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Genetic Moderation of Phenotypic and Neural Indicators of Peer Influenced Risk-taking Behavior: An Experimental Investigation

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Genetic Moderation of Phenotypic and Neural Indicators of Peer Influenced Risk-taking Behavior: An Experimental Investigation

by

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Abstract

Risk-taking behavior (RTB) is defined as behavior involving the probability of reward with concurrent probability of some negative outcome. Peer influence is among the most robust predictors of RTB, such that greater peer influence, particularly deviant or delinquent peer influence, is associated with increased RTB. Evidence suggests that those with genetic predispositions for RTB may also be more susceptible to peer influence as a function of genotype. Given that genetic polymorphisms within the dopaminergic system have evidenced associations with various forms of RTB and delinquent peer affiliation, it is possible that these genes may interact with peer influence to predict increased RTB, a process called gene × environment interaction (G×E). We expected that those genetically at risk would take more risks in the presence of a peer than alone. To test this effect, five polymorphisms within the dopaminergic system were genotyped in a sample of 85 undergraduate students. Participants completed a behavioral risk task alone and in the presence of a peer providing "risky" feedback. No significant G×Es were identified for any of the dependent variables. However, participants took significantly more risks in the presence of a risky peer than when taking risks alone. These results suggest that G×E may not be a relevant process for peer-influenced RTB during late adolescence. It is possible that G×E is a relevant process during early adolescence, while gene-environment correlation (rGE) is the dominant process during late adolescence. Future research would benefit from testing whether these genes are relevant to G×E in early adolescence, as well as to rGE during late adolescence.

Genetic Moderation of Phenotypic and Neural Indicators of Peer Influenced Risk-taking Behavior: An Experimental Investigation

Risk-taking behaviors (RTB), defined as behavior that involves the probability of reward with concurrent probability of some negative outcome (e.g., alcohol and drug use, gambling, and sexual risk taking), are an especially problematic and prevalent set of behaviors during late adolescence (DiClemente, Hansen, & Ponton, 1995; Leigh, 1999; Resnick et al., 1997). It is estimated that late adolescents are responsible for 30% of all fatal drunk driving accidents, 85% of those that drink and drive also report binge drinking, 44% report having two or more active sexual partners, and 75% report non-condom use (Centers for Disease Control, 2012a; U.S. Bureau of the Census, 1990; Williams et al., 1992). These behaviors contribute to many negative outcomes, such as HIV/AIDS. Indeed, those 13 – 24 account for the second largest percentage of new HIV infections (26%; 12,200), immediately following ages 25 – 34 (31%; 14,500; Centers for Disease Control, 2012b). Moreover, once RTBs are established in late adolescence they continue to be major contributors to serious health problems in adulthood, including drug and alcohol dependence, sexually transmitted diseases, and cancer (DiClemente, Hansen, & Ponton, 1995; U.S. Preventative Services Task Force, 1989; Zuckerman, Ball, & Black, 1990). In addition, risky behaviors commonly co-occur (Igra & Irwin, 1995; Irwin & Shafer, 1992; Osgood, Johnston, O'Malley, & Bachman, 1988). For instance, substance using adolescents are 6 times more likely to be sexually active, and sexually experienced adolescents are 8 times more likely to use illicit substances (Fisher, Eke, Cance, Hawkins, & Lam, 2008). This in turn

increases risk for serious negative outcomes. As a result, researchers have made ample efforts to identify the influences and causes of these co-occurring maladaptive behaviors. From this research, two important themes have consistently emerged: the influence of peers and genetic vulnerabilities.

Peer Influences on RTB

Among the many influences on RTB, the role of peers has received considerable attention. A wealth of literature has repeatedly shown that peer influence predicts negative behaviors and outcomes, including drug and alcohol use, tobacco use, sexual intercourse, academic failure, and future RTB (Ali & Dwyer, 2009; Cavalca et al., 2012; Chassin, Hussong, & Beltran, 2009; Duncan, Duncan, & Strycker, 2000; Holman & Sillars, 2012). More importantly, recent work has shown that peer influence does not exert a stable force on negative behavior throughout development. Instead, peers are more or less influential for certain behaviors during crucial developmental stages. In one study, Gardner & Steinberg (2005) randomly assigned participants to complete a risk-taking task alone or in the presence of age and sex-matched peers. They found that adolescents (13–16) and late adolescents (18–22) took more risks, focused more on the benefits of risky behavior (as opposed to the costs), and made riskier decisions while in the presence of peers than alone. In contrast, peer presence had little effect on RTB in adults. Similarly, deviant peer influence is considerably more correlated with RTB during late adolescence than all other ages (Kaplan, Martin, & Robbins, 1984). Thus, peer influenced RTB seems to be particularly powerful during late adolescence.

Neural Indicators of RTB and Susceptibility to Peer Influence

With the advent of brain imaging technology, advances within the field of psychology have allowed researchers to study the neural correlates of RTB as a means to better understand

its etiology and biological influences. Indeed, research has found support for biological correlates of RTB. In a recent study by Fein & Chang (2008), participants were asked to complete a behavioral measure of RTB while monitored via electroencephalogram (EEG). F-ERN (feedback error-related negativity, a neural response to negative feedback) was observed over the medio-frontal cortex during trials in which excessive risk was taken. This suggests that RTB is linked with immediate physiological response in neural substrates associated with decision-making. Moreover, results revealed that amplitude was weaker in participants with greater family history of alcohol problems. In other words, these participants were less neurologically sensitive to negative feedback. Similar findings have been obtained in fMRI studies (Cservenka & Nagel, 2012). Thus, there is evidence to suggest that RTB is represented by specific brain components in neural substrates.

Other research suggests that the same neural areas that reflect RTB also reflect susceptibility to peer influence (Nelson, Leibenluft, McClure, & Pine 2005). For instance, Grosbras et al. (2007) utilized fMRI and observed that participants with low resistance to peer influence evidenced significantly less activation in the frontal cortex, particularly areas associated with decision-making, while completing a peer-influenced task. Thus, it appears that peer influence and RTB are represented in the same neurological areas. Recent research has also shown that brain components (e.g., ERN, FRN) are not necessarily stable, but show increased or decreased amplitude in response to experimental manipulation. Specifically, Segalowitz et al. (2012) examined changes in event related potential (ERP) amplitude as a function of peer presence during a laboratory risk-taking task. Results indicated that participants had smaller FRN amplitude in response to negative feedback while in the presence of peers than while completing the task alone. In sum, there is evidence that the presence of peers influences RTB both

phenotypically (i.e., laboratory indices of risk-taking) as well as neurologically, as shown by reduced FRN amplitude (i.e., reduced sensitivity to negative feedback).

Genetic Contributions to RTB and Peer influence

Although there are robust effects for peer-influenced RTB, not every individual is susceptible to deviant peer influence. Indeed, a variety of constructs moderate the strength of peer influence, including characteristics of the influencing peer and those of the adolescent. For example, the behaviors of popular individuals are more likely to be replicated by their peers than the behaviors of their less popular counterparts (Cillessen & Rose, 2005; Mayeux, Sandstrom, & Cillessen, 2008; Parkhurst & Hopmeyer, 1998; Prinstein, Meade, & Cohen, 2003; Rancourt & Prinstein, 2010). Alternatively, resistance to peer influence functions as a protective factor for peer influences on RTB. Research has shown that resistance to peer influence decreases throughout adolescence and increases during the transition to adulthood, albeit with a sharp decline between the ages of 18 and 22 (Steinberg & Monahan, 2007). This reduction maps on quite well with the same developmental window that RTB, including binge drinking and sexual risk taking, show an increase.

