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Do Executive and Reactive Disinhibition Mediate the Effects of Familial Substance Use Disorders on Adolescent Externalizing Outcomes?

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Abstract

The present study examined the potential mediating roles of executive and reactive disinhibition in predicting conduct problems, ADHD symptoms, and substance use among adolescents with and without a family history of substance use disorders. Using data from 247 high-risk adolescents, parents, and grandparents, structural equation modeling indicated that reactive disinhibition, as measured by sensation seeking, mediated the effect of familial drug use disorders on all facets of the adolescent externalizing spectrum. Executive disinhibition, as measured by response disinhibition, spatial short term memory, and "trait" impulsivity, was associated with ADHD symptoms. Moreover, although executive functioning weakness were unrelated to familial substance use disorders, adolescents with familial alcohol use disorders were at risk for "trait" impulsivity marked by a lack of planning. These results illustrate the importance of "unpacking" the broad temperament style of disinhibition and of studying the processes that underlie the commonality among facets of the externalizing spectrum and processes that that predict specific externalizing outcomes.

Introduction

It is well-documented that children of substance-abusing parents are at increased risk for a number of negative outcomes including internalizing and externalizing symptomatology (e.g., Barnard & McKeganey, 2004; Chassin, Pitts, DeLucia, & Todd, 1999; Johnson & Leff, 1999; Nunes, Weissman, Goldstein, McAvay, Beckford, Seracini, Verdeli, & Wickramaratne, 2000; Sher, 1991) and alcohol and drug use disorders (Chassin et al., 1999; Merikangas, Stolar, Stevens, Goulet, Preisig, Fenton et al., 1998; Rounsaville, Kosten, Weissman, Prusoff, Pauls, Anton et al., 1991; Weinberg, Rahdert, Colliver & Glantz, 1998; Zhou, King, & Chassin, 2006). In the case of parental alcoholism, one theoretical mechanism proposed to underlie risk for externalizing disorders among offspring is the "deviance proneness" pathway (Sher, 1991). This empirically supported mechanism (Chartier, Hesselbrock, & Hesselbrock, 2009; Chassin, Pillow, Curran, Molina & Barrera, 1993; Schuckit, Smith, Danko, Trim, Bucholz, Edenberg et al., 2009) proposes that children of alcoholics (COAs) show disinhibited temperament (elevated sensation seeking, impulsivity, lack of constraint, and cognitive dysfunction), which is hypothesized to interact

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with poor parenting to produce poor academic achievement and affiliation with deviant and substance using peers, who are the proximal influence on offspring substance use.

Recent research suggests that disinhibition confers a broad and general risk for externalizing behaviors among offspring of substance abusers (King, Keyes, Malone, Elkins, Legrand, Iacono, & McGue, 2009). However, less is known about potential unique predictors of different indicators of the externalizing "spectrum" (Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002), and whether different forms of disinhibition predict different externalizing outcomes for children of substance abusers. The overall aim of the present study was to test the potential mediating roles of two forms of disinhibition, namely executive and reactive disinhibition in predicting conduct problems, attention deficit hyperactivity disorder (ADHD) symptomatology, and substance use among children with and without a family history of substance use disorders.

Familial Substance Use Disorders and Offspring Disinhibition

Within the large literature on disinhibition, multiple terms such as impulsivity, behavioral disinhibition and behavioral undercontrol have been used loosely and often interchangeably (see Dick, Smith Olausson, Mitchell, Leeman, O'Malley, & Sher, 2010 for review). Although these constructs are related, they are not identical. Moreover, both human and animal data demonstrate that these broad constructs (i.e., disinhibition, impulsivity, and behavioral undercontrol) are not unitary (see Dick, Smith, Olausson, Mitchell, Leeman, O'Malley, & Sher, 2010 for review). For example, Whiteside and Lynam (2001) found a multifaceted model of propensity to rash action, which included lack of premeditation (i.e., acting without forethought), sensation seeking (i.e., seeking out novel or thrilling experiences), lack of perseverance (i.e., difficulty maintaining sustained attention in the context of distraction) and negative urgency (rash responding in the context of negative affect). Further research (Cyders & Smith, 2007) suggested the addition of positive urgency (rash responding in the context of positive affect).

Other theoretical models have suggested that disinhibition depends on two processes, namely executive disinhibition and reactive disinhibition (Blaskey, Harris, & Nigg, 2008; Nigg, 2000). Executive disinhibition refers to failures of "top-down" effortful cognitive processes to suppress, or intentionally control, a response in order to achieve a higher goal (Nigg, 2000; 2003). Executive disinhibition likely involves lack of both premeditation and perseverance in Whiteside and Lynam's (2001) model. In contrast, reactive disinhibition (Nigg, 2003; Nigg, 2006), refers to "bottom-up" processes with dysregulations in behavior in the context of novel or affect-laden situations. Both positive and negative urgency as well as sensation seeking might reflect reactive disinhibition. Similar distinctions are represented in models of "hot" and "cold" cognition (Kerr & Zelazo, 2004; Potenza & De Wit, 2010; Rubia, in press), models of "motor impulsivity" and "choice impulsivity" (Urcelay & Dalley, in press), and in Steinberg's (2010) dual systems model of adolescent risk taking in which impulsivity (an indicator of executive disinhibition) and sensation seeking (an indicator of reactive disinhibition) follow different developmental courses.

Although it is well-documented that children of substance use disordered parents are at-risk for disinhibition (e.g., Edwards, Leonard & Das Eiden, 2001; Sher, Walitzer, Wood,& Brent, 1991; King et al., 2009; Tarter, 1988), it is unclear whether familial substance use disorder confers risk for both executive and reactive disinhibition. Some previous studies have shown that familial substance use disorder is associated with executive disinhibition in the form of poor executive functioning among offspring (Aytaclar, Tarter, Kirisci, & Lu, 1999; Giancola, Moss, Martin, Kirisci, & Tarter, 1996; Harden & Pihl, 1995; Nigg, Glass, Wong, Poon, Jester, Fitzgerald, Puttler, Adams, & Zucker, 2004; Peterson, Finn, & Pihl, 1992). However, findings are not consistent. For example, Corral and colleagues found

familial alcoholism effects on working memory but not on Wisconsin Card Sort performance (Corral, Holguín, & Cadaveira, 1999), and found the opposite pattern in a follow-up assessment (Corral, Holguín, & Cadaveira, 2003). Nigg and colleagues (2004) found that boys from families with co-morbid parental alcoholism and antisocial personality disorder did not differ from controls in their executive functioning. Rather, boys with alcoholic but non-antisocial parents had the poorest executive functioning. Moreover, numerous studies have failed to find a relation between executive functioning deficits and familial substance use disorders (Bates & Pandina, 1992; Bauer & Hesselbrock, 1999; Schafer et al., 1991; Schuckit, Butters, Lyn, & Irwin, 1987; Spadoni, Norman, Schweinsburg, & Tapert, 2008; Stevens, Kaplan, & Hesselbrock, 2003; Wiers, Gunning, & Sergeant, 1998; Wilson & Nagoshi, 1988). There are several possible explanations for these inconsistencies including the widely varying measurement of executive functioning (Nigg et al., 2004), inconsistencies in testing the role of parental co-morbidities, and small sample sizes.

