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Anxiety Modulates Preference for Immediate Rewards Among Trait-Impulsive Individuals: A Hierarchical Bayesian Analysis

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Abstract

Trait impulsivity—defined by strong preference for immediate over delayed rewards and difficulties inhibiting prepotent behaviors—is observed in all externalizing disorders, including substance-use disorders. Many laboratory tasks have been developed to identify decision-making mechanisms and correlates of impulsive behavior, but convergence between task measures and self-reports of impulsivity are consistently low. Long-standing theories of personality and decision-making predict that neurally mediated individual differences in sensitivity to (a) reward cues and (b) punishment cues (frustrative nonreward) interact to affect behavior. Such interactions obscure one-to-one correspondences between single personality traits and task performance. We used hierarchical Bayesian analysis in three samples with differing levels of substance use (N = 967) to identify interactive dependencies between trait impulsivity and state anxiety on impulsive decision-making. Our findings reveal how anxiety modulates impulsive decision-making and demonstrate benefits of hierarchical Bayesian analysis over traditional approaches for testing theories of psychopathology spanning levels of analysis.

Keywords

substance use, impulsivity, anxiety, delay discounting, Bayesian statistics, open data

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Impulsivity, defined behaviorally as a preference for immediate over delayed rewards, actions taken without forethought, and difficulties inhibiting prepotent behaviors (Neuhaus & Beauchaine, 2017; Sagvolden, Johansen, Aase, & Russell, 2005), is a highly heritable trait that confers vulnerability to all externalizing spectrum disorders (Beauchaine, Zisner, & Sauder, 2017), including attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, conduct disorder (CD), substance-use disorders (SUDs), and antisocial personality disorder (ASPD). In structural models of adult psychopathology, all of these disorders load on a single, highly heritable latent vulnerability trait (see e.g., Krueger et al., 2002). A similar heritable trait emerges in structural models of child psychopathology, with the exceptions of ASPD and SUDs, given limited opportunity for children to engage in criterion behaviors (Tuvblad, Zheng, Raine, & Baker, 2009). This shared latent vulnerability is often characterized as *trait impulsivity* on the basis of common genetic, neural, cognitive, and behavioral

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processes observed across disorders (e.g., Beauchaine, Zisner, & Sauder, 2017; Gatzke-Kopp et al., 2009; Gizer, Otto, & Ellingson, 2017). Individuals who are highly impulsive early in life—as manifested in the hyperactive-impulsive and combined presentations of ADHD—are at considerable risk for developing more severe forms of externalizing conduct across development (Beauchaine & McNulty, 2013; Beauchaine, Zisner, & Sauder, 2017). Such progression is most likely in contexts of adversity, including family dysfunction (Patterson, Degarmo, & Knutson, 2000), child maltreatment (e.g., Shin, Cook, Morris, McDougle, & Groves, 2016), delinquent peer affiliations (e.g., McGloin & O'Neill Shermer, 2008), and exposure to neighborhood violence and criminality (Lynam et al., 2000; Meier, Slutske, Arndt, & Cadoret, 2008).

Given the high heritability of impulsivity and its associations with concurrent and future externalizing outcomes, many candidate biomarkers and endophenotypes of externalizing liability have been proposed, including neural functions, autonomic responses, and performance on laboratory tasks (e.g., Ersche, Turton, Pradhan, Bullmore, & Robbins, 2010; Foell et al., 2016; Ortiz & Raine, 2004; Patrick et al., 2006). As reviewed elsewhere, biological and behavioral markers could be useful for early identification of vulnerability given sufficient measurement precision (e.g., Beauchaine & Constantino, 2017). Such efforts are challenging, however, because impulsivity, like most human behavioral traits, is distributed continuously in the population and becomes impairing only when expressed at extremes. Accordingly, impulsivity and related constructs, such as self-control, figure prominently in theories of personality (e.g., Corr, 2004; Hampson, 2012). Other literatures link excessive impulsivity to certain mood disorders (Lombardo et al., 2012), personality disorders other than ASPD (McCloskey et al., 2009), and vulnerability to psychopathology more broadly (e.g., Beauchaine, Hinshaw, & Bridge, 2019; Carver & Johnson, 2018). These conceptualizations are consistent with burgeoning efforts to identify transdiagnostic features of mental illness (e.g., Beauchaine, Constantino, & Hayden, 2018; Beauchaine & Hinshaw, 2020; Beauchaine & Thayer, 2015; Robbins, Gillan, Smith, de Wit, & Ersche, 2012). Impulsivity is therefore a construct of considerable interest both as an individual difference and as a marker of vulnerability to psychopathology. In this article, we considered complexities of measuring impulsivity, including possible explanations for low correspondences between self-reports and lab tasks.

Approaches to Measuring Impulsivity

Historically, impulsivity has been measured in many ways, often at different levels of analysis, including self-reports, informant reports, and assorted behavioral/cognitive tasks (for reviews, see Neuhaus & Beauchaine, 2017; Oas, 1985; Rung & Madden, 2018; Vassileva & Conrod, 2019). For example, when assessing clinical levels of impulsivity among children and adolescents, informant reports are commonly used. Such reports show high reliability and strong predictive validity to concurrent and future psychological function (see e.g., Achenbach & Edelbrock, 1991; Beauchaine, Zisner, & Sauder, 2017). Among adults, self-reports are commonly used given ease of administration and similarly strong reliability and predictive validity (e.g., Patton, Stanford, & Barratt, 1995). Note that many adult measures assess multiple facets of impulsivity (Sharma, Markon, & Clark, 2014; Whiteside & Lynam, 2001). For example, the Barratt Impulsiveness Scale (BIS-11) assesses nonplanning, motor, and attentional impulsivity (Patton et al., 1995). High scores on the nonplanning subscale (BIS-NP), which captures preferences for immediate over delayed rewards, have been observed consistently among individuals who abuse substances, including alcohol, nicotine, stimulants, and heroin (Dom, Hulstijn, & Sabbe, 2006).