Genetic influences have also been implicated as potential moderators of RTB and peer influence. While we know that RTB (including alcohol and substance dependence, sexual risk taking, and gambling) is quite heritable from twin studies (heritability estimates range 34% - 58%), there is also a strong possibility that those who are more genetically at risk for RTB are also more susceptible to the influence of their maladaptive counterparts (Slutske, Zhu, Meier, & Martin, 2011; Verwij, Zeitsch, Bailey, & Martin, 2009; Vrieze, McGue, Miller, Hicks, & Iacono, 2013). For instance, there is evidence that association with deviant peers is highly heritable. Indeed, heritability of peer group deviance is estimated to be ~30% during pre-adolescence

(between ages 8 and 11), and rises to as high as 50% during late adolescence and emerging adulthood (between 22 and 25; Kendler et al., 2007). Other research has observed genetic correlations (r = .42 - .64) between peer influence and RTB (Button et al., 2009; Hicks et al., 2013; Rowe & Osgood, 1984). In other words, a large proportion of genetic characteristics responsible for peer influence are also responsible for RTB, suggesting peer influence and RTB share some common genetic basis.

As a result, researchers have begun to identify particular genes that may confer risk for RTB. For instance, genes within the dopaminergic system are associated with a variety of externalizing and risk-taking behaviors, including impulsivity, novelty seeking, aggressive behavior, drug and alcohol dependence, binge drinking, and behavioral measures of RTB (Amstadter et al., 2012; Eisenberg et al., 2007; Golimbet, Alfimova, Gritsenko, & Ebstein 2007; Han et al., 2006; Pinto et al., 2009; van der Zwaluw, Kuntsche, & Engels, 2011). These genes have also been linked to variability in brain components, including the FRN and ERN, and neural substrates during RTB (e.g., gambling, response inhibition) and peer influenced behavior (Antolin et al., 2009; Congdon, Constable, Lesch, Canli, & Turhan, 2009; Heitland et al., 2012; Konishi et al., 2004; Orsinksky, Hewig, Alexander, & Hennig, 2012). However, not every gene within the dopaminergic system has evidenced associations with RTB. Only select polymorphisms, or variations of genetic expression, including those within the COMT, DAT1, DRD2, and DRD4 genes, seem to place individuals at particular risk for RTB. For example, carriers with versions of the COMT val^{158}met polymorphism, 10-repeats of DAT1, variations within DRD2 Taq1a and -141 ins/del, and DRD4 7-repeats are more likely to engage in a variety of RTB, such as novelty seeking, rule-breaking, binge drinking, and financial risk-taking, respectively (Burt & Mikolajewski, 2008; Dreber et al., 2009; Golimbet et al., 2007; van der

Zwaluw et al., 2011). These findings suggest that even at the individual level genes can account for variation in RTB.

Other research has found that the same genes that account for RTB have evidenced associations with peer influence. For example, DAT1 10-repeat, DRD4 7-repeat, and DRD2 Taq A1 carriers have higher delinquent peer affiliation, over and above variables such as delinquent involvement, self-control, and alcohol and drug use (Beaver, Wright, & DeLisi, 2008; Beaver, Gibson, DeLisi, Vaughn, & Wright 2012; Vaughn, DeLisi, Beaver, & Wright, 2009). These findings suggest that peer influence makes individuals already genetically liable for RTB associate with friends that may increase their RTB involvement. Thus, it appears that genes within the dopaminergic system may be a part of the common genetic factor responsible for peer influence and RTB.

While this evidence speaks more to peer selection, it also provides suggestive evidence that there may be specific genetic profiles linked with being more or less resistant to peer-influenced RTB. On a broader level, this is referred to as a G×E interaction, which suggests that those with particular genetic vulnerabilities are more vulnerable to social "pushes" in the development of antisocial and risk behavior. The presence of G×E in antisocial and RTB has been demonstrated using adoption (Cadoret, Cain, & Crowe, 1983; Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Cloninger, Sigvardsson, Bohman, & von Knorring, 1982), twin (Button, Scourfield, Martin, Purcell, & McGuffin, 2005; Boutwell, Beaver, Barnes, & Vaske, 2012; Tuvblad, Grann, & Lichtenstein, 2006), and molecular genetic studies (Caspi et al., 2002). As one example, Button et al., (2007) found that genetic overlap of deviant peer influence and risk behavior was higher in those with risky peers than those with less risky peers, even after accounting for a gene-environment correlation effect. In other words, those with a higher genetic

predisposition for risky behavior were more genetically predisposed to peer-influenced risky behavior. This suggests that those with certain genetic vulnerabilities may be more susceptible to peer-influenced RTB. However, the relationship between peer influence and RTB as a function of specific vulnerability within the dopaminergic system has yet to be tested.

Limitations of Genetic Research

While there is evidence for relationships between these polymorphisms, peer influence, RTB, and neural substrates, it is well known that effect sizes for individual polymorphisms predicting complex phenotypes, such as RTB, generally range from small to moderate (Plomin, Haworth, & Davis, 2009). These limitations are compounded when considering the typically limited sample sizes used in psychiatric genetics. Thus, efforts to replicate relationships between specific polymorphisms and behavioral phenotypes have proven difficult (e.g., Hawi, Millar, Daly, Fitzgerald, & Gill, 2000). An additional explanation is that many complex phenotypes are polygenic in nature (Goldman, Oroszi, & Ducci, 2005). In other words, multiple genes influence phenotypes, such that each gene has a small effect on phenotypic expression.

To address this issue, researchers have begun to develop methods by which risk indices for specific behaviors are assigned based on an individual's genetic makeup. These indices take into account a *set* of genes that confer risk for a phenotype, as opposed to individual genes. Indeed, dopaminergic genetic risk indices have been shown to account for a significant amount of variation in sensation seeking and cocaine dependence (Derringer et al., 2010; Derringer et al., 2012). As such, behavior can be examined via a more comprehensive picture of the underlying biological factors that account for those behaviors, while at the same time addressing the difficulties of replicating genetic effects.

Current Study

The current study implements a transdisciplinary perspective in examining peer influences on risk behavior in an experimental setting. Of particular interest are the relationships between dopaminergic genetic risk, peer influence, and RTB. We also sought to elucidate how genetic risk might confer neurological vulnerability to peer influenced RTB. Research informed by this transdisciplinary approach is necessary to disentangle the complexities of human behavior. As such, identification of these processes will provide researchers and clinicians insight into the various levels influencing RTB.

Consistent with previous literature, we expected to observe main effects for peer influence and aggregate genetic risk on RTB, such that RTB is higher when in the presence of risky peers and higher for those with high genetic risk. Previous literature has also suggested that those with certain genetic risk are more likely to be influenced by peers. Therefore, we expected to observe a G×E interaction, such that those with high polymorphic genetic risk would be be more susceptible to the influence of peers. Neurologically, we hypothesized that those with high genetic risk would show blunted (more positive) FRN during trials in which the balloon pops, especially in the presence of peers.