Disinhibition and the Externalizing Spectrum

Executive and reactive disinhibition have both been related to externalizing outcomes such as ADHD, conduct problems, and substance use, or what has been called the externalizing spectrum (Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002). These externalizing disorders are frequently co-morbid and there is evidence that the common externalizing factor is highly heritable (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Kruger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). However, in spite of the high co-morbidity among facets of the externalizing spectrum, there is also evidence for distinction among facets. For instance, Farmer and colleagues (2009) found support for a two-factor model of externalizing behavior in which one factor was characterized by ADHD and ODD symptoms such as inattention, intrusion, impulsivity, and hyperactivity and the second factor was characterized by a general rule-breaking tendency and included symptoms of conduct disorder, antisocial behavior, and substance abuse. Moreover, studies have shown that after controlling for conduct problems, the relation between ADHD and substance use disappears (see Flory & Lynam, 2003 for a review). Taken together these findings suggest that there may be a stronger common diathesis for conduct problems and substance use disorders than for ADHD and substance use disorders.

Moreover, Nigg (2003) proposed that ADHD results from a primary deficit in “top-down” executive inhibition and a secondary weaker deficit in reactive “bottom up” processes, whereas conduct disorder results from a primary reactive disinhibitory process and a secondary weaker executive disinhibition process. Multiple pathway models of ADHD similarly implicate both executive and reactive disinhibition (i.e., Sonuga-Barke, 2003; 2005; Urcelay & Dalley, in press), although the relative importance of reactive disinhibition is still unclear (Blaskey et al., 2008; Wahlstedt, Thorell, & Bohlin, 2008). With regards to conduct problems and substance use disorders, there is empirical support for a reactive disinhibitory pathway (Finn, Mazas, Justus, & Steinmetz, 2002; Herba, Tranah, Rubia & Yule, 2006; Sher, Bartholow & Wood, 2000; Urcelay & Dalley, in press; van Goozen, Cohen-Keetenis, Snoek, Matthys, Swaab-Barneveld, & van Engeland, 2004). However, evidence for an executive disinhibition pathway to conduct problems and substance abuse is less consistent. Specifically, a number of researchers have failed to find a link between executive functioning deficits and conduct problems and substance use (i.e., Giancola & Parker, 2001; Herba, Tranah, Rubia, & Yule, 2006; Oosterlaan, Scheres, & Sergeant, 2005; Nigg, Wong, Martel, Jester, Puttler, Glass, et al., 2006; van Goozen et al., 2004; Wiers et al., 1998). Nigg and colleagues (2006) found that poor performance on the stop task, a measure of executive disinhibition, predicted substance use among adolescents but performance on set-shifting, working memory, and additional neuro-cognitive measures did not.

In addition to theories of direct effects of executive and reactive disinhibition on substance use, Finn (2002) proposed the cognitive-motivational theory (CMT), an interactive model in which working memory capacity moderates the effect of novelty seeking to influence decision making and alcohol use. Specifically, high novelty seeking individuals find rewards to be more salient than punishments and, when they also have low working memory capacity, they are at risk for disinhibition because of difficulty in keeping less salient punishment cues in mind (Finn, Mazas, Justus, & Steinmetz, 2002). This disinhibition then increases risk for alcohol use and problems (Chassin, Pitts, DeLucia, & Todd, 1999). Therefore, the CMT suggests a need to consider the interaction between executive and reactive disinhibition rather than just their individual contributions. Dual process models propose similar interactions between controlled and automatic regulatory processes (Hofman et al., 2009; Grenard et al., 2008).

Taken together, these studies provide consistent support for an executive disinhibitory pathway to ADHD symptomatology and for a reactive disinhibitory pathway to conduct problems and substance use/abuse. In addition, reactive disinhibition may represent a weaker secondary path to ADHD symptoms and executive disinhibition may represent a weaker secondary path to conduct problems and substance use although findings remain unclear. The current study tested whether indicators of executive and reactive disinhibition predicted distinct facets of the externalizing spectrum among adolescents, namely ADHD symptoms, conduct problems, and substance use. Moreover, we tested whether these indicators mediated the effects of familial alcohol and drug disorders on externalizing outcomes among adolescent offspring. Finally, we tested interactions between executive and reactive disinhibition on externalizing outcomes.

Specificity of the Effects of Familial Alcohol Use Disorder

Previous research clearly indicates that individuals with familial alcohol use disorder (AUD) are at heightened risk for a disinhibited temperament (Sher, Grekin, & Williams, 2005), and that familial alcoholism risk for disinhibition and externalizing problems broadly shows significant heritability (Elkins, McGue, Malone, & Iacono, 2004; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; King et al., 2009). However, studies of familial alcoholism risk often fail to consider commonly co-occurring familial psychopathology, particularly antisociality and drug use disorders (DUD). Because there are differing personality correlates of alcohol disorders witih and without co-occurring antisocial and drug disorders (McGue, Slutske, & Iacono, 1999) as well as differing parenting and family environments (Jacob & Johnson, 1997; Ohannessian, Hesselbrock, Kramer, Kuperman, Bucholz, Schuckit, & Nurnberger, 2004; Wong, Zucker, Puttler, & Fitzgerald, 1999), the mechanisms that mediate the effects of familial AUD versus drug use disorder (DUD) may differ. Indeed, previous studies suggest that parent antisociality and drug disorders exert unique negative effects, over and above co-occurring parent alcohol disorder on child externalizing symptomatology, substance use, and the parent-child relationship (Chassin, Rogosch, & Barrera, 1991; Loukas, Fitzgerald, Zucker, von Eye, 2001). Interestingly, Elkins and colleagues (2004) demonstrated that parental drug disorders had a unique effect on offspring constraint, above and beyond parental alcohol disorders, suggesting that an offspring temperamental style marked by low constraint may be most accurately attributable to familial drug use disorder, that often co-occurs with AUDs, rather than to AUDs in absence of DUDs. Therefore, the present study tested whether familial alcohol and drug use disorder and parental antisocial behavior exerted unique effects on offspring reactive and executive disinhibition and on externalizing outcomes.

In summary, the primary aim of the present study was to test whether executive and reactive disinhibition differentially mediated the influence of familial substance use disorders on three adolescent outcomes from the externalizing spectrum: ADHD symptoms, conduct

problems, and substance use while also examining the effects of familial alcoholism in the context of co-occurring DUDs and antisociality

Method

Participants

Participants were drawn from Wave 6 of a larger, ongoing longitudinal study of familial alcoholism across three generations (e.g. Chassin et al., 1991). There were three annual waves of data collection and three additional follow-ups separated by five years. At Wave 1 (1988), the total sample consisted of 454 adolescents (generation 2; “G2s”) and their parents (generation 1; “G1s”). 246 of these adolescents had at least one biological alcoholic parent who was also a custodial parent (COAs), and the remaining 208 adolescents were demographically matched controls without an alcoholic parent (matched on adolescent age, family composition, ethnicity, and socioeconomic status). Beginning at Wave 4 (18–25 years of age), G2 biological siblings within the same age range of the original G2s were also interviewed. At Wave 6, we conducted interviews with their children (generation 3; “G3s”). Sample retention has averaged more than 90% since the advent of the study.