Self-reports aside, behavioral and cognitive approaches used to assess impulsivity include set-shifting tasks (e.g., Avila, Cuenca, Félix, Parcet, & Miranda, 2004), continuous-performance tasks (e.g., Conners & MHS Staff, 2000), and go/no-go tasks (e.g., Bezdjian, Baker, Lozano, & Raine, 2009). More recently, monetary delay-discounting tasks (DDTs) have gained popularity. DDTs, which we used here, assess how individuals assign value to delayed rewards by presenting them with sequences of choices between smaller magnitude rewards received sooner and larger magnitude rewards received later (e.g., Green & Myerson, 2004). Performance is quantified by individuals' *discounting rates*, which describe how precipitously they discount rewards as a function of increasing time delay to receipt of reward. Steeper discounting rates have been observed among individuals with ADHD, CD, and ASPD and among those who abuse alcohol, nicotine, heroin, and cocaine (e.g., Beauchaine, Ben-David, & Sela, 2017; Bickel & Marsch, 2001; Bobova, Finn, Rickert, & Lucas, 2009; Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005; Petry, 2001; Wilson, Mitchell, Musser, Schmitt, & Nigg, 2010).

Despite frequent use of both self-report and task measures of impulsivity, correspondences between the approaches are usually weak (see Sharma et al., 2014). Meta-analyses have shown average low correlations between multidimensional self-reports and behavioral measures of $r \approx .10$ (Cyders & Coskunpinar, 2011). These low correspondences are attributed to several sources, including low test–retest reliability of behavioral tasks (Cyders & Coskunpinar, 2011; Hedge, Powell, & Sumner, 2017), state dependence of behavioral tasks

relative to self-reports (Cyders & Coskunpinar, 2011; Koff & Lucas, 2011), and failures of behavioral tasks to capture the multidimensional nature of impulsivity (Duckworth & Kern, 2011).

An additional possibility, which we examined here, is that impulsivity is determined in part by *functional dependencies* among different neurobehavioral substrates of behavior (e.g., Beauchaine & Constantino, 2017; Beauchaine & Hinshaw, 2020). Such perspectives date at least to the mid-20th century, when Gray (1970, 1987) proposed that propensities toward approach behaviors derive from *competing effects* of individual differences in sensitivity to (a) reward cues (trait impulsivity) and (b) frustrative nonreward/punishment cues (trait anxiety). Gray's perspective (see also Gray & McNaughton, 2000), which generated a large body of research on psychophysiological correlates of impulsivity (e.g., Beauchaine, Katkin, Strassberg, & Snarr, 2001; Fowles, 2000), is currently instantiated in reinforcement sensitivity theory (RST; Corr, 2001, 2004). RST specifies neural substrates of and functional interactions among cognitive-emotional valuation systems of activation and inhibition (Corr, 2008), including implications for externalizing behavior (Corr & McNaughton, 2016). Although full articulation of RST is beyond the scope of this article, it suggests that concurrently assessed dimensions of impulsivity (approach) and anxiety (avoidance) might better account for performance on specific tasks compared with measures of impulsivity alone.

RST and similar perspectives are supported behaviorally by consistent evidence that trait anxiety mollifies externalizing risk among vulnerable children and adolescents (see Beauchaine, Zisner, & Sauder, 2017; Schatz & Rostain, 2006). For example, anxiety symptoms predict better responses to certain treatments among externalizing children (Jensen et al., 2001). Furthermore, youths with CD and comorbid anxiety are less aggressive, experience less peer rejection, and face fewer police contacts than youths with CD alone (Walker et al., 1991). In contrast, low trait anxiety is a hallmark of callous unemotional traits—which predict clinical severity of conduct problems (e.g., Enebrink, Andershed, & Långström, 2009; Frick & White, 2008; Tremblay, Pihl, Vitaro, & Dobkin, 1994). Thus, externalizing behaviors are often *potentiated* by low levels of anxiety, consistent with RST.

To date, few studies have examined mechanisms through which anxiety moderates impulsive behaviors. At the neurobiological level of analysis, computational models of reward learning and delay discounting suggest that impulsivity–anxiety interactions may emerge from opponent dopaminergic and serotonergic systems in which dopamine facilitates learning from reward-prediction errors across time and serotonin modulates cost and risk valuation of potential rewards (Cools, Nakamura, & Daw, 2011; Doya, 2002, 2008; Long, Kuhn, & Platt, 2009; Macoveanu et al., 2013). Among healthy control participants, tryptophan (a serotonin precursor) depletion induces steeper delay discounting and stronger memory decay of previously experienced negative outcomes (Schweighofer et al., 2008; Tanaka et al., 2009).

At the neural level, experimentally induced anxiety attenuates value signals generated by the ventromedial prefrontal cortex when encoding rewards, which yields more risk-averse decision-making (Engelmann, Meyer, Fehr, & Ruff, 2015). Furthermore, comorbid anxiety among externalizing males is associated with less severe structural compromises in several brain regions implicated in impulsive decision-making, including the ventral striatum and the anterior cingulate cortex (Sauder, Beauchaine, Gatzke-Kopp, Shannon, & Aylward, 2012). Behaviorally, both typically developing children and children with ADHD show better response inhibition on stop-signal tasks if they experience symptoms of anxiety (Bloemsma et al., 2012; Manassis, Tannock, & Barbosa, 2000; Zinbarg & Revelle, 1989). In addition, computational models derived from prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992) have revealed that participants diagnosed with generalized anxiety disorder show stronger risk aversion relative to healthy control participants when making choices among both certain and probabilistic rewards/punishments (Charpentier, Aylward, Roiser, & Robinson, 2017). Likewise, individual differences in social anxiety, trait anxiety, and worry in both clinical and nonclinical samples are associated with risk aversion in the Balloon Analogue Risk Task, which mixes reward and punishment cues (Maner et al., 2007). Collectively, such findings are captured by RST through the *joint-subsystem hypothesis*, which postulates a positive relation between anxiety and indecision (e.g., arising from goal conflict between reward magnitude and delay). Thus, anxiety and associated indecision allow for more thorough risk assessment, attenuating subjective valuations of reward relative to risk (see Corr, 2004, 2008).