Method

Participants

Participants included 85 undergraduate students (70.24% female) recruited via the University of South Florida Department of Psychology subject pool. Eligible participants were English-speaking, non-Hispanic European-Americans (EAs) between the ages of 18 and 22 (M = 19.18, SD = 1.34). This age range was selected because research indicates that youths are particularly vulnerable to peer influence for risky behavior prior to age 23 (Gardner & Steinberg, 2005; Steinberg & Monahan, 2007). We limited data collection to non-Hispanic European-Americans for the following reasons. First, to eliminate population stratification effects. Population stratification may lead to false positives in the associations between specific polymorphisms and phenotypes due to differential frequency distributions of alleles between ethnic groups (Freedman et al., 2004). Secondly, we limited collection to EAs because allelic base rates for the selected polymorphisms are limited in non-EAs. For example, base rates for COMT met/met and DAT1 9-repeat are as low as 11.1%, and 38%, respectively, among non-EAs. On the other hand, these alleles are distributed more liberally among EAs (20%, 50%; Forbes et al., 2009).

We were concerned that participants with "risky" genotypes would be less likely than those with "un-risky" genotypes to participate in research, which might result in un-representative distributions of genotypes. To protect from this we sampled high and low impulsive participants, a construct related to the genotypes of interest, by administering the

Barratt Impulsiveness Scale (BIS-11) prior to enrollment in the study. Specifically, participants were recruited if they scored greater than or equal to one-half standard deviation above or less than or equal to one-half standard deviation below the mean for the BIS-11 total score. We expected the sample mean and standard deviation to be 62.0 and 9.0, respectively (Helfritz & Stanford, 2006; Stolenberg, Batier, & Birgenheir, 2008; Whitney, Jameson, & Hinson, 2004). Thus, participants with BIS-11 scores greater than or equal to 66 and less than or equal to 58 were recruited. This technique has been documented in the literature using the BIS-11 (Stanford, Greve, Boudreaux, & Mathias, 1996).

Measures

Impulsivity Screening

To assess impulsivity participants were administered the Barratt Impulsiveness Scale (BIS-11; Barratt, 1959). The BIS-11 is a widely used 30-item self-report measure that yields three facets of impulsivity: motor impulsiveness (e.g., actions without thinking), attentional impulsiveness (e.g., lack of careful thinking), and non-planning impulsiveness (e.g., failing to plan for the future; Patton, Stanford, & Barratt, 1995). It has been used numerous times in college populations, and has established reliability and validity (Patton, Stanford, & Barratt, 1995; Stanford et al., 1996; Stolenberg, Batier, & Birgenheir, 2008).

Demographic Information

A variety of demographic variables, including age, gender, handedness, and family history of mental illness were collected via self-report. We also collected data on family history of psychiatric disorders and substance abuse as more distal indicators of genetic risk for RTB. Particularly, family history of substance abuse was collected for the following substances: alcohol, tobacco, marijuana, cocaine, crack, amphetamines, methamphetamines, ecstasy, heroin,

methadone, opiates, barbiturates, sedatives, and inhalants. As for psychiatric diagnoses, family history of conduct disorder and antisocial personality disorder were collected.

Phenotypic Risk Index of Behavioral Disinhibition

The 160-item Externalizing Inventory was used to index self-reported externalizing behavior, including, but not limited to, drug and alcohol use problems, theft, physical-relational-destructive aggression, and impulsivity. The 160-item Externalizing Inventory is composed of a subset of items from the original 415-item measure developed by Krueger, Markon, Partrick, Benning, & Kramer (2007). It is highly correlated with the original 415-item version and has evidenced reliability and validity (Patrick, Kramer, Krueger, & Markon, 2013). Total scores for this measure were split into two groups (high and low externalizers) via a median split of the data.

Genetic Risk Indices for Behavioral Disinhibition

A distal marker of genetic risk for behavioral disinhibition was created via a sum across family history status for five variables: any substance abuse, alcohol abuse, nicotine abuse, conduct disorder, and antisocial personality disorder. First, to create the family history variable for any substance abuse, participants were categorized into family history positive and family history negative (coded as 0 for absent and 1 for present) for substance abuse by whether they reported a family history of any of the following: marijuana, cocaine, crack, amphetamines, methamphetamines, ecstasy, heroin, methadone, opiates, barbiturates, sedatives, and inhalants. This value was added to the family history status of alcohol abuse, nicotine abuse, conduct disorder, and antisocial personality disorder (each coded 0 for absent and 1 for present) for each participant. The result (family history for behavioral disinhibition) ranged from 0 to 4 (no participants endorsed a family history of antisocial personality disorder). Thereafter, a median

split was conducted to code participants into family history positive (FH+, coded as 1) and family history negative (FH-, coded as 0) for behavioral disinhibition.

Last, as a proximal indicator of genetic risk for behavioral disinhibition, five candidate genetic variations in genes involved in the dopaminergic system were analyzed, including DAT1, DRD2, DRD4, and COMT. Each polymorphism has evidenced associations with a number of externalizing behaviors and traits, as well as associations with neural indices of RTB. DNA sources were collected via buccal (cheek) cells. These samples were isolated and analyzed by a co-investigator at Moffitt Cancer Center. Analysis of DRD2 Taq1a, DRD2 -141 ins/del, and COMT val^{158}met were completed using TaqMan allelic differentiation analyses. DAT1 9/10-repeat and DRD4 7/more-repeat were analyzed using PCR products based electrophoresis. Genotype frequencies were as follows: DAT1: 9-repeat carriers: 66.7%, 10/10: 33.3%; DRD4: 7-repeat carriers: 22%, Others: 78%; DRD2 -141 ins/ins: 85%, ins/del: 15%, del/del: 0%, DRD2: A1A1: 12.5%, A1A2: 17.5%, A2A2: 70%; COMT: val/val: 41.97%, val/met: 48.15%, met/met: 9.88%. These genetic variations assort independently, such that carriers of one genetic variant are no less or more likely to carry another genetic variant.

Genetic Risk Index

We defined aggregate genetic risk by creating a risk index that represents the collective contribution of five genetic polymorphisms on peer-influenced RTB. To do this, we coded each individual polymorphism according to which allelic combinations have evidenced associations with behavioral disinhibition. In other words, polymorphisms were coded as 1, 2, or 3 (or 1 or 2 for those polymorphisms with just two allelic combinations) based on empirical support for an

association between a polymorphism and an index of behavioral disinhibition.[1] Each polymorphism was then entered into an Ordinary Least Squares (OLS) regression equation predicting total score on the Externalizing Inventory (phenotypic risk). We then defined the risk index by multiplying the regression weight for each polymorphism in the OLS regression equation with the corresponding relative risk for behavioral disinhibition (using the coding scheme described above). The values of polymorphic risk for each participant were then summed. The equation determining the value for aggregate genetic risk was as follows:

$$(Risk_1 * b_1) + (Risk_2 * b_2) + \ldots + (Risk_5 * b_5)$$

Similar methods have been utilized previously to yield a total risk score across a set of individual polymorphisms (Derringer et al., 2010; Derringer et al., 2012; Purcell et al., 2009; Wray, Goddard, & Visscher 2007). Lastly, we conducted a median split on the genetic risk index to create two groups. Those with values for the risk index that fell on the bottom half were coded as low and those on the top half high, representative of those with low and high genetic risk, respectively.