The current analyses used Wave 6 data from a subsample of 247 G3 children who were at least 11.5 years old and had complete data on the three measures of executive disinhibition. We selected those who were at least 11.5 years of age to be appropriate for the substance use outcome. Of the 289 excluded G3s, 206 were excluded because they were younger than 11.5 years old, and 83 were excluded because they were not interviewed in-person and were thus missing data on the cognitive tasks measuring executive disinhibition. Of the G3s included in our analyses, approximately 50.0% were male, 50.0% were non-Hispanic Caucasian, 36.6% were Hispanic, and 13.4% identified themselves as American Indian or some other race/ethnicity. 50.8% of adolescents had at least one biological parent with a lifetime alcohol disorder and 41.0% had at least one biological parent with a lifetime drug disorder (as defined by DSM-IV criteria for abuse or dependence). Of the 41.0% of adolescents with at least one biological drug disordered parent, 79.0% also had at least one biological alcohol disordered parent.

Analyses (chi-square and t-tests) comparing the included and excluded G3 participants on demographic and study variables indicated that included participants were significantly older (by design), scored lower on the IQ measure, were more likely to have used substances, and reported more symptoms of ADHD, higher levels of impulsivity and conduct problems. Moreover, included participants demonstrated stronger performance on all three cognitive executive disinhibition measures. There were no significant differences between included and excluded participants in gender, family structure, parental alcohol and drug disorder status, family history density of alcohol and drug disorders, or sensation seeking.

Recruitment

The original recruitment is described in detail elsewhere (Chassin, Barrera, Bech, and Kossak-Fuller, 1992). COA families were originally recruited using court records, health maintenance organization questionnaires, and community telephone screening. Selection criteria were a child between the ages of 10.5 and 15.5 years, Arizona residence, Hispanic or non-Hispanic Caucasian ethnicity, English-speaking, and no cognitive limitations that would preclude interview. Direct interview was used to confirm that a biological G1 parent met Diagnostic and Statistical Manual of Mental Disorders—Third Edition (DSM-III; American Psychiatric Association, 1980) criteria for lifetime alcohol abuse or dependence. The resulting alcoholic sample had rates of other psychopathology similar to rates reported for a

community-dwelling alcoholic sample (Helzer & Pryzbeck, 1988). However, those who refused participation were more likely to be Hispanic, suggesting some caution in generalization. Reverse directories were used to locate non-alcoholic families in the same neighborhood. Controls were screened and recruited via telephone interviews to match alcoholic families in ethnicity, family structure, age (within 1 year), and socioeconomic status (using the property value code from the reverse directory). Interview data confirmed that neither biological parent met DSM-III criteria (or Family History Research Diagnostic Criteria [FH-RDC]; Endicott, Andreason, & Spitzer, 1975) for lifetime alcohol abuse or dependence.

Procedure

At all waves, computer-assisted interviews were conducted either at the family's residence or at the Arizona State University campus. Parents provided informed consent and adolescents provided assent. To encourage self-disclosure, family members were interviewed simultaneously in separate rooms and a Department of Health and Human Services Certificate of Confidentiality was used to emphasize confidentiality. To maximize privacy, participants could enter their responses on the keyboard rather than verbally.

Measures

Family structure—G3s were categorized as living either in a two-parent home (regardless of the biological relation of the child to the parent, 74.8%) or any other living situation.

G3 adolescent intelligence—*G3* adolescents completed the Kaufman Brief Intelligence Test (K-BIT, Kaufman & Kaufman, 1990). The K-BIT score was a composite of the standard scores of two subtests: verbal intelligence, which included expressive vocabulary and word definitions, and nonverbal intelligence, which included a section of Matrices. The mean IQ was 102.52 (*SD* =11.14).

G3 adolescent family history density of alcohol and drug use disorder—Family history density (FHD) scores were calculated for each G3 adolescent participant based upon the lifetime history of their biological parents (G2s) and grandparents (G1s) using all available direct interview data from the Computerized Diagnostic Interview Schedule (C-DIS; Robins, Cottler, Bucholz, Compton, North, & Rourke, 2000) and family member report using Family History-Research Diagnostic Criteria (FH-RDC; Endicott, Andreason, & Spitzer, 1975). Density scores (separately for alcohol and drug disorders) were calculated using the methods of Stoltenberg and colleagues (1998) and Zucker, Ellis, and Fitzgerald (1994). Parent lifetime diagnoses were weighted by 0.5 and the lifetime disorder variables for grandparents were weighted by 0.25. Each nonalcoholic relative was given a score of 0. The appropriate weighted lifetime disorder variables were summed to create a specific FHD score which ranges in value from 0 to 2. The mean density score for G3 familial alcoholism was .63 (*SD* = .49) and .36 (*SD* = .42), for G3 familial drug disorder density.

G2 parent antisociality—Parents (i.e., G2s) antisocial behavior was assessed with the computerized Diagnostic Interview Schedule (C-DIS; Robins et al., 2000). Because this measure did not assess conduct problems before the age of 15, parent antisociality was calculated using a symptom count of endorsed behaviors since age 15. We considered parents who endorsed 5 or more symptoms to show clinically elevated levels of antisociality (11.8%).[1]

G3 adolescent reactive disinhibition: Sensation seeking—Consistent with Nigg's (2000) conceptualization of reactive disinhibition as including the propensity to seek out novel experiences, we used sensation seeking as a measure of reactive disinhibition.

Because the outcome measures were adolescent-reported, to minimize reporter effects, we used parent report of six items from Zuckerman's 1979 scale (Zuckerman, 1979). Items included "likes being where there is something going on all the time," "would do almost anything on a dare," and "likes to do things on the spur of the moment" (Cronbach alphas of .78 and .87). To minimize missing data, we used the average of mother and father report or either reporter if only one was interviewed (correlation between mother and father reports r=.41, p<.001). We tested our hypothesized one-factor structure using confirmatory factor analysis. Results confirmed the model but indicated that one item did not significantly load ("likes work that has lots of excitement") and this item was dropped. The remaining five items were used as indicators of the latent sensation seeking construct. Factor loadings ranged from .65 to .77.

G3 adolescent executive disinhibition: working memory, spatial short term memory, response inhibition

Working memory—Working memory was assessed with the Letter-Number Sequencing (LNS) subtest from the Weschler Intelligence Scale for Children, Fourth Edition (WISC-IV; Wechsler, 2003). Participants listened to a combination of numbers and letters and then recalled the numbers first in ascending order followed by the letters in alphabetical order. The mean raw score was 18.73 (SD=2.99).[2]

Spatial short term memory—Spatial short term memory was assessed with the Matrix Span Task (MST; Kane, Hambrick, Tuholski, Wilhelm, Payne, & Engle, 2004). MST is a computerized assessment of attention and memory, specifically in the spatial domain. Participants were briefly presented with 4 × 4 matrices in which one cell was highlighted. They attempted to hold in memory the spatial location of the highlighted cell for later recall. Participants were then shown a series of screens each displaying one matrix (series sizes range from 2–5), and were cued to recall and attempt to mark which cells had been filled in that set on a response sheet. There were a total of 20 trials, divided equally into set sizes of 2, 3, 4, and 5 matrices. The test trials were preceded by 3 practices trials with a set size of 2. We followed Conway and colleagues' (2005) guidelines for scoring such that partial credit was awarded if the participant correctly selected some elements in the trial, but incorrectly selected other elements in trial. The mean score was 0.60 (SD=.15).

Response inhibition—Response inhibition was assessed with the Immediate Memory Task (IMT; Dougherty, Marsh, & Mathias, 2002) as a measure of response inhibition. In this computerized task a 5 digit number was presented every second and the participant was asked to press a button only if the current number exactly matched the prior number. On a third of the trials the current number was identical to the prior number (target trials); on a third of the trials the current number was different from the prior number on all 5 digits (foil trials); and on a third of the trials the current number was different from the prior number by only one digit (catch trials or commission errors). Commission errors have been interpreted as a measure of impulsive responding (Dougherty et al., 2002; Dougherty, Mathias, Marsh, & Jagar, 2005) and were used in the current analyses. The mean score was 1.46 (SD=.25).