Despite the relevance of RST to decision-making, to our knowledge, no studies have tested interactive mechanisms through which impulsivity and anxiety affect impulsive decision-making even though main effects of both are well characterized (e.g., Avila & Parcet, 2001; Bloemsma et al., 2012; Duckworth & Kern, 2011; Manassis et al., 2000; Xia, Gu, Zhang, & Luo, 2017; Zhao, Cheng, Harris, & Vigo, 2015). Dependence of impulsive decisions on both trait impulsivity and anxiety may help to explain why self-report and behavioral measures of impulsivity show low correspondence (Cyders & Coskunpinar, 2011). Indeed, we would expect any 1:1 correspondence between trait and behavioral

measures of impulsivity to be diminished to the extent that impulsive and anxious tendencies interact to affect decision-making (see Beauchaine & Hinshaw, 2020). In addition, a fuller understanding of interactive effects between impulsivity and anxiety may help to explain mixed findings regarding differential effects of anxiety across different forms of impulsive decision-making and different groups of participants. Indeed, some studies have found that anxiety decreases impulsive decision-making through increased risk aversion, whereas others have shown increased impulsivity through steeper delay discounting (e.g., Charpentier et al., 2017; Schweighofer et al., 2008; Tanaka et al., 2009).

Modeling Functional Dependencies and Etiological Complexity

Quantifying complex functional dependencies among biobehavioral systems, such as those described above, presents significant barriers to testing theories of personality and psychopathology (Beauchaine & Constantino, 2017). In the present example, multiple neural mechanisms affected behavior in ways that are not well accounted for by traditional main effects regression models used in psychology. Instead, statistical models that account for functional dependencies among predictors across levels of analysis are needed. Traditional approaches linking DDT performance to personality traits first quantify behavioral summary statistics separately for each participant (e.g., discounting rates) and then use regression to estimate relations among those summary statistics and outcomes of interest (e.g., personality measures). This *two-stage* approach—as it is often termed in the cognitive-neuroscience literature—does not allow for statistical constraint across levels of analysis (see Turner, Forstmann, Love, Palmeri, & Van Maanen, 2017). In summarizing behavioral data before entering it into a secondary statistical model for hypothesis testing, the two-stage approach assumes implicitly that participants share no *group-level information* (e.g., knowing the average discounting rate across participants does not inform estimates at the individual level) and that behavioral summary statistics are estimated with *infinite precision* (i.e., discounting rates are estimated without error).[1] When these assumptions are not met, the two-stage method inflates measurement error. In turn, inflated measurement error leads to overconfident, biased estimates of model parameters and effects, particularly when numbers of observations for a measure are not fixed across participants or within conditions. In classic test-theory terms, such estimates are *nonportable* (see Rouder & Haifa, 2019). Note that self-report measures are often constructed using stringent criteria to help enforce portable estimates (e.g., ensuring high test–retest reliability, requiring all participants to answer the same questions). Summary measures from behavioral statistics rarely meet these standards (e.g., Hedge et al., 2017).

A solution to these problems is to construct a single model that (a) simultaneously pools behavioral data within and across participants to estimate both individual- and group-level summary statistics and (b) assumes theoretically relevant relations between behavioral (e.g., discounting rate) and external (e.g., personality traits) measures (e.g., Rouder & Haaf, 2019; Turner et al., 2017). Hierarchical Bayesian analysis (HBA; Craigmile, Peruggia, & Van Zandt, 2010; Kruschke, 2015; Lee & Wagenmakers, 2013; Rouder & Lu, 2005; Shiffrin, Lee, Kim, & Wagenmakers, 2008) is a framework that jointly estimates relations among task performance measures and individual-level personality measures (or any other combination of levels). HBA produces posterior distributions that convey how much *certainty* we have in parameter estimates given the data. Such information is not readily derived from traditional (frequentist) hierarchical-modeling approaches that rely on maximum likelihood estimation. As demonstrated here, HBA allowed us to construct *competing models* and to use formal Bayesian comparison techniques to determine which model best accounts for observed data while penalizing model complexity (for more information on benefits of Bayesian modeling, see Ahn, Krawitz, Kim, Busemeyer, & Brown, 2011; Craigmile et al., 2010; Rouder & Lu, 2005; Wagenmakers, 2007).

Objectives of the Current Study

Here we used an adaptive version of the DDT, HBA, and Bayesian model comparison to show that current levels of anxiety moderate effects of trait impulsivity on decision-making. We present data from three groups of participants ($N = 967$) with low to severe substance-use patterns. The descriptive models we developed revealed that high state anxiety decreases rates at which trait-impulsive individuals discount future rewards while performing the DDT. However, such findings appear to apply only to individuals who report concurrently high trait impulsivity *and* state anxiety. To better explain our pattern of findings, we developed a more mechanistic model that assumes anxiety and impulsivity are linked to cognitive mechanisms of reward/risk valuation and delay valuation, respectively. Given formal correspondence among our explanatory model and other models used in the decision-making literature, we can offer testable predictions regarding anxiety-impulsivity effects in alternative forms of impulsive decision-making (e.g., risky decision-making paradigms).

Table 1. Demographic Characteristics by Group

Characteristic	Group				
	Student (n = 132)	MTurk (n = 800)	SUD (n = 35)	F	η^2
Age	20.1 (4.6)	35.1 (10.8)	35.8 (10.3)	$F(2, 964) = 124.0$	.2
Sex (male/female)	61/71	363/437	25/10	—	—
AUDIT score	4.9 (3.3)	9.6 (7.3)	14.9 (11.7)	$F(2, 789) = 26.3$	.06
DAST-10 score	0.6 (0.9)	2.4 (2.1)	7.7 (2.9)	$F(2, 964) = 179.9$	.27

Note: Values in parentheses are standard deviations. Because of experimenter error, participants recruited near the beginning of the study were not shown a portion of the Alcohol Use Disorder Identification Test (AUDIT) questionnaire. Summary statistics and statistical tests for the AUDIT were therefore computed on data collected from participants who completed the full questionnaire. Reduced sample sizes were 91, 674, and 27 for the student, Amazon Mechanical Turk (MTurk), and substance-use-disorder (SUD) groups, respectively. AUDIT scores ≥ 7 in women (8 in men) indicate harmful/hazardous alcohol use. Drug Abuse Screening Test (DAST-10) scores > 2 indicate problematic substance use. On average, the MTurk and SUD groups—but not the student group—reported problematic alcohol and substance use. For sex across groups, $\chi^2 = 9.1$.