Laboratory Measure of Solitary Risk-taking Behavior

To obtain a baseline measurement of risky behavior participants were asked to complete the Balloon Analog Risk Task (BART) on E-Prime (Psychology Software Tools, Inc., Pittsburgh, PA), a version modified for EEG collection, individually, as well as in the presence of peers providing "risky" feedback. The two BART conditions were counterbalanced to eliminate order effects. During the standard, individual BART, participants were asked to fixate on a balloon in the middle of the screen and administer "pumps" to the balloon. They were

[1] The polymorphisms were coded as follows: DAT1: 9-repeat carriers = 1, 10/10-repeat = 2; DRD2 Taq1a: A2/A2 = 1, A1/A2 = 2, A1/A1 = 3; DRD2 -141 ins/del: ins/del = 1, ins/ins = 2; DRD4: 7-repeat = 2, others = 1; COMT: val/val = 1, val/met = 2, met/met = 3.

informed that earnings increase incrementally for every pump. For the first pump, 50 cents was added to a temporary bank account, 52 for the second, 54 for the third, and so on. These incremental increases were used as a means to increase reward for subsequent risky behavior. Participants were instructed to click a button that "collects" the money and moves it to a permanent bank account that stores all of the money collected if they wanted to quit pumping at any point. However, participants were warned that the balloon could explode at any moment during the task, which would result in 0 cents earned that trial. Consistent with incremental increases in value, each successive pump incrementally increased the probability it could pop. For the second pump there was a 1/19 chance of explosion, a 1/18 chance for the third, and so on until the twentieth pump, where there was a 100% chance that the balloon would explode. A breakpoint was not set on the first trial in order to eliminate the participants receiving negative feedback for an "un-risky" decision. In line with this algorithm, the 10^{th} pump was indicative of the average point of explosion, with number of pumps administered ranging from 1 to 20. Participants were unaware of these probabilities, as well as the average breakpoint. In order to obtain accurate ERP data, participants were required to complete 60 trials while monitored via EEG (Fein & Chang, 2008). In addition, a random delay of 1-1.2 seconds between the participants' response and feedback (e.g., button clicks and explosion) was added to separate the events temporally and allow focused processing of the feedback. ERP data was collected during button clicks/balloon pumps and explosions, indicative of positive and negative feedback, respectively. Lastly, participants were told that they would receive a bonus at the end of the experiment, equivalent to 2.5% of their total earnings (money in the permanent bank account). Participants were not aware of the exact percentage.

The primary behavioral dependent variables for the BART were mean number of balloon explosions and mean adjusted pumps, or the average number of pumps during trials in which money was collected (Lejuez et al., 2002). For ERP collection, the primary dependent variable was average feedback related negativity (FRN) amplitude during pumps in which the balloon popped. The BART was designed to be a behavioral indicator of RTB, but has been used in collection of neurological data as well (Fein & Chang, 2008). The BART has established reliability, validity, and evidenced associations with a number of negative outcomes and traits, including psychopathology, drug use, and impulsivity (Hopko et al., 2006; Hunt, Hopko, Bare, Lejuez, & Robinson, 2005; Lejuez et al., 2002; White, Lejuez, & de Wit, 2008).

Laboratory Measure of Peer-Influenced Risk-taking Behavior

We also assessed in-lab RTB as a function of "risky peer" presence. Participants were asked to complete the BART while receiving feedback from a risky peer while monitored with EEG. In order to manipulate peer presence, participants were asked to complete the BART while receiving suggestions/pumping strategies from a peer that was completing the second part of the experiment and was familiar with the task. In addition, the participant was told that the confederate earned a substantial amount of money when they completed the task (i.e., the maximum amount possible, $20). In reality, the peer was a confederate research assistant that monitored the participant's performance from directly behind them. The research assistant was assigned to monitor the number of pumps each participant administered, as well as the outcome of the trial (i.e., explosion or collection of money). The confederate made the following statement if the participant collected money and administered greater than the average amount of pumps to explosion: "That's what I would have done". If the participant administered fewer than the average number of pumps to explosion, the peer said, "I would have done more". If the

balloon popped under any condition (i.e., above or below the mean explosion), the participant did not receive feedback. Feedback was spoken to the participant approximately 1-2 seconds after trials had been completed to reduce error in collection of ERP data. Participants were told that they were required to listen to the feedback before beginning a new trial, but that following the peers instructions would have no effect on whether they were paid or not. Participants and confederates were matched on age, race, and gender to reduce the likelihood of third variable effects.

In order to determine whether the participant was aware of the manipulation, a post-hoc scale was administered after the peer-influenced BART. Particularly, participants were asked about their perception of the peer immediately after the task. Two independent coders rated the participants' responses as positive, neutral, or negative. A response such as "I followed the peers suggestions" was coded as positive. A response indicative of awareness that the confederate was a research assistant was coded as negative. An example of a neutral response would be, "The suggestions did not affect me". A similar post-hoc assessment has been used effectively as a manipulation check in previous research (Cavalca et al., 2012). None of the participants reported that they knew the confederate was a research assistant working on the study. Thus, all responses were coded as neutral or positive. Participants were also asked if the person providing feedback was familiar to them. Only one participant indicated that they knew the confederate. This participant was excluded from all subsequent analyses.

Electroencephalographic (EEG) Data

FRN data was acquired through 128-electrode Geodesic Sensor Nets (EGI, Eugene) for the BART. The data was referenced to the vertex .1-100 Hz analog filtering at a sampling rate of 250 Hz, digitally filtered at 20 Hz, and segmented into 1000 milliseconds epochs (200

milliseconds before balloon pop or inflation and 800 milliseconds after balloon pop or inflation). Each epoch was then screened for artifacts (i.e., eye blinks, participant movement) and the remaining, cleaned data was sorted by condition (pop and inflation) and averaged to create the ERPs per condition. Only conditions that had fewer than 10 acceptable trials were included in the final analyses for the BART. Average ERPs were then baseline corrected to a pre-stimulus period of 200 milliseconds. Individual ERPs were then averaged across all subjects to create the grand average (see Figure 2). Specifically, FRN data was the grand average across all failures (inflations resulting in explosion).

Procedure

Participants volunteered to participate in the study through an online subject pool (SONA). The study contained three components, including collection of self-report measures via Qualtrics, an in-lab experimental session, and an in-lab psychopathology assessment. A visual depiction of the study procedure is presented in Figure 1. Participants were instructed to complete a variety of self-report measures prior to the experimental session. Following registration, a link to the online questionnaire was provided. Participants were required to follow the link to a Qualtrics page where they were provided with an informed consent form describing the study as examining the relationships between risk preferences, biology, and behavior. Following consent, participants entered the last four digits of their university ID and provided self-reports in the following order: BIS-11, Demographic Information, and Externalizing Inventory[2]. To reduce the likelihood of priming inhibition/disinhibition participants were instructed to schedule a laboratory session within 2 – 5 days. Participants' university IDs were

[2] A number of other measures were administered as well, although these details are not relevant to the current manuscript. Additional information regarding these measures and the order of administration is available upon request.

subsequently matched to the study ID they were provided during the subsequent laboratory sessions.

Prior to the laboratory session, a confederate research assistant was matched to the participant on age, gender, and ethnicity. Next, EEG nets were fitted to the participant. They were then randomized into one of two conditions (peer BART first or individual BART first) and lead to a testing room with a single computer. To assess solitary RTB, participants were provided instructions and asked to complete 60 trials of the BART. A brief break was provided afterward. Participants were then lead to the testing room and asked to complete the BART once again. For the peer BART, they were provided instructions for the task and introduced to a "peer" that was familiar with the task and would be monitoring them while they completed it. The peer would be providing feedback/pumping strategies following each trial. They were told to listen to the feedback after each trial, but informed that they *were not required* to follow through with the feedback. Additionally, they were told that the confederate was familiar with the task and had been successful at it in the past (i.e., had won the maximum amount of money possible – "They have won something like $20"). The confederate was then instructed to sit directly behind the participant and provide instructions following each trial. The dyad was asked to remain quiet unless the confederate was providing feedback, in which case only the confederate was allowed to speak.