[1]To select a cut-off of 5 symptoms, we examined early conduct problems among our original target participants at wave 3 (average age 15.2). Those with externalizing symptoms 1 standard deviation above the mean were considered to have conduct problems before age 15. Using this cut-off, 7.8% of original target participants were classified as demonstrating antisocial behavior at wave 6. We then determined that a CDIS cut-off of 5 ASP symptoms yielded approximately the same prevalence of antisocial behavior in the larger sample. Because the gender and family history of alcoholism of original target participants, their siblings, and spouses are relatively similar, we deemed this an appropriate cut-off for the sample.

[2]In the current sample, raw scores were used for analyses because the other 2 measures of executive disinhibition were not scaled for age. Accordingly, age was used as a covariate in the model.

G3 adolescent impulsivity—A measure of G3 adolescent impulsivity was created from parent-report of items adapted from the Junior Eysenck Impulsiveness Questionnaire (J-EIQ; Eysenck, Pearson, Easting, Allsopp, 1985). Exploratory factor analysis was conducted on the original 23 items to extract factors of impulsivity to identify a factor tapping executive disinhibition. The following 6 items were extracted because they mapped most closely onto the construct of the lack of premeditation: generally does and says things without stopping to think; usually thinks carefully before doing anything; mostly speaks before thinking things out; often buys things on impulse; thinks that planning takes the fun out of things; sometimes puts down the first answer that comes into his/her head during a test and forgets to check it later. Confirmatory factor analysis supported this as a single factor: ($\chi^2(9) = 20.91$, $p = .01$, CFI = .98, RMSEA = .06, SRMR = .03) and all factor loadings were significant at $p<.001$. Standardized factor loadings ranged from .58 to .82.

G3 outcomes: conduct problems, ADHD symptoms, and substance use—Because parents are not good reporters of adolescent substance use and to avoid introducing reporter effects, we used adolescent self report of all externalizing outcomes.

Conduct problems—Adolescents self-reported their conduct problems using 28 items from the rule-breaking and aggression subscales of the Youth Self Report externalizing syndrome scale (YSR; Achenbach & Rescorla, 2001). Response options ranged from 0 “Not True” to 2 “Very True.” “Loud” was dropped because of overlap with the ADHD scale and three substance use items were dropped because they overlapped with the substance use outcome (e.g., drinking alcohol without parents’ approval). Cronbach alpha was .89. The mean score was .28 (SD=.25; range 0–1.29)

ADHD symptoms—Adolescents self-reported on 6 items from the DSM-oriented Attention-Deficit Hyperactivity Problems scale of the Youth Self Report (YSR; Achenbach & Rescorla, 2001). Sample items are: fails to finish, can’t concentrate, and talks too much. Response options ranged from 0 “Not True” to 2 “Very True.” “Loud” was dropped because of overlap with the conduct problems outcome. Cronbach alpha was .77. The mean score was .68 (*SD* = .51; range 0–2).

Substance use—Adolescents indicated whether they had ever used alcohol, cigarettes, or marijuana. Adolescents were divided into those who had (29.3%) or had not (70.7%) used substances.

Results

Zero-order correlations among study variables are in Table 1. None of the executive disinhibition measures were significantly correlated with family history density of drug disorder or alcohol disorder (with the exception of Letter Number Sequencing). In contrast, high levels of adolescent sensation seeking were related to denser family histories of drug and alcohol disorder (see Table 1).

As expected, high family history density of AUD was significantly correlated with high family history density of drug disorder. Although family history density of alcoholism (AUD) was significantly correlated with parent antisociality, this correlation was reduced to non-significance when family history density of DUD was controlled. In contrast, family history density of DUD was significantly correlated with parent antisociality, and remained significantly correlated when family history density of AUD was controlled. These results indicate significant overlap in parent antisociality and family history of DUD, and suggest that the relation between parent antisociality and family history of AUD was accounted for family history of DUD.

With regards to the outcomes, familial alcohol and drug use disorders were significantly related to conduct problems and substance use, but not to ADHD symptoms. Adolescent sensation seeking was related to all of the outcomes. ADHD symptoms were related to poor performance on MST and IMT but not LNS. Conduct problems and substance use were not significantly correlated with poor performance on measures of executive disinhibition except for a surprising association between substance use and stronger performance on the IMT. However, once age was controlled, the relation was no longer significant (*pr*= −.03; *p* = n.s).

Model Specification

We tested whether parent-reported sensation-seeking (latent variable) and executive disinhibition (as measured by working memory, spatial short term memory, and response inhibition) mediated the effects of family history densities of alcohol and drug disorders and parent antisocial behavior on three adolescent-reported outcome variables: substance use (dichotomous), ADHD symptoms, and conduct problems. Family history of AUD, DUD and parent antisocial behavior were entered as exogenous predictors, with paths estimated to the 4 mediator variables (sensation seeking and 3 executive disinhibition measures) and all three outcome variables. However because there was no significant unique effects of parent antisociality on the two mediators and three outcome variables, it was trimmed from the final model.

Adolescent age, gender, IQ, and family structure were initially entered as exogenous covariates, with paths estimated to the mediator and outcome variables, and significant paths from covariates to mediators and outcomes were retained in the final model. Preliminary analyses tested all covariate by predictor interactions in predicting mediators and outcomes and also tested mediators by covariate/predictors interactions in predicting outcomes. Because all preliminary interactions were non-significant they were trimmed from the final model. Therefore, in terms of covariates, in our final model, paths were estimated from family structure to substance use, from adolescent IQ to IMT and LNS, and from adolescent age to IMT, LNS, MST, conduct problems, and substance use. Because gender had no significant unique effects and all interactions between gender and predictors/mediators were non-significant, it was trimmed from the final model. Correlations among exogenous variables were modeled and residual variances among the mediators and among the outcome variables were allowed to covary.

Models were tested using MPlus version 6.0 (Muthén & Muthén, 1998–2010). Because the final model included a binary dependent variable (substance use), the weighted least squares estimator with mean and variance adjustments (WLSMV) was used, which computes OLS parameter estimates for continuous outcomes and probit parameter estimates for categorical outcomes. Missing data on endogenous variables were estimated as a function of the observed exogenous variables under the missingness at random assumption (Schafer & Graham, 2002). Because participants were nested within families, standard errors were adjusted for non-independence of observations using the complex function in MPlus.

Model fit was estimated with the robust WLSMV chi-square statistic, comparative fit index (CFI), root mean square error of approximation (RMSEA), and the weighted root mean square residual (WRMR). The robust WLSMV chi-square statistic was developed and shown to have good statistical properties in testing model fit with relatively small sample sizes ($N \approx 200$) and categorical outcomes (Nussbeck, Eid, & Lischetzke, 2006). CFI values greater than .95, RMSEA values less than .06, WRMR values less than .90, and a non-significant chi-square statistic were considered to indicate good model fit (Hu & Bentler, 1999; Yu & Muthén, 2002).

Final Model

The final model (see Figure 1) showed good fit to the data ($\chi^2(61) = 70.25$, $p = .20$, CFI = .98, RMSEA = .02, WRMR = .62). For ease of presentation, Figure 1 does not show non-significant paths. However, all significant and non-significant standardized path coefficients are presented in Table 2.