Results offered potential insight into mechanisms through which anxiety serves a protective role among impulsive individuals yet *potentiates* impulsive decision-making among individuals without elevated trait impulsivity. We concluded that (a) main effects of single biobehavioral systems are often insufficient to describe task performance among individuals with psychopathology (see Beauchaine & Hinshaw, 2020; Beauchaine et al., 2018), (b) methods such as HBA offer principled means of testing complex theories of psychopathology that span levels of analysis, and (c) future research should gravitate away from searching for one-to-one correspondences between traits and task performance toward constructing statistical models that link levels of analysis in theoretically motivated ways (see Beauchaine & Constantino, 2017; Beauchaine & Hinshaw, 2020).

Method

Participants

Data were collected from three independent samples. Demographic characteristics of each sample appear in Table 1. The first sample comprised adult undergraduates (n = 132) who participated for credit in an introductory psychology course. Students were recruited from a general pool, so we anticipated lower scores on both trait impulsivity and state anxiety than among the other groups, described below, who were selected for substance-use behaviors. There were no exclusion criteria for students. Including the student group was important so we could determine whether state anxiety shows moderating effects on trait impulsivity when both are within normal ranges (Corr, 2004, 2008; Corr & McNaughton, 2016).

Each participant provided informed consent before completing questionnaires (including the Barratt Impulsiveness Scale [BIS-11] and State-Trait Anxiety Inventory [STAI]). The study protocol (2016H0108) was approved by The Ohio State Biomedical Sciences Institutional Review Board. Participants then completed two sessions of the DDT. Following the DDT, participants were debriefed and either given course credit or paid.

The second group (n = 800) was recruited through Amazon Mechanical Turk (MTurk), an online platform through which people participate in various tasks or surveys for money. Prior research has demonstrated the utility of MTurk for rapid and large-scale collection of valid and reliable data for clinical and behavioral research (Mason & Suri, 2011; Shapiro, Chandler, & Mueller, 2013). MTurk participants were eligible if they lived in the United States, had approval ratings of 90% or above on past work (Mason & Suri, 2011), and reported problematic use of cigarettes, alcohol, marijuana, stimulants, or opioids during prescreening. Only individuals who (a) believed they had a problem or (b) reported having a relative or friend who was concerned with their substance use were enrolled. After prescreening, MTurk participants were excluded if they failed more than one of four attention-check questions randomly dispersed among questionnaires (e.g., "Most people would rather lose than win" was failed if a participant selected "true"). In addition, we excluded MTurk participants who completed the DDT but failed to complete the trait-impulsivity or state-anxiety questionnaires described below (eight total). MTurk participants were paid $10/hr. We anticipated that this group would show higher levels of trait impulsivity than students given prescreening criteria.

The third group (SUD group; $n = 35$) comprised current patients at a local inpatient alcohol- and drug-treatment clinic. Participants were eligible if they met *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–5*; American Psychiatric Association, 2013) criteria for any alcohol or substance-use disorder according to the Structured Clinical Interview for DSM-5, Research Version (SCID-5; First, Williams, Karg, & Spitzer, 2015). Exclusion criteria included any history of head trauma with loss of consciousness for more than 5 min, a history of psychotic disorders, eight or more seizures, electroconvulsive therapy, or any neurological disorder. Participants were offered gift cards to a local grocery store at a rate of $10/hr. We expected that participants from the SUD group would show the highest levels of trait impulsivity.

Measures

Barratt Impulsiveness Scale. The BIS-11 is a 30-item self-report questionnaire that assesses three facets of impulsivity, including nonplanning, motor, and attentional impulsivity (Patton et al., 1995). We used the BIS-NP, which comprises 11 questions and (a) is most closely aligned with conceptualizations of trait impulsivity reviewed above and (b) is a consistent correlate of DDT performance (e.g., Koff & Lucas, 2011). Internal consistency (Cronbach's α) and 1-month test–retest reliability (r) of the BIS-NP both exceeded .7 (Stanford et al., 2009).

STAI. The STAI is a 40-item self-report measure that assesses state and trait anxiety (Spielberger, 1983). We used the state anxiety measure (STAI-S) because we hypothesized that current levels of anxiety, although affected by trait levels, would more potently moderate effects of trait impulsivity (i.e., BIS-NP) on discounting behavior. This hypothesis is based on known casual effects that state anxiety has on risk sensitivity/reward valuation (e.g., Engelmann et al., 2015). Test–retest reliability of the STAI-S has ranged from $r = .16$ to .83 for time periods spanning 1 week to many months (Barker, Wadsworth, & Wilson, 1976; Spielberger, 1983). Internal consistency (Cronbach's α) exceeded .80 (Spielberger, 1983). See Table S1 in the Supplemental Material available online for bivariate correlations between all impulsivity and anxiety subscales.

Alcohol Use Disorder Identification Test. The Alcohol Use Disorder Identification Test (AUDIT) comprises 10 items that are used to assess risk for alcohol-use disorder (Bohn, Babor, & Kranzler, 1995). We included the AUDIT to measure ranges of alcohol use across groups. A score of 8 or more among men (7 among women) indicates strong likelihood of hazardous or harmful alcohol use. A score above 20 suggests alcohol-use disorder. The AUDIT is both reliable ($r > .80$) and internally consistent ($\alpha > .80$; Daeppen, Yersin, Landry, Pécoud, & Decrey, 2000; Hays, Merz, & Nicholas, 1995).