During session two, participants attended a laboratory session and provided genetic samples via buccal cheek swabs. These samples were refrigerated until they were analyzed in Dr. Park's laboratory. Thereafter, participants completed a structured clinical interview with a trained research assistant (MINI). They were told how much money they earned and paid for

their performance on the BART. Participants were then debriefed regarding the deception used during the first laboratory session.

Statistical Analyses

To assess phenotypic and genetic (both distal and proximal) risk for peer-influenced RTB and neural indices of peer-influenced RTB a series of repeated measures ANOVAs predicting 3 dependent variables separately (i.e., average FRN amplitude, mean adjusted pumps, and explosions) were conducted. All analyses were run using risk for behavioral disinhibition (high versus low externalizers, family history + and - for behavioral disinhibition, and high versus low genetic risk) as a between-subjects factor, and experimental condition (i.e., individual BART and peer-influenced BART) as the within-subjects factor. The first set of ANOVAs used a median split of Externalizing Inventory total scores, while the second set of ANOVAs used FH+/FH- for behavioral disinhibition as the between-subjects factor. Last, genetic risk was used as a between-subjects factor to determine whether the selected dopaminergic polymorphisms moderated the relationship between RTB and peer influence.

Results

Moderated Effects for Phenotypic Indicators of RTB

Correlations between all dependent variables and moderators are presented in Table 1. We first tested whether variables more distal to genetic risk for behavioral disinhibition (i.e., a phenotypic variable) moderated the relationship between solitary RTB and peer influenced RTB. Using a median split of externalizing inventory total scores as a between-subjects factor, results revealed a significant within-subjects effect for mean number of pumps [$F(1, 81) = 10.08$, $p < .001$, $d = .99$], such that participants administered more pumps in the peer condition than in the solitary condition. Participants with high externalizing scores administered significantly more pumps than participants with low externalizing scores [$F(1, 81) = 8.03$, $p = .006$, $d = .63$]. The interaction was not significant [$F(1, 81) = 2.13$, $p = .15$, $d = .33$], suggesting that the peer influence manipulation did not have significantly different effects for high and low externalizers. Similar results were found for number of explosions [within-subjects: $F(1, 81) = 18.35$, $p < .001$, $d = .95$; between-subjects: $F(1, 81) = 7.13$, $p = .01$, $d = .59$; interaction: $F(1, 81) = 1.91$, $p = .17$, $d = .31$].

Next, family history of behavioral disinhibition was added as a between subjects-factor to test whether distal genetic risk moderated the relationship between RTB and peer influence. There were significant within-subjects effects for both average number of pumps [$F(1, 81) = 18.97$, $p < .001$, $d = .97$] and number of explosions [$F(1, 81) = 18.77$, $p < .001$, $d = .96$]. Participants administered more pumps and experienced more explosions in the peer condition

than in the solitary condition. No between-subjects effects were observed for average number of pumps [$F(1, 81) = .28$, $p = .60$, $d = .11$] or number of pops [$F(1, 81) = .09$, $p = .77$, $d = .06$]. These results suggest that participants with a family history of behavioral disinhibition did not significantly differ on number of pumps or number of explosions. Neither an interaction for mean number of pumps [$F(1, 81) = .28$, $p = .60$, $d = .11$] nor an interaction for number of explosions [$F(1, 81) = 2.31$, $p = .13$, $d = .34$] by family history of behavioral disinhibition was observed. In other words, participants with a family history of behavioral disinhibition experienced similar changes in number of pumps and explosions from the solitary condition to the peer influenced condition.

Last, we tested whether polymorphic genetic risk across 5 genetic variants moderated the relationship between RTB and peer-influence. For mean number of pumps, there was a significant within-subjects effect [$F(1, 65) = 18.28$, $p < .001$, $d = 1.06$], such that participants administered more pumps in the peer condition than in the solitary condition. The between-subjects effect [$F(1, 65) = .16$, $p = .70$, $d = .09$] and interaction effect [$F(1, 65) = .42$, $p = .52$, $d = .16$] were not significant. Thus, participants with high and low genetic risk had similar levels of average pumps, and were influenced similarly by the manipulation. Similar results were found for number of pops: within-subjects [$F(1, 65) = 12.99$, $p = .001$, $d = .90$], between-subjects [$F(1, 65) = .66$, $p = .42$, $d = .20$], interaction [$F(1, 65) = .01$, $p = .94$, $d = .00$].[3]

[3] Some research suggests that increased levels of neural dopamine transmission efficiency results in elevated RTB. Thus, we utilized an alternative coding scheme for the genetic data, whereby genes were coded in terms of their functional, dopaminergic properties. Polymorphisms were coded higher if they contributed to increased efficiency for dopamine transmission. The following coding scheme was used for the genetic data: DAT1 10/10 = 1, 9/10 = 2, 9/9 = 3, COMT val/val = 1, val/met = 2, met/met = 3, DRD2 -141insdel, ins/del = 1, ins/ins = 2, DRD2 Taq1A A1/A1 = 1, A1/A2 = 2, A2/A2 = 3, and DRD4 7/7 = 1, other repeats = 3). Values were summed across these polymorphisms for each participant. A median split was then used to separate participants into high and low dopaminergic risk groups, with higher values indicative of higher efficiency of dopamine transmission. Similar results to the empirical coding scheme (presented above) were observed for average number of pumps: within-subjects [$F(1, 65) = 19.40$, $p <$

Moderated Effects for Neural Indicators of RTB[4]

We first tested whether phenotypic risk moderated the relationship between FRN for the solitary and peer influenced condition. All analyses were conducted on mean amplitude between 216-264 ms after stimulus onset over a montage of medial fronto-central electrodes (see Figure 2).[5] The within-subjects [$F(1, 47) = 1.72$, $p = .20$, $d = .38$], between-subjects [$F(1, 47) = .11$, $p = .74$, $d = .09$] , and interaction [$F(1, 47) = .01$, $p = .91$, $d = .00$] effects were all non-significant. Thus, FRN in the peer-influenced condition was not significantly different than in the solitary condition, and FRN did not significantly differ as a function of high and low externalizing scores. Moreover, there were similar changes in FRN for high and low externalizers across the two conditions.

Next, family history of behavioral disinhibition was tested as a moderator of the relationship between neural indicators of RTB and peer influence. No significant within-subjects [$F(1, 47) = 1.94$, $p = .17$, $d = .41$] or between-subjects effects [$F(1, 47) = .003$, $p = .96$, $d = .00$] were observed for the FRN. This suggests that the FRN was not significantly different between the peer and solitary RTB conditions, and that FRN amplitudes did not differ across family history positive and negative participants. Further, a non-significant interaction term was observed for the FRN, suggesting that the manipulation influenced both family history positive and negative participants similarly [$F(1, 47) = .57$, $p = .46$, $d = .22$].

Last, we tested whether polymorphic genetic risk moderated the relationship between RTB and peer influence. There were not significant within-subjects [$F(1, 39) = 2.80$, $p = .10$, $d =$

.001], between-subjects [$F(1, 65) = .70$, $p = .41$], interaction [$F(1, 65) = .22$, $p = .64$]. The same results were found for number of pops: within-subjects [$F(1, 65) = 11.61$, $p = .001$], between-subjects [$F(1, 65) = .44$, $p = .51$], interaction [$F(1, 65) = .78$, $p = .38$].