Results indicated that older adolescents exhibited lower levels of executive disinhibition on all three measures (i.e. IMT, LNS, MST), reported more ADHD symptoms and conduct problems and were also more likely to have used substances. Adolescents with higher IQs showed less executive disinhibition on LNS and IMT. Adolescents from single-parent families were more likely to have used substances than were adolescents from two-parent homes.

Adolescents with higher density of familial drug use disorders showed higher levels of adolescent sensation seeking. Familial drug disorders were unrelated to executive disinhibition and did not have any unique effects on outcomes above and beyond the mediators. Density of familial alcoholism did not uniquely predict sensation seeking or executive disinhibition mediators, nor did it have a unique effect on the outcomes.

An examination of the effects of the mediators on the three outcomes indicated that higher levels of sensation seeking significantly predicted higher levels of ADHD symptoms, higher levels of conduct problems, and higher likelihood of lifetime substance use. Weaker performance on MST and IMT, but not LNS, predicted ADHD symptoms. Executive disinhibition task performance did not uniquely predict conduct problems or substance use, with the exception that poorer IMT performance predicted more conduct problems.

Mediation Analyses

To test the indirect effect of family history density of drug disorders on the three outcome variables via sensation-seeking, 95% asymmetric confidence limits were used (MacKinnon, Lockwood, & Williams, 2004). Confidence intervals that do not include the value zero indicate significant mediation. Results indicated that sensation seeking significantly mediated the effect of family history density of drug disorders on adolescent substance use ($CI._{95}$ = .008; .260), ADHD symptoms ($CI._{95}$ = .004; .105), and conduct problems ($CI._{95}$ = .002; .052).[3]

Moderation Analyses

To examine the interactive effect of executive disinhibition and sensation seeking in predicting adolescent externalizing outcomes, we specified three multiple group models testing sensation seeking as a mediator. Sensation seeking was converted to a manifest variable (i.e. mean score of five items). High and low levels of each of the three executive disinhibition tasks were used as grouping variables in three separate analyses. That is, we tested whether the effects of sensation seeking on the adolescent outcomes were the same across adolescents' high and low scores on each of the tasks measuring executive disinhibition. We used a mean split to dichotomize adolescents as scoring either high or low on each of three tasks. For the LNS and MST tasks, there was no evidence of moderation. That is, there were no significant improvements in model fit when the paths from sensation seeking to the outcome variables were allowed to vary across high/low levels of the LNS or MST tasks, compared to models in which all paths were constrained to be equal across high/

[3]Given the lack of a significant unique effect of family history of alcohol use disorders on any of the outcomes, and the lack of an effect of family history of alcohol use disorders on sensation seeking, we did not test the indirect effect of family history of alcohol use disorders on the outcomes.

low levels of these tasks. In contrast, performance on the IMT showed evidence of moderation in that there was significant improvement in model fit when the paths from sensation seeking to the outcome variables were allowed to vary for low and high IMT scores ($\Delta \chi^2(2)= 15.07$, $p < .000$). Sensation seeking significantly predicted higher levels of all externalizing outcomes for adolescents who performed poorly on the IMT, but not for adolescents who performed well on the IMT.[4]

Other Operationalizations of Executive Disinhibition

The use of three separate correlated measures of executive disinhibition may have underestimated the effects of executive disinhibition by testing unique effects of each one over the other rather than the effects of what they have in common. Accordingly, we tested a model in which the three executive disinhibition tasks were specified as indicators of a latent variable. The model showed fair fit to the data ($\chi^2(72) = 129.37$, $p <.001$, CFI = .86, RMSEA = .05, WRMR = .99). The pattern of results was the same as the final model presented above with the following differences: 1) the latent variable for executive disinhibition marginally significantly predicted ADHD symptoms (p=.08) and 2) the latent variable for executive disinhibition did not significantly predict CD symptoms.[5]

"Trait" Impulsivity Model

Another possible reason that sensation seeking showed significant effects on all outcomes whereas executive disinhibition did not predict substance use might be shared method variance. That is, both the externalizing outcomes and the sensation seeking mediator were measured by reports about behavior whereas executive disinhibition was measured by cognitive tasks. Although the use of different reporters (parent-reported sensation seeking and adolescent-reported substance use) reduces shared method variance, we tested an additional model using parent reports of adolescent impulsivity in place of the cognitive tasks to indicate executive disinhibition. Model specification was as described above with the following exceptions: 1) parent-reported impulsivity (latent variable) replaced the executive disinhibition mediators in the previous model; 2) adolescent IQ was trimmed from this model because it did not uniquely predict the mediators or outcome variables; 3) the path from adolescent age to parent-reported impulsivity was non-significant and was therefore trimmed from this model. Finally, this model had a larger sample size (n=310) because participants who were not interviewed in person (and thus lacked data on the cognitive tasks) still had data on parent-reported impulsivity. This larger sample might also allow for detection of executive disinhibition effects that would be missed by the cognitive task models presented above.

This model (see Table 3 and Figure 2) also showed good fit to the data ($\chi^2(113) = 144.74$, $p = .02$, CFI = .96, RMSEA = .02, WRMR = .74). For ease of presentation, Figure 2 does not show non-significant paths. The main findings from the cognitive task model were replicated. That is, executive disinhibition (impulsivity) predicted ADHD symptomatology and conduct problems but not substance use and reactive disinhibition (sensation seeking) predicted substance use. There were some differences between this model and the cognitive task model: 1) familial alcohol use disorder uniquely predicted parent-reported impulsivity; 2) parent-reported sensation seeking no longer uniquely predicted ADHD symptoms and conduct problems.[6]

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[4]We also tested moderation following the Aiken and West (1991) method by creating interaction terms using the cross-product of each cognitive task multiplied by sensation seeking. There was no evidence of significant moderation using this method.

[5]Because IQ was correlated with executive disinhibition, we also estimated a model without covarying IQ. The model fit the data well ($\chi^2(52) = 69.81$, $p = .05$, CFI = .95, RMSEA = .04, WRMR = .65) and results for the final model reported above were replicated, with the following differences: 1) high levels of family history of DUD were marginally related to sensation seeking (p=.07); 2) and high levels of family history of AUD significantly predicted poor performance on MST and IMT.

Mediation Analyses

To test the indirect effect of family history density of drug and alcohol disorders on the externalizing outcome variables via sensation-seeking and impulsivity, 95% asymmetric confidence limits were again used (MacKinnon et al., 2004). Confidence intervals that do not include the value zero indicate significant mediation. Consistent with the previous model, results indicated that sensation-seeking significantly mediated the effect of family history density of drug disorders on adolescent substance use ($CI._{95}$ = .004; .275). Interestingly, parent-reported impulsivity significantly mediated the effect of family history of alcohol use disorders on adolescent ADHD symptoms ($CI._{95}$ = .021; .137) and conduct problems ($CI._{95}$ = .008; .052).

