcement learning and decision-making with the hBayesDM package. *Comput. Psychiatr.* **1**, 24–57 (2017).
68. N. D. Daw, “Trial-by-trial data analysis using computational models” in *Decision Making, Affect, and Learning: Attention and Performance XXIII*, M. R. Delgado, E. A. Phelps, T. W. Robbins, Eds. (Oxford University Press, 2011), 23, pp. 3–38.
69. Stan Development Team, RStan: The R Interface to Stan (Version 2.10.1, 2016). <http://mc-stan.org/>. Accessed 19 January 2017.
70. B. Carpenter *et al.*, Stan: A probabilistic programming language. *J. Stat. Softw.* **76**, 27933 (2017).
71. A. Gelman, D. B. Rubin, Inference from iterative simulation using multiple sequences. *Stat. Sci.* **7**, 457–472 (1992).
72. J. S. Siegel *et al.*, Statistical improvements in functional magnetic resonance imaging analyses produced by censoring high-motion data points. *Hum. Brain Mapp.* **35**, 1981–1996 (2014).
73. T. Yarkoni, R. A. Poldrack, T. E. Nichols, D. C. Van Essen, T. D. Wager, Large-scale automated synthesis of human functional neuroimaging data. *Nat. Methods* **8**, 665–670 (2011).