[4] As suggested by previous research, all FRN data was analyzed excluding outliers and left-handed subjects (Willems, Van der Haegen, Fisher, & Francks, 2014)

[5] This range was selected because the wave for the peer-influenced condition and the wave for the solitary condition appeared to separate and conjoin at 216 and 264 ms, respectively.

.54], between-subjects [$F(1, 39) = .83$, $p = .37$, $d = .29$], or interaction effects [$F(1, 39) = .03$, $p = .86$, $d = .06$]. These results suggest that the FRN did not change as a function of experimental condition, there were not group differences between high and low polymorphic genetic risk, and participants in the high and low genetic risk groups had similar neural changes across the solitary and peer-influenced RTB conditions.[6]

[6] Again, we utilized the dopaminergic-risk coding scheme detailed above as an alternative way to conceptualize the genetic risk score. The same results were found for the FRN: within-subjects [$F(1, 39) = 1.92$, $p = .17$], between-subjects [$F(1, 39) = 2.85$, $p = .10$], interaction [$F(1, 39) = 3.92$, $p = .06$].

Discussion

The current study sought to elucidate the moderating role of phenotypic and genetic indices of behavioral disinhibition on the relationship between peer-influence and both phenotypic and neural indices of RTB. An experimental, within-subjects design was used to disentangle the effects of peer influence on RTB. Particularly, participants were required to complete a behavioral task measuring RTB alone and in the presence of a "risky" peer. Genetic data across 5 dopaminergic polymorphisms was collected and tested (collectively) as a moderator of the relationship between RTB and "risky-peer" influence. Moreover, ERPs (particularly the FRN) were collected in response to negative feedback during the behavioral task as an additional index of sensitivity to peer influence.

Results revealed significant within-subjects effects for peer influence on phenotypic RTB, such that participants took significantly more risks when a "risky" peer was providing feedback than when they were alone. These results are consistent with previous reports of robust effects for peer influence, particularly deviant peers, on RTB, alcohol use disorder symptoms, and the broader construct of externalizing behavior (Ali & Dwyer, 2009; Cavalca et al., 2012; Chassin, Hussong, & Beltran, 2009; Duncan, Duncan, & Strycker, 2000; Holman & Sillars, 2012). A significant difference in FRN was not observed between the solitary and peer-influenced condition. Previous research has observed significant changes in FRN between solitary and peer-influenced RTB during a risk-taking task (Segalowitz et al., 2012), although these findings were observed in a sample of 15-year-old males. In contrast, the current sample

included males and females ages 18-22. It is possible that reductions in FRN during a risk-taking task in the presence of peers is a phenomenon that is age and gender specific, such that only males exhibit blunted FRN amplitude during *middle* adolescence.[7] Indeed, previous research has shown that male adolescents 13-16 years old are the most susceptible to peer-influenced RTB when compared with females and participants aged 18 and older (Brown, Clasen, & Eicher, 1986; Gardner & Steinberg, 2005; Parsons, Halkitis, Bimbi, & Barkowski, 2000). In addition, the findings Segalowitz et al. (2012) observed were a function of *familiar* peer presence. Particularly, participants were asked to complete a risk-taking task in the presence of two of their personal friends. Research has shown that adolescents are more likely to use impression management strategies in response to familiar, as opposed to unfamiliar peers (Gardner & Martinko, 1988; McPhee 1996). Given that impression management is related to blunted neural sensitivity (Santesso, Segalowitz, & Schmidt, 2005), it is possible that a reduction in FRN was not observed because participants were less likely to use impression management strategies in the presence of the *unfamiliar* peer. Future research would benefit from examining whether peer familiarity moderates the relationship between peer influence and FRN amplitude. An alternative explanation may concern the ecological validity of the manipulation. Given that everyday group decision-making typically involves friends or familiar peers, laboratory studies that include familiar peers may be more ecologically valid social contexts that more accurately capture the dynamics of group decision-making outside of the laboratory (Vidmar, 1970; Wallach & Kogan, 1965; Yinon et al., 1975). Thus, it is possible that FRN was not blunted in the current study

[7] We conducted all analyses that used FRN as the dependent variable using only the males in the current sample. Results were identical to the FRN data presented with both males and females. Thus, it is possible that the effects Segalowitz et al. (2012) observed are more of a function of age, rather than gender.

because of the relative lack of ecological validity involved in using unfamiliar peers during the risk-taking task.

In terms of between-subjects effects, high externalizing participants took significantly more risks than low externalizing participants. These results are not surprising given the phenomenological overlap between RTB and externalizing behavior. Indeed, previous research has observed similar patterns (Hunt, Hopko, Bare, & Robinson, 2005). There was an absence of significant between-subjects effects for all of the genetic moderators of the dependent variables. In other words, no significant differences were observed across those considered to be at high and low genetic risk for behavioral disinhibition (across various levels of genetic risk – from distal to proximal) for any of the dependent variables. Previous research has shown that externalizing behavior is genetically influenced by familial factors and dopaminergic polymorphisms (Burt & Mikolajewski, 2008; Dreber et al., 2009; Golimbet et al., 2007; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; van der Zwaluw et al., 2011). It is possible that the current study may underpowered to detect the small genetic effects of dopaminergic genes and family history on RTB. Indeed, the effects of individual polymorphisms and family history on complex phenotypes are often small and require large sample sizes (Kendler, Davis, & Kessler, 1997; Plomin, Haworth, & Davis, 2009). Last, no significant interaction effects for any of the moderators were observed, such that the change in RTB from the solitary to the peer-influenced condition was not significantly different for those at high and low phenotypic and (proximal and distal) genetic risk. These (non-significant) findings were also observed for the FRN.

It is important to consider these findings in the context of four important methodological limitations. The first limitation concerns the system of genes examined. While there is clear support for the role of dopaminergic polymorphisms in externalizing behavior, it is less clear

what impact these genes have on susceptibility to peer influence. It is possible that genetic polymorphisms within other genetic systems may play a more integral role in susceptibility to peer influence. For instance, genes within the serotonergic system are associated with social affiliative behavior, social status, and behavioral disinhibition (Insel & Winslow, 1998; Kalueff, Olivier, Nonkes, & Homberg, 2010; Young & Leyton, 2002). One study found that carriers of the short version of the serotonin transporter gene (5-HTTLPR) had reduced risk taking and increased responsiveness to social evaluation (Crişan et al., 2009). Further, research has shown that administration of selective serotonin receptor inhibitors that act on receptors associated with variations in serotonergic genes increase social affiliative behavior (Knutson et al., 2014). Thus, it is possible that genes within the serotonergic system interact with peer influence to predict RTB and the broader construct of behavioral disinhibition. Future studies would benefit from examining whether genetic polymorphisms across several genetic systems may lend themselves to susceptibility to peer influence during adolescence. Such research would advance our understanding of which systems of genes, and which specific genetic polymorphisms, predispose an individual to susceptibility to peer-influence of behavioral disinhibition.