Moderation Analyses

We also tested this trait model for a potential sensation seeking by executive disinhibition (i.e., parent-reported impulsivity) interaction. A multi-group model tested whether the effects of parent-reported sensation seeking (specified as a manifest variable) on the three adolescent externalizing outcomes varied across high and low levels of parent-reported impulsivity (mean split). There was not a significant improvement in model fit when the three paths from sensation seeking to the outcome variables were allowed to vary across high and low levels of impulsivity ($\Delta \chi^2(2)= 4.23$, $p = .121$). Therefore, the influences of sensation seeking on the adolescent outcomes did not significantly vary across parent-reported impulsivity.[7]

Discussion

The present study examined executive and reactive disinhibition as mechanisms underlying familial substance use disorder risk for adolescent externalizing outcomes, and tested whether executive and reactive disinhibition differentially predicted different facets of the externalizing spectrum. Our results demonstrated that adolescent reactive disinhibition, as measured by sensation seeking, was a mediator of the influence of familial drug disorders on adolescent substance use (over and above the effects of executive disinhibition). In contrast, executive disinhibition, as measured by three executive functioning tasks, was not predicted by familial alcohol or drug use disorders or parent antisociality (although trait impulsivity was related to familial alcohol use disorder), and executive disinhibition was not a unique predictor of adolescent substance use. Rather, poor spatial short term memory, response disinhibition and trait impulsivity were significant unique predictors of ADHD symptoms, and response disinhibition and trait impulsivity were significant unique predictors of conduct problems. These results provide clear support for Nigg's (2000; 2003) theorized distinction between executive and reactive disinhibition and illustrate the importance of "unpacking" the broad temperament style of "undercontrol." Not only were the two types of disinhibition differentially predicted by familial risk, but they conferred risk differentially to different forms of adolescent externalizing behaviors

Relation of reactive and executive disinhibition to density of familial alcohol and drug disorders

The current findings showed that adolescents with higher levels of reactive disinhibition as measured by sensation seeking also had more dense family histories of drug disorders.

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[6]This trait model was also estimated using the restricted subsample of adolescents who were included in the cognitive task model and showed good fit to the data $\chi^2(113) = 140.27$, $p < .05$, CFI = .96, RMSEA = .03, WRMR = .71. The pattern of results was the same as with the larger sample except that sensation seeking marginally significantly predicted substance use (p=.07).

[7]We also tested for moderation using the Aiken and West (1991) method by computing the cross-product of sensation seeking and impulsivity. There was no evidence of significant moderation using this method.

However, although sensation seeking was related to familial drug disorders it was not uniquely related to density of familial alcoholism. In interpreting this finding, it is important to remember that adolescents with more drug disordered relatives are also more likely to have denser histories of familial alcoholism, whereas adolescents with alcohol disordered relatives do not necessarily have as high a likelihood of having drug disordered relatives. This asymmetry in the overlap of alcohol and drug disorders seen in the current study is also evident in epidemiological data, which show that individuals with drug disorders are more likely to have comorbid alcohol disorders whereas individuals with alcohol disorders are less likely to show comorbid drug disorders (Kessler, 2007). In addition, high familial drug disorder density likely reflects a more severe family history risk (including drug disorders, alcohol disorders, and antisociality) than does high familial alcohol disorder density (Dick et al., 2007). Interestingly, previous research also indicates that alcohol disorder comorbid with drug disorder is more likely to result from pathways related to lack of constraint whereas alcohol disorder in the absence of drug disorder is more likely to reflect pathways involving negative emotionality or physiological responses to alcohol (McGue et al., 1999; Chassin et al., 2004; Grekin, Sher, & Wood, 2006). Thus, our finding that adolescents with high familial drug disorder densities show high levels of reactive disinhibition may reflect risk pathways involving lack of constraint. Moreover, our finding that adolescents with particularly severe familial risk show elevated reactive disinhibition is consistent with the findings of Nigg and colleagues (2004) who found that sons of *antisocial* alcoholic parents, but not sons of non-antisocial alcoholic parents, exhibited deficits in regulatory control in the context of rewards.

In contrast, (with the exception of a relation between familial alcohol density and trait impulsivity), our results did not show a relation between executive disinhibition and either familial alcohol or drug disorders. Specifically, performance on cognitive tasks reflecting executive functions was not predicted by familial alcohol or drug disorders, or parental antisociality. Interestingly, when IQ was not controlled, familial alcoholism was related to executive functioning tasks, suggesting that relations between familial alcoholism and executive functioning may be spurious or better accounted for by the third variable of IQ. In fact, research findings concerning executive functioning deficits associated with familial alcoholism risk is decidedly mixed. Consistent with our findings, a number of researchers have also failed to find familial alcoholism effects on executive functioning (i.e., Spadoni et al., 2008; Stevens et al., 2003; Wiers et al., 1998). However, others have found evidence of an association (i.e. Corral et al, 1999; 2003; Nigg et al., 2004). A possible explanation for the differing findings may be differences in tasks designed to tap executive functioning. For example, Nigg et al. (2004) found differences between sons of non-antisocial alcoholics and controls on tasks designed to tap response suppression and response regulation, but not on tasks tapping set shifting and working memory and visual working memory planning which may be more analogous to our measures of executive disinhibition.

Although familial alcoholism was not related to executive disinhibition as measured by cognitive tasks, familial alcoholism was related to “trait” impulsivity. Many other studies have also noted differences in findings produced by “task” and “trait” measures (see Dick et al., 2010 for a recent review). Our results may suggest that although adolescents with familial alcohol and drug use disorders may not be at risk for specific executive functioning weakness, those with familial alcohol use disorders may be at risk for a broader temperamental style marked by a lack of planning or premeditation. Alternatively, however, given that trait impulsivity was parent-reported, the relation found between familial alcoholism and offspring trait impulsivity might also reflect shared measurement variance.

Relations of executive and reactive disinhibition to adolescent externalizing outcomes

The central finding of the current study was that executive and reactive disinhibition differentially predicted different facets of the externalizing spectrum. Most important, as predicted, adolescent substance use was related only to reactive disinhibition, and not to executive disinhibition. This was true regardless of whether executive disinhibition was operationalized as performance on cognitive tasks or as trait impulsivity, so that the findings were robust and could not be accounted for by shared method variance. Thus, our results clearly indicate a reward-driven risk pathway, rather than an executive dysfunction pathway, to adolescent substance use. This finding is consistent with a number of recent studies demonstrating the association between reward response weakness, or sensation seeking, and substance use (e.g., Castellanos-Ryan, Rubia, & Conrod, 2010; Cyders, Flory, Rainer, & Smith, 2009; Magid, Maclean, & Colder, 2007; Urcelay & Dalley, in press; Whiteside & Lynam, 2003; Whiteside, Lynam, Miller, & Reynolds, 2005). For example, consistent with our findings, Cyders and colleagues (2009) found that sensation seeking, but not planning or perseverance, was associated with college student drinking. Similarly, Castellanos-Ryan and colleagues (2010) found that response disinhibition among adolescents was associated with conduct problems, whereas “hot” reward response weakness was associated with binge drinking. Our results corroborate their claims that adolescents who become dysregulated in the context of “hot” rewards or motivation are at risk for substance use and extend this work to suggest that “cold” executive functioning weakness, may not be a primary pathway to adolescent substance use. Therefore, adolescent substance use may be better conceptualized as a result of difficulty “putting on the breaks,” when rewards are present, rather than difficulty with thoughtful planning or persistence in working toward a goal. Given that substance use is associated with physiological responses that may be temporarily rewarding, it makes sense that individuals who demonstrate disinhibition in the context of rewards may be particularly vulnerable to substance use.