Drug Abuse Screening Test. The Drug Abuse Screening Test (DAST-10) is a 10-item brief version of the 28-item DAST, which is used to assess past 12-month problematic substance use (Skinner, 1982). As with the AUDIT, we included the DAST-10 to measure variation in problematic substance use across groups. A DAST-10 score above 2 indicates problematic substance use (Cocco & Carey, 1998). The DAST-10 shows acceptable test–retest reliability ($r > .70$) and good internal consistency ($\alpha > .80$) across validation studies (see Yudko, Lozhkina, & Fouts, 2007).

SCID-5. The SCID-5 (First et al., 2015) was used to evaluate eligibility for the substance-use treatment clinic group, primarily to assess which substances caused the most dysfunction for participants. All SCID-5s were conducted either by (a) trained graduate students in a clinical psychology doctoral program or (b) trained research assistants. Final diagnostic decisions were rendered by W-Y. Ahn using a combination of SCID-5 assessments and patient medical records to ensure patients did not meet exclusion criteria.

Behavioral task

The monetary DDT comprises a sequence of binary choices between rewards varying in magnitude (dollars) and time of delivery (days, weeks, months, years). Each DDT trial consists of a choice between a smaller, sooner (SS) reward or a larger, later (LL) reward (e.g., "Would you rather have $10 now or $20 in one week?"). After choice data are collected, impulsivity is captured by participants' discounting rates—a model parameter that measures how steeply they discount values of temporally delayed rewards. A hyperbolic model (Mazur, 1987) is often used to describe discounting rates because it is simple and fits choice patterns better than many alternatives (e.g., exponential, power; but see Cavagnaro, Aranovich, McClure, Pitt, & Myung, 2016). Steeper discounting rates have been observed among individuals with a wide range of externalizing conditions (ADHD, CD, ASPD) and among individuals who abuse various substances (Beauchaine, Ben-David, & Sela, 2017; Bickel & Marsch, 2001; Bobova et al., 2009; Bornovalova et al., 2005; Petry, 2001; Wilson et al., 2010).

We used a DDT (Ahn et al., 2020) that uses a version of Bayesian active-learning, adaptive-design optimization (ADO) to improve task efficiency and the precision of parameter estimation (see Myung, Cavagnaro, & Pitt,

2013). Trial by trial, ADO selects dollar–day pairs that are expected to improve parameter estimation the most. Participant-level parameters (discounting rate, *k*, and choice sensitivity, *c*) are updated between trials using Bayesian updating, and delays and monetary values are then selected using a grid search over potential dollar-day pairs such that participants' choices minimize uncertainty in parameter estimates. This DDT version makes it possible to collect data 3 to 8 times more rapidly and 3 to 5 times more precisely than traditional staircase approaches (Ahn et al., 2020). Although each participant's parameters were estimated as the participant progressed through the task, modeling was conducted on raw choice data to facilitate hierarchical modeling. All three groups underwent two sessions of ADO-DDT separated by a 5-min break. Data from both sessions were combined to fit models described below. The student and SUD groups both underwent 42 trials per session, whereas the MTurk group underwent 20 trials per session. We used fewer trials for MTurk participants because analyses of data from the other groups, who were tested first, showed that additional trials rarely improved parameter estimation (test–retest reliability of delay-discounting estimates exceeded $r = .95$ after 20 or fewer ADO trials) and to minimize off-task behavior (Ahn et al., 2020).

Data analysis

First, we conducted Bayesian *t* tests to determine whether trait impulsivity and state anxiety varied across groups in predicted directions (i.e., students < MTurk < SUD). We used the R package *BEST*, which conducts Bayesian estimation of mean differences among groups as described by Kruschke (2015). *BEST* estimates parameters for means, standard deviations, and normality within groups, and differences among estimated means are used to infer group differences. We used default, noninformative prior distributions for all parameters. We then interpreted each distribution using highest density intervals, which we describe in detail under Interpreting Bayesian Models.

Next, we developed two classes of competing models to test the hypothesis that state anxiety moderates trait impulsivity to predict discounting rates on the DDT. We termed the first class of models *descriptive* in that they take the form of traditional interaction models used throughout psychology (albeit within a hierarchical Bayesian framework). This allowed us to determine general relations among state anxiety, trait impulsivity, and delay discounting. We termed the second class of models *explanatory* in that they make specific assumptions about how people value both rewards and delays in a way that gives rise to the interactive effect between impulsivity and anxiety we found with the descriptive model.[2] Below, we describe *base* and *trait* versions of both classes of models, which assume that personality measures have either no relation to or are linearly related to delay-discounting-model parameters, respectively.

Base descriptive model. The base descriptive model assumes that each participant discounts delayed rewards according to a hyperbolic function (Mazur, 1987) of the following form:

$$V = \frac{A}{1 + kt}, \tag{1}$$

where V is the value of the delayed reward, A is the actual (objective) amount of the reward, k ($0 < k < \infty$) is the discounting rate, and t is the time delay measured in weeks. With this parameterization, as k increases, t leads to greater decreases in V, which indicates steeper discounting of decision-making. V is computed for both the immediate and delayed options on each trial, and the subsequent values are then entered into a logistic equation to produce the probability of selecting the LL option:

$$Pr(\text{LL}) = \frac{1}{1 + e^{-c(V_{\text{LL}} - V_{\text{SS}})}}. \tag{2}$$

Here, V_{LL} and V_{SS} reflect values of the LL and SS choice options after being discounted in Equation 1, and c ($0 < c < 5$) is a choice-sensitivity (i.e., inverse temperature) parameter that captures how deterministically (c closer to 5) as opposed to randomly (c closer to 0) participants make choices according to differences in V_{LL} and V_{SS}.