Second, the current study utilized self-reported family history as a moderator of the relationship between peer influence and RTB. It is possible that these reports do not accurately represent family history status, especially considering that respondents to family history measures are more likely to report higher rates of parental psychopathology if they themselves meet criteria for that psychiatric disorder (Kendler, Silberg, Neale, Kessler, Heath, & Eaves, 1991). Thus, future research that examines the moderating role of family history on peer influence and RTB would benefit from utilizing a multi-informant design. A third limitation concerns the ethnic diversity of the sample. Non-Hispanic European-Americans were

specifically selected in order to reduce the likelihood of population stratification effects on the genotypes examined. However, previous research has shown that non-white participants are significantly more vulnerable to peer influence during risk taking tasks than white participants (Gardner & Steinberg, 2005). Other studies have found that minority adolescents are more likely to engage in sexual risk and delinquent behavior (Blum et al., 2000; Hawkins, Laub, & Lauritsen, 1998; Koniak-Griffin & Brecht, 1995; Neumark-Sztainer et al., 1996; Piquero & Buka, 2002 Santelli, Lowry, Brener, & Robin, 2000). Thus, future research would benefit from examining whether genetics moderate the relationship between peer-influence and RTB in a sample of non-white participants. Alternatively, use of a much larger and ethnically diverse sample would further our understanding the moderating role of genetics in the relationship between peer-influence and behavioral disinhibition.

Fourth, and potentially the most important limitation, concerns the age range of the sample selected. Indeed, previous research suggests that, while late adolescents are particularly susceptible to deviant peer influence (Steinberg & Monahan, 2007), genetic influences on *peer group deviance* increase monotonically during adolescence (Kendler, Gardner, Gillespie, Aggen, & Prescott, 2007). In other words, genetic factors become increasingly important for peer group deviance later in adolescence. It is possible for genetic variance to increase as a function of age, as adolescents begin to spend less time with family and more time with friends (Larson & Richards, 1991), and become active agents, as opposed to passive recipients, in their environments. This process, called *active gene-environment correlation*, occurs when exposure to varying environmental conditions is a function of genotype. For instance, an adolescent with a genetic predisposition for behavioral disinhibition might begin to select "risky" peers as they transition into adulthood because of their phenotypic similarity. These peers then afford the

adolescent increased opportunities to express their genetic predisposition for behavioral disinhibition. Given that Kendler et al. (2007) observed an increase in genetic variance for peer group deviance throughout adolescence, it is possible that the relationship between peer influence and RTB is a function of active gene-environment correlation, and not gene-environment interaction.

Other research has utilized longitudinal, genetically informed designs to disentangle the effects of peer group deviance on RTB. For instance, Kendler, Schmitt, Aggen, & Prescott (2008) examined the interaction between peer-group deviance and genetic risk for externalizing behavior in the prediction of alcohol consumption over 5 time points between 8 and 25. Results revealed that genetic risk for externalizing behavior interacted with peer group deviance at ages 12-14, but not age 15-17, 18-21, or 22-25. These results demonstrate that exposure to risky peers augment the impact of genetic risk for externalizing behavior on RTB during early adolescence, but have less of an impact on genetic risk for externalizing behavior during late adolescence. Other research has found similar patterns (Kendler, Jacobson, Myers, & Eaves, 2008). Taken together, these results suggest that deviant peer *selection*, alternatively termed assortative friendship (Rose, 2002), social selection (Patterson, Dishion, & Yoerger, 2000), or the "shopping model" (Dishion, Patterson, & Griesler, 1994), and not risky peer *influence* may be the dominant social process at work during late adolescence in the development of RTB. More specifically, it is likely that the relationship between genetic risk, peer influence, and behavioral disinhibition is a function of active gene-environment correlation during late adolescence, and not gene-environment interaction. Given that genetics interact with peer group deviance to predict behavioral disinhibition in early adolescence, future studies would benefit from examining the impact of dopaminergic genes during this critical period in development. Alternatively, future

studies would benefit from examining which specific genetic polymorphisms predict deviant peer selection.

While the current study is qualified by these important limitations, several implications can be drawn from the set of findings. For instance, it is important to consider that the effect sizes for the peer-manipulation were consistently large across each of the phenotypic, dependent variables. This was also the case for the ERP data. While previous literature has observed similar effects using different manipulations of "risky" peer-influence, designs that seek to optimize the effects of peer-influence may benefit from utilizing the manipulation used herein. Moreover, designs that are methodologically limited and are unable to incorporate familiar peers might utilize the technique illustrated above. The current study also replicates previous findings that deviant peers significantly influence RTB, despite the fact that genetic factors do not moderate this relationship in late adolescence. As such, the current study adds to the extant literature by identifying novel avenues for exploring the relationship between peer influence and RTB on phenotypic, neural, and genetic levels.

Tables and Figures

Table 1. Correlations between dependent variables and moderators.

Variable	EXT[1]	FamHx BD[1]	Genetic Risk[1]	SBART Pumps	PBART Pumps	SBART Pops	PBART Pops	SBART FRN[2]	PBART FRN[2]
EXT[1]	---								
FamHx BD[1]	.14	---							
Genetic Risk[1]	.42***	-.16	---						
SBART Pumps	.30**	.06	-.004	---					
PBART Pumps	.22*	.01	-.10	.53***	---				
SBART Pops	.29**	.10	-.08	.78***	.62***	---			
PBART Pops	.21	-.06	-.1	.42***	.77***	.57***	---		
SBART FRN[2]	.07	.14	.16	.03	-.01	-.04	-.22	---	
PBART FRN[2]	.02	-.10	.13	.03	.10	.11	.14	.31	---

Note: EXT = Externalizing Inventory Total Score; Fam Hx BD = Family History Behavioral Disinhibition; SBART = Solitary BART; PBART = Peer-influenced BART; FRN = Feedback-related Negativity in response to Pops; [1]Median split variables. [2]Excludes outliers and left handed subjects; * $p < .05$, ** $p < .01$, *** $p < .001$