Steinberg’s (2010) dual systems model of adolescent risk taking describes the differential development of reward sensitivity and cognitive control such that around the time of puberty, adolescents experience a dramatic increase in reward sensitivity, whereas the development of impulse control appears to be a more gradual process occurring slowly throughout adolescence and emerging adulthood. Consequently, risky behavior may increase during adolescence because of the gap between elevated reward sensitivity and immature cognitive control. Moreover, because peer social interactions are particularly rewarding for adolescents, they may show most risk-taking in peer contexts (Steinberg, 2010). Because adolescent substance use typically occurs in a peer context, disruptions of adolescent self-regulation in highly rewarding contexts may be particularly relevant for adolescent substance use.

With regards to the relations between disinhibition and ADHD symptoms, ADHD symptoms were predicted by trait impulsivity, poor spatial working memory, and response disinhibition and inconsistently predicted by sensation seeking (in the model including cognitive tasks but not in the model including trait impulsivity). Our findings that executive functioning and trait impulsivity predict ADHD symptoms are consistent with a large body of research showing executive disinhibition deficits in ADHD (e.g., Barkley, 1997; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Also, the relations between ADHD and sensation seeking are consistent with some research that suggests a weaker secondary pathway of reactive disinhibition to ADHD (Nigg, 2003; Sonuga-Barke, 2003; 2005; Urcelay & Dalley, in press). Because studies do not consistently find relations between ADHD and sensation seeking (see e.g., Blaskey et al., 2008), more work is needed to understand the role of sensation seeking, or “bottom-up” reactive processes, in the development of ADHD.

Our results also showed that conduct problems were associated with response disinhibition and trait impulsivity, and inconsistently associated with sensation seeking. That is, sensation seeking predicted conduct problems in the cognitive task model but not when trait impulsivity was considered. Although it is possible that trait impulsivity is a more salient underlying pathway to conduct problems than is sensation seeking, future research is necessary to more fully clarify these mechanisms given the inconsistent findings for sensation seeking.

Further research is also needed to understand the extent to which ADHD and conduct problems share a common diathesis in terms of executive and reactive disinhibition, or are affected to different degrees by executive and reactive disinhibition, or differ in their diatheses. Of course, other influences may also determine whether an individual develops ADHD or conduct problems or both. For example, some researchers have suggested that ADHD and conduct disorder share a common diathesis but that the quality of parenting influences the nature of the resulting symptomatology(see Beauchaine, Hinshaw, & Pang, 2010 for a review). Further study to understand the overlap and diatheses between ADHD and conduct problems, and the heterogeneity of each is warranted, especially with regards to dimensions of disinhibition.

We found only limited evidence for Finn's (2002) cognitive-motivational theory, which posits that working memory interacts with novelty seeking to influence alcohol use. Our findings indicated that for adolescents with response inhibition weakness, sensation seeking was a significant predictor of all three facets of the adolescent externalizing spectrum (i.e., ADHD symptoms, conduct problems, and substance use) whereas for adolescents with strong response inhibition, sensation seeking did not significantly predict outcomes. Other substance use researchers (e.g., Grenard et al., 2008) also suggest that strong executive functioning allows for "cool" reflective controlled processes to over-ride the effects of reactive disinhibition on substance use outcomes. The lack of broader support for these interaction effects in the current data may be due to statistical limitations of the multiple group model tests which dichotomize task performance as well as the low statistical power associated with interaction tests.

Executive and reactive disinhibition as mediators of familial risk for adolescent externalizing outcomes

We found that reactive disinhibition, but not executive disinhibition, mediated the influence of familial drug disorder risk on adolescent externalizing outcomes, supporting a novelty-driven disinhibitory pathway of familial drug disorder risk. However, our findings are not definitive about whether this reactive disinhibitory pathway (indicated by sensation seeking) raises risk broadly for all externalizing outcomes or only for substance use. When executive disinhibition was operationalized by cognitive task performance, our results are consistent with sensation-seeking as a broad diathesis of externalizing spectrum risk (Iacono, Malone, & McGue, 2008). However, when executive disinhibition was conceptualized as parent-reported impulsivity, sensation seeking predicted only substance use outcomes, supporting a much more specific pathway underlying familial risk for adolescent substance use outcomes. What is clear in both cases, however, is that reactive disinhibition and not executive disinhibition, predicted adolescent substance use. Thus, in terms of mediators of intergenerational transmission of risk for adolescent substance use, it may be a propensity for disinhibition in the "hot" context of novelty rather than a "cool" executive functioning weakness that is the pathway of risk.

Although the present study makes new contributions to the literature on the risk mechanisms underlying the adolescent externalizing spectrum, several limitations should be noted. First, our data are cross-sectional. Although reverse directions of effect are unlikely (e.g.,

adolescent cognitive task performance is not likely to influence family history density of substance use disorders and the low levels of substance use in these young adolescents are not likely to substantially influence their executive and reactive disinhibition), longitudinal data are important to further our understanding of these risk pathways. Moreover, different forms of disinhibition may be important for different stages of substance use from initiation to maintenance, the development of clinical substance use disorder or cessation (Urcelay & Dalley, in press). Also, we did not measure clinically diagnosed parent or grandparent antisocial personality disorder, and relied on a proxy measure which might underestimate the unique effects of familial antisociality. Finally, it would be useful to have additional measures of reactive disinhibition.

In summary, the current study was the first to examine both executive and reactive disinhibition, in one model, as potential mechanisms underlying familial substance use disorder risk for multiple facets of the adolescent externalizing spectrum. Our results provide clear evidence for Nigg's (2000; 2003) proposed distinction between executive and reactive disinhibition as well as for similar models that distinguish between "cold" and "hot" failures of self-regulation (e.g., Kerr & Zelazo, 2004; Potenza & De Wit, 2010; Rubia, in press; Steinberg, 2010). Executive and reactive disinhibition were not only differentially predicted by familial risk, but they conferred risk differentially to different facets of the externalizing spectrum. Specifically, reactive disinhibition significantly mediated the effect of familial drug use disorder on adolescent substance use. In contrast, executive disinhibition, as measured by response disinhibition, spatial short-term memory, and working memory was not associated with familial substance use disorders, and did not predict adolescent substance use, although executive disinhibition was related to ADHD and conduct problems. Moreover, executive disinhibition, as measured by "trait" impulsivity or a lack of planning or premeditation, was associated with familial alcohol use disorder, adolescent ADHD symptoms, and conduct problems, but not with adolescent substance use. These findings illustrate the importance of "unpacking" the broad construct of disinhibition and of studying the processes that underlie the commonality among facets of the externalizing spectrum as well as processes that that predict specific externalizing outcomes.

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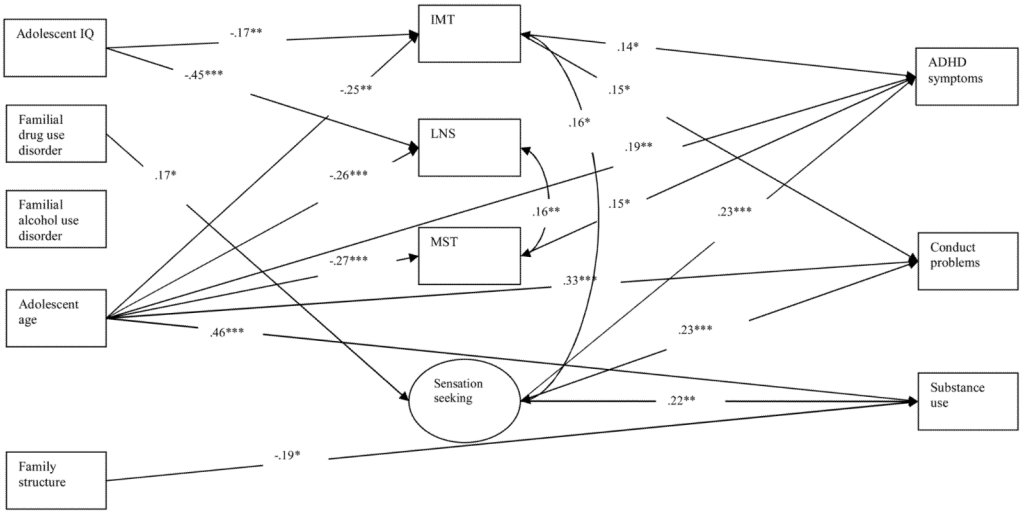

Figure 1.