We used HBA to simultaneously estimate group- and participant-level parameters separately for each of the three groups (Kruschke, 2015; Lee & Wagenmakers, 2013; Rouder & Lu, 2005; Shiffrin et al., 2008). HBA estimates posterior distributions that quantify uncertainty for each parameter, which makes it ideal for drawing reliable inferences of parameters in complex hierarchical models (e.g., Ahn et al., 2011). Details on the prior distributions and on detailed fitting procedures (including all the models overviewed below) are in the Supplemental Material.

Trait descriptive model. To test our hypothesis of an impulsivity-anxiety dependency in affecting discounting, we implemented Bayesian regression by reparameterizing k so it was determined by a linear combination of BIS-NP, STAI-S, and the interaction of BIS-NP and STAI-S (Boehm, Steingroever, & Wagenmakers, 2018). To do so, we first standardized each measure by mean-centering

and rescaling by the standard deviation separately within each group. Standardizing measures within each group allowed us to test whether within-participant competing effects of trait impulsivity and state anxiety varied across groups. We then estimated deviations in the group-level discounting rate attributable to anxiety and impulsivity using the following regression (for more details, see Equation S1 in the Supplemental Material):

$$\mu_k = \beta_0 + \beta_1 \times \text{BIS-NP} + \beta_2 \times \text{STAI-S} + \beta_3 \times \text{BIS-NP} \times \text{STAI-S}. \quad (3)$$

Here, β weights are interpreted similarly as in a standard multiple regression. Intuitively, β_0 is now interpreted as the group average discounting rate (μ_k from Equation S1), and other β weights account for participant-level variance in k that is attributable to their respective BIS-NP and STAI-S scores. Note that we omitted participant-level subscripts in Equation 3 for simplicity. Use of personality or trait measures to statistically constrain individual-level delay-discounting estimates allows for the trait descriptive model to account for uncertainty in behavioral data when estimating personality-behavior relations. This contrasts with the traditional two-stage method, described above, which reduces behavioral summary statistics to single-point (infinitely precise) estimates before probing personality–behavior relations.[3]

Base explanatory model. Given our pattern of findings across groups from the trait descriptive model, we developed a more explanatory, mechanistic model of the interaction between impulsivity and anxiety using models derived from research on computational neuroscience and decision-making and from translational research on delay discounting (e.g., Cools et al., 2011; Doya, 2002, 2008; Ho, Mobini, Chiang, Bradshaw, & Szabadi, 1999; Luckman, Donkin, & Newell, 2017). We made a simple extension to the traditional hyperbolic model, which assumes that reward magnitudes (e.g., $10) and delays (e.g., in 2 weeks) are valued independently and then combined in a way that naturally gives rise to an interactive effect:

$$V = \frac{A^{\alpha}}{1 + kt}. \quad (4)$$

In Equation 5, α ($0 < \alpha < +\infty$) is a reward-magnitude-valuation parameter that controls how sensitive people are to differences in reward (independent of delay) across choices on each trial. Note that changes in α can lead to similar behaviors compared with changes in the traditional discounting rate k. As α approaches 0, rewards are valued more for their frequency than for their objective values, which leads to indifference between either reward offered on each trial (e.g., receiving $10 once is equivalent to receiving $1 once). Conversely, as α approaches $+\infty$, people become very sensitive to even small differences between rewards (e.g., receiving $10.25 once is strongly preferred over receiving $10 once). This extended model can be viewed as a variant of the multiplicative-hyperbolic-discounting model used in animal research (e.g., Ho et al., 1999); the major difference is that we assumed a power function for reward valuation as opposed to a hyperbolic-saturating function.

As defined mathematically in Equation 4, α corresponds to the "risk sensitivity/aversion" parameter from prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992) given that it leads to an increase in risk aversion at the behavioral level of analysis when $\alpha < 1$. Although our DDT did not involve risky decision-making, model-comparison studies offer strong evidence that the risk aversion parameter (α) is preserved within participants across risky- and intertemporal-choice paradigms (Luckman et al., 2017). Therefore, although we did not interpret α as risk aversion per se, it is a useful theoretical correspondence that leads to specific predictions regarding how anxiety may influence impulsive decisions in the DDT (for details, see the Trait Explanatory Model section). RST predicts that anxiety leads to risk assessment (Corr, 2004, p. 324), and we can encode this prediction in the model by assuming that state anxiety is linked to α. Therefore, we refer to α as *reward sensitivity* because of its direct interpretation but emphasize that it produces risk aversion at the level of observed behavioral data, consistent with RST.

Finally, unlike in the descriptive models, we did not estimate c (choice sensitivity) as a free parameter and instead set c to 1 for all participants when fitting the explanatory models. We made this decision because α and c have similar functions in the model, which results in collinearity among parameters.[4] Note that when $c = 1$ for all participants, the model described by Equation 4 produced better interactive effects between α and k, which are described in more detail below (see the Trait Explanatory Model section). See Figure 1 for graphical depiction of independent and interactive effects of α and k.

Trait explanatory model. Evidence suggests that temporal valuation of rewards (i.e., discounting rate, k) is related to impulsivity/excessive approach, whereas reward valuation/risk aversion (α) is related to anxiety/excessive avoidance. Although k has traditionally been thought to capture impulsivity, correlational and