Figure 1: Visual depiction of study procedure

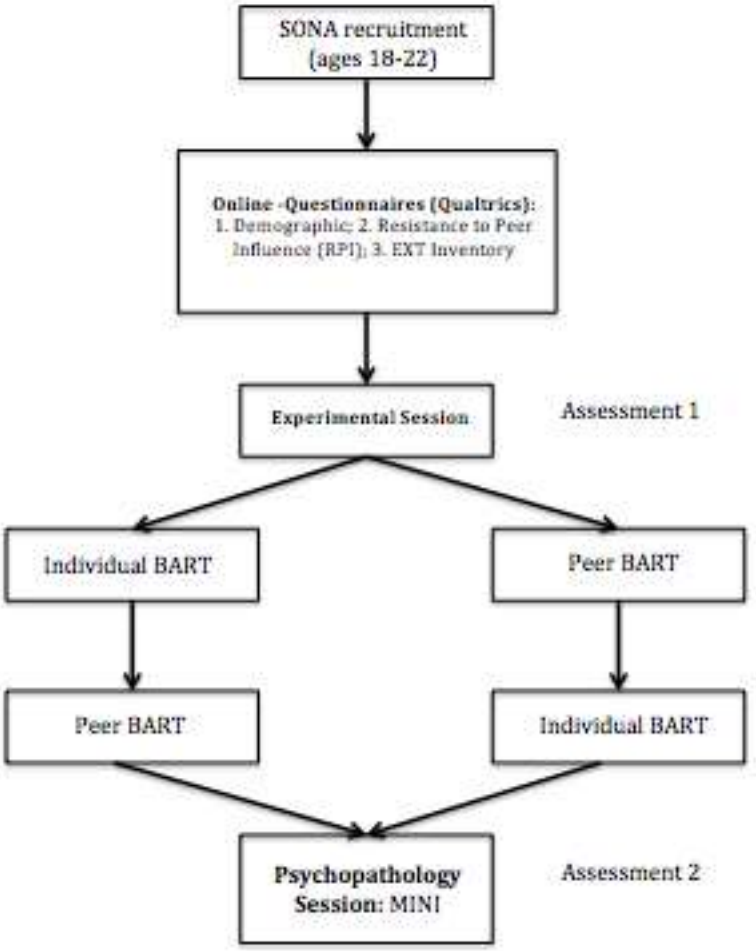
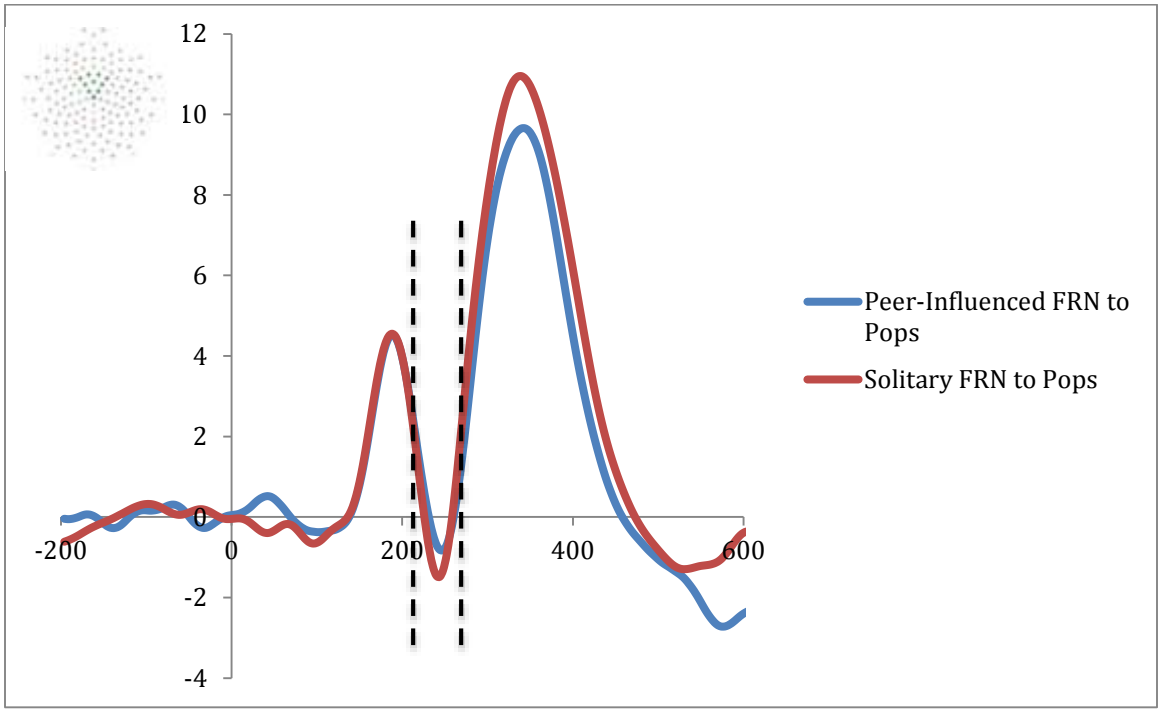


Figure 2: Grand average FRN across solitary and peer-influenced BART.



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Appendix A: Covariate Analyses Results

Previous research indicates that a number of constructs moderate the relationship between peer-influence and RTB, such as gender, childhood socioeconomic status, resistance to peer influence, social anxiety disorder symptoms, and perceived popularity of the confederate (Cillessen & Rose, 2005; Cohen & Prinstein, 2006; Mayeux, Sandstrom, & Cillessen, 2008; Parkhurst & Hopmeyer, 1998; Prinstein, Meade, & Cohen, 2003; Prinstein, 2007; Rancourt & Prinstein, 2010; Steinberg & Monahan, 2007; Sweitzer, Halder, Flory, Craig, Gianaros, Ferrell, & Manuck, 2013) Thus, analyses were also conducted using these covariates. Results are presented below for each dependent variable. As shown, none of the effects were significant. It is possible these results were found due to insufficient power to detect the small effects observed when using covariates.

Appendix B: Covariate Analyses Table

Average Number of Pumps

	Between-Subjects Variable		
	Externalizing	Family History	Genetics
Within-subjects	$F(1, 50) = 1.85$, $p = .18$	$F(1, 50) = 2.45$, $p = .12$	$F(1, 50) = 1.44$, $p = .24$
Between-Subjects	$F(1, 50) = .91$, $p = .35$	$F(1, 50) = .85$, $p = .36$	$F(1, 50) = .68$, $p = .41$
Interaction Effect	$F(1, 50) = .01$, p .94	$F(1, 50) = 1.97$, $p = .35$	$F(1, 50) = .39$, $p = .54$

Number of Pops

	Between-Subjects Variable		
	Externalizing	Family History	Genetics
Within-subjects	$F(1, 50) = .10$, p .75	$F(1, 50) = .25$, $p = .62$	$F(1, 50) = .80$, $p = .38$
Between-Subjects	$F(1, 50) = .77$, $p = .38$	$F(1, 50) = .78$, $p = .38$	$F(1, 50) = .14$, $p = .71$
Interaction Effect	$F(1, 50) = .08$, $p = .78$	$F(1, 50) = 1.38$, $p = .25$	$F(1, 50) = .04$, $p = .84$

FRN

	Between-Subjects Variable		
	Externalizing	Family History	Genetics
Within-subjects	$F(1, 26) = 1.12$, $p = .30$	$F(1, 26) = 2.52$, $p = .12$	$F(1, 26) = .27$, $p = .61$
Between-Subjects	$F(1, 26) = 1.67$, $p = .21$	$F(1, 26) = .92$, $p = .35$	$F(1, 26) = .53$, p .47
Interaction Effect	$F(1, 26) = .33$, $p = .57$	$F(1, 26) = 1.0$, $p = .33$	$F(1, 26) = .72$, $p = .40$

Appendix C: IRB Approval Letter

8/20/2013
Troy Webber, B.A.
Psychology 4202 East Fowler Ave, PCD4118G
Tampa, FL 33620

RE:
Full Board Approval for Initial Review
IRB#: Pro00013874
Title: Genetic Moderation of Phenotypic and Neural Indicators of Peer Influenced Risk-taking Behavior: An Experimental Investigation
Study Approval Period: 8/16/2013 to 8/16/2014

Dear Mr. Webber:

On 8/16/2013, the Institutional Review Board (IRB) reviewed and APPROVED the above application and all documents outlined below.
Approved Item(s):
Protocol Document(s): 17462_2013.07.05_ProtV.2_clean.docx
Consent/Assent Document(s)*: Informed Consent, Ver. 1, 8.1.13 clean.pdf

*Please use only the official IRB stamped informed consent/assent document(s) found under the "Attachments" tab. Please note, these consent/assent document(s) are only valid during the approval period indicated at the top of the form(s).

As the principal investigator of this study, it is your responsibility to conduct this study in accordance with IRB policies and procedures and as approved by the IRB. Any changes to the approved research must be submitted to the IRB for review and approval by an amendment.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638.

Sincerely,

John Schinka, Ph.D.,
Chairperson USF Institutional Review Board