Results of final structural equation model-task model

Notes: *p<.05, **p<.01, ***p<.001; IMT=Immediate Memory Task; LNS=Letter Number Sequencing; MST=Matrix Span Task; Family structure is coded 1=two-parent home and 0=single parent home or other living situation; IMT and MST was coded such that high scores indicate poor performance. Sensation seeking was coded such that high scores indicate high levels of sensation seeking; High scores on ADHD symptoms and conduct problems indicate more endorsed symptoms.

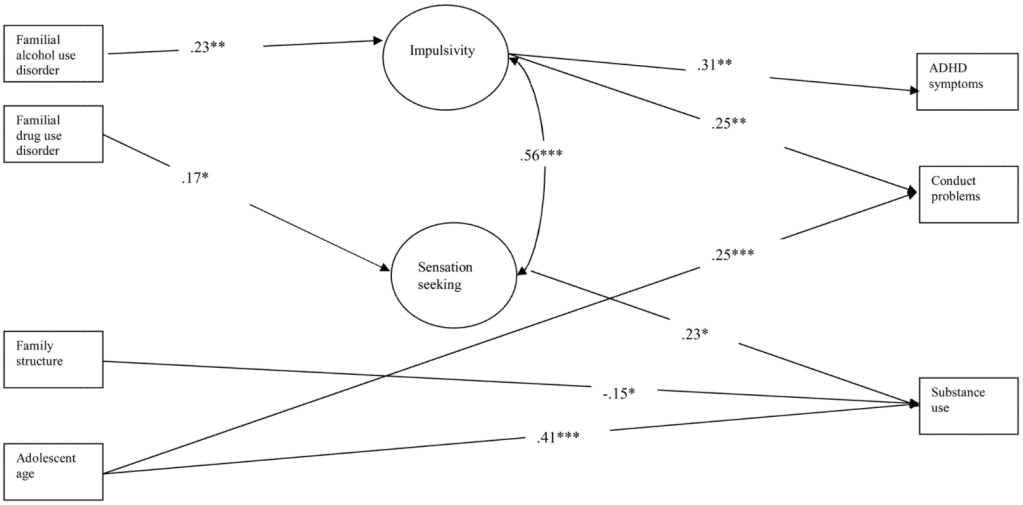

Figure 2.

Results of "trait" impulsivity model

Notes: *p<.05, **p<.01, ***p<.001; Family structure is coded 1=two-parent home and 0=single parent home or other living situation; Impulsivity was coded such that high scores indicate high levels of impulsivity; Sensation seeking was coded such that high scores indicate high levels of sensation seeking; High scores on ADHD symptoms and conduct problems indicate more endorsed symptoms.

Table 1

Zero-order correlations among study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. Gender	--														
2. Adolescent age	.01	--													
3. Family structure	.13*	−.07	--												
4. Intelligence	−.13*	−.11	.10	--											
5. FHD-alcohol	.03	.04	−.15*	−.14*	--										
6. FHD-drug	−.01	−.06	−.17**	−.02	.47***	--									
7. Parent antisociality	−.05	.02	.05	−.05	.20**	.23***	--								
8. Sensation seeking	.06	.12*	.03	−.08	.16*	.18**	.07	--							
9. MST	−.23***	−.19**	−.10	−.44***	.11	.04	.04	.05	--						
10. LNS	.12	−.19**	−.04	−.45***	.14*	.05	.04	.07	.38***	--					
11. IMT	.22***	−.25**	.09	−.15*	.07	−.05	−.05	.10	.12*	.21**	--				
12. Impulsivity	.15*	.04	−.04	−.21**	.24***	.20**	.02	.55***	.11	.18**	.19**	--			
13. ADHD symptoms	−.05	.11	−.05	−.09	.09	.07	.06	.27***	.15*	.10	.13*	.34***	--		
14. Substance Use	−.03	.40***	−.21**	−.08	.20**	.17**	.12	.26***	.04	−.08	−.13*	.19**	.28***	--	
15. Conduct problems	.04	.27***	−.11	−.08	.17**	.18**	.08	.31***	.03	.04	.09	.33***	.66***	.43***	--

Note:

* p<.05,

** p<.01,

*** p<.001. MST=Matrix Span Task; LNS=Letter Number Sequencing; IMT=Immediate Memory Task. Gender was coded 1=male, 0=female; family structure was coded 1=two-parent home, 0=single parent home or other; parent antisociality was coded 0= no diagnosis, 1= diagnosed; substance use was coded 0=non-users, 1=users; higher scores on FHD-alcohol, FHD-drug, ADHD symptoms, sensation seeking and conduct problems indicate greater levels of the construct; higher scores on MST, LNS, and IMT indicate higher levels of executive disinhibition. The mean of the 5 sensation seeking items was used to include sensation seeking in this table. The 5 sensation seeking items were used as indicators of the latent variable in the final model.

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Table 2

Standardized path coefficients for final structural equation model

Predictors	**Mediators**			
	Sensation seeking	**MST**	**LNS**	**IMT**
FHD-alc	.11	.07	.09	.12
FHD-drug	.17*	−.02	−.02	−.12
Adolescent IQ	--	--	−.45***	−.17**
Family structure	--	--	--	--
Adolescent age	--	−.27***	−.27***	−.25***

Predictors/Mediators	**Outcomes**		
	ADHD symptoms	**Conduct problems**	**Substance use**
FHD-alc	−.01	.04	.13
FHD-drug	.03	.12	.12
Adolescent IQ	--	--	--
Family structure	--	--	−.19*
Adolescent age	.18*	.33***	.46***
Sensation seeking	.23**	.23**	.22**
MST	.15*	.04	.12
LNS	.02	.01	−.11
IMT	.14*	.15*	−.03

Note: The standardized path coefficient is reported first, followed by the standard error in parentheses. MST=Matrix Span Task; LNS=Letter Number Sequencing; IMT=Immediate Memory Task;

* p<.05,

** p<.01,

*** p<.001

Table 3

Standardized path coefficients for “trait” impulsivity model

Predictors	Mediators	
	Sensation seeking	Impulsivity
FHD-alc	.12	.23**
FHD-drug	.17*	.08

Predictors/Mediators	Outcomes		
	ADHD symptoms	Conduct problems	Substance use
FHD-alc	−.03	.02	.17†
FHD-drug	.02	.10	.13
Family structure	--	--	−.15*
Adolescent age	--	.25**	.41***
Sensation seeking	.05	.10	.23*
Impulsivity	.31**	.25**	.01

Note: The standardized path coefficient is reported first, followed by the standard error in parentheses.

† p<.06; p<.05,

** p<.01,

*** p<.001