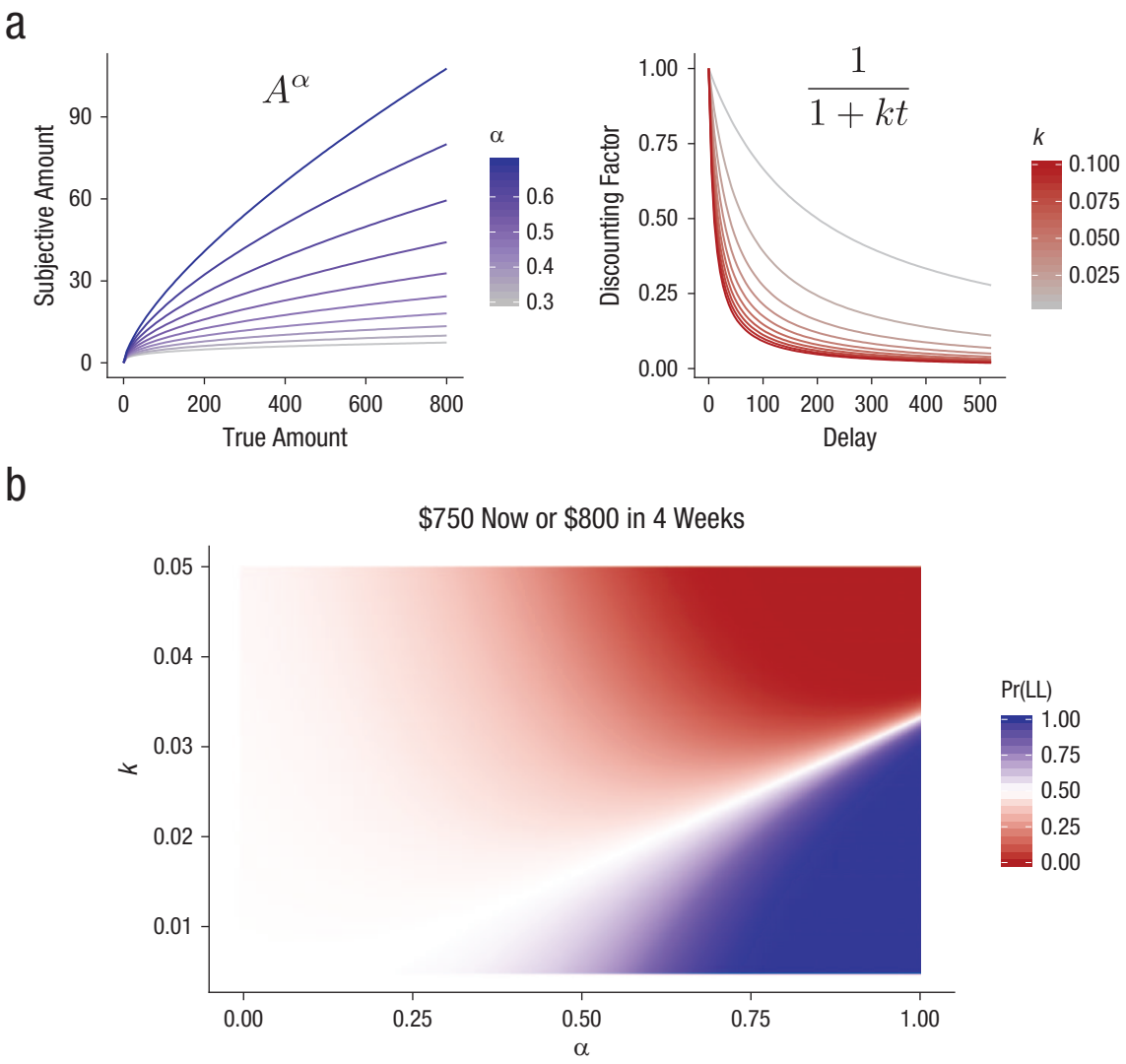

Fig. 1. Graphical depiction of the explanatory model described in the main text. (a) The explanatory model consists of two separate valuation mechanisms: one capturing reward magnitude sensitivity (α) and another capturing the traditional reward delay-discounting rate (*k*). As α decreases toward 0, the subjective difference between two rewards of different magnitudes becomes increasingly small and vice-versa. As *k* increases toward +∞, rewards become increasingly discounted with time and vice-versa. (b) The explanatory model assumes that both valuation mechanisms shown in Fig. 1a are combined such that they give rise to interactive effects (we constrained the parameter ranges for visualization purposes). When reward sensitivity is low (as α approaches 0), the discounting rate (*k*) has a dampened effect on the resulting preference, and both choices become more equally preferred. Conversely, when reward sensitivity is high (as α approaches +∞), the effect of *k* becomes increasingly strong, such that the larger, later (LL) or smaller, sooner (SS) choice becomes strongly preferred, dependent on specific choices and discounting rate. Assuming that reward magnitude and delay sensitivity are related to state anxiety and trait impulsivity, respectively (see the Trait Explanatory Model section), the model offers a more formal account of how anxiety and impulsivity may interact to produce impulsive or nonimpulsive decisions.

experimental studies reveal a correspondence among trait and state measures of anxiety and behavioral/computational-model parameters reflecting risk aversion, which is captured by α, as described above (see Approaches to Measuring Impulsivity; e.g., Charpentier et al., 2017 Engelmann et al., 2015; Maner et al., 2007). Therefore, we assume that individual-level α and *k* parameters are systematically related to individual differences in state anxiety and trait impulsivity across participants, respectively:

$$\mu_{\alpha 0} = \beta_{\alpha 0} + \beta_{\alpha 1} \times \text{STAI-S}$$
$$\mu_{k 0} = \beta_{k 0} + \beta_{k 1} \times \text{BIS-NP}. \quad (5)$$

As in Equation 3 (for the trait descriptive model), μ_α and μ_k indicate group-level means for reward (α) and delay (k) valuation parameters, which are estimated as a linear combination of a group-level "intercept" ($\beta_{\alpha 0}$) and an "effect" ($\beta_{\alpha 1}$) of individual differences in state anxiety (and likewise for impulsivity). Because this is the first empirical test of a model of this kind, we also tested the opposite model in which BIS-NP and STAI-S were assumed to relate to α and k, respectively (termed the *trait explanatory incongruent* model; we use the term *incongruent* for clarity, although it is possible that impulsivity and anxiety do in fact relate to α and k in this way despite empirical evidence suggesting otherwise). We also conducted a sensitivity analysis to determine whether our choice of BIS and STAI subscales appreciably affected our inference (see the Supplemental Material). In general, results held across subscales; the model presented in the main text showed the strongest hypothesized relations (see Fig. S6 in the Supplemental Material).

By setting the choice-sensitivity (c) parameter for the explanatory models to 1, "competition" between reward valuation (α) and delay discounting (k) can lead to patterns of impulsive decision-making that explain likely anxiety–impulsivity interactions.[5] As α approaches 0, the effect of the discounting rate attenuates, which leads to near indifference between the SS and LL options irrespective of the magnitude of k. Conversely, as α approaches $+\infty$, the effect of k strengthens such that having a high k leads to consistent choices of the SS option and vice-versa. Therefore, if state anxiety and trait impulsivity are negatively and positively associated with α and k (through Equation 5), respectively, then the trait explanatory model offers a more formal explanation of how state anxiety may interact with trait impulsivity to lead to impulsive decision-making (see Fig. 1b for a graphical depiction).

Model comparison. To compare descriptive models in a fully Bayesian manner, we used the leave-one-out information criterion (LOOIC), which approximates how well a model should generalize to new data (Vehtari, Gelman, & Gabry, 2017). Because we fit descriptive models separately to each group, we used LOOIC to estimate how well the models should perform on new participants sampled from the same groups (i.e., within student, MTurk, and SUD groups). In contrast, to compare explanatory models, which were fit to all groups simultaneously, we used a leave-one-group-out measure. We fit explanatory models simultaneously to the (log pointwise predictive density, or LPPD) student and MTurk groups and then made predictions on individual-level choices for each participant in the SUD group using state anxiety and trait impulsivity scores alone. Further details on the model-comparison measures are included in the Supplemental Material.

Interpreting Bayesian models. To interpret Bayesian models, we report highest density intervals (HDIs) to summarize posterior distributions, which are analogous but not equivalent to frequentist confidence intervals. An x% HDI covers the range of parameter values comprising x% of the area of the posterior distribution, in which every value falling inside the interval is more probable than any value falling outside the interval. Using the trait descriptive model as an example, a 95% HDI = [0.15, 0.3] on β_1 indicates that the most probable 95% of values for β_1 fall between 0.15 and 0.3. Intuitively, it is useful to imagine the behavior of the HDI as we use a smaller and smaller x%. As x approaches 0, the interval converges to the single most probable parameter value (i.e., the mode of the distribution). As x approaches 100, HDI continues to highlight the x% of most probable parameter values until covering the entire range of the distribution. In this way, HDI extends the concept of a mode from a point estimate to a range of values. Therefore, HDIs differ from frequentist confidence intervals in that they make direct assertions about which parameter values are most probable, whereas frequentist confidence intervals make probability statements only about the proportion of confidence intervals containing a given value under repeated sampling. Note that we do not endorse binary interpretations of "significant differences" using HDIs but instead use them as a general measure of evidence (e.g., "Which discounting rate estimates are most probable?" "Which values best represent the effect of trait impulsivity on discounting rates?"). Again using the trait model as an example, a 95% HDI = [0.15, 0.30] on β_1 indicates strong evidence for a positive effect given that the range of 95% most probable values are well above 0 and that the 95% range is itself relatively narrow (i.e., the estimate is precise). Conversely, a 95% HDI = [–0.3, 0.4] on β_1 indicates weak evidence for no effect given that the range is both centered around 0 and relatively wide (i.e., the estimate is not precise). For detailed discussion of HDIs, their uses, and their similarities and differences with respect to frequentist confidence intervals, see Chapter 11 of Kruschke (2015).

Results

State, trait, and behavioral differences

Here, we report HDIs on estimated differences in mean trait impulsivity (BIS-NP) and state anxiety (STAI-S) scores among groups in addition to estimated group-level discounting rates for each group. As depicted in Figure 2a, trait impulsivity varied across groups in the

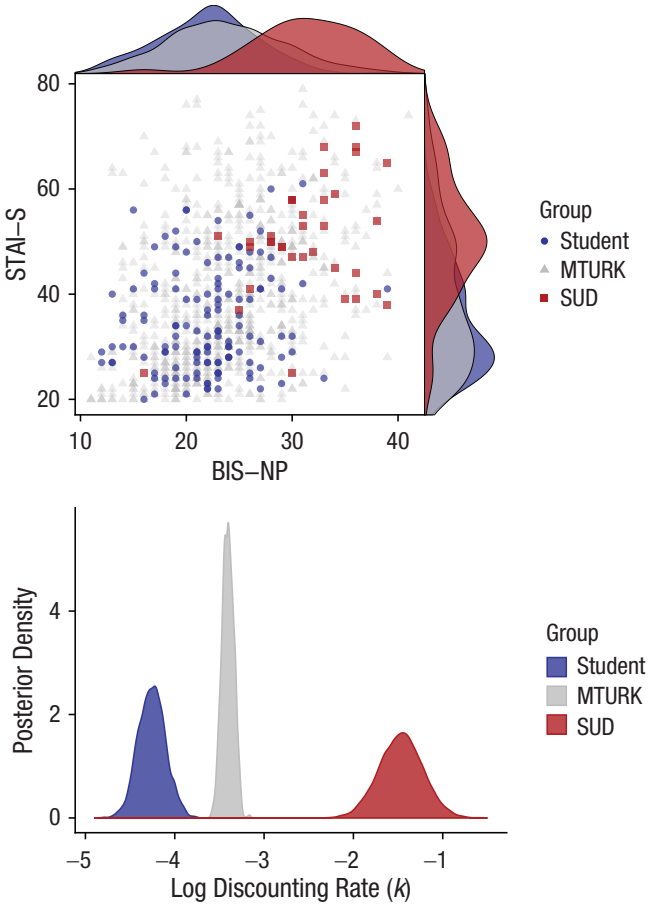

Fig. 2. Trait impulsivity, state anxiety, and behavioral impulsivity across groups. (a) Scatterplot with marginal distributions for summed scores of trait impulsivity (nonplanning subscale of the Barratt Impulsiveness Scale [BIS-NP]) and state anxiety (state anxiety measure of the State-Trait Anxiety Inventory [STAI-S]) across groups. Pearson's correlations between BIS-NP and STAI-S scores for each group were as follows—student: r = .17; Amazon Mechanical Turk (MTurk): r = .38; and substance-use-disorder (SUD) group: r = .39. (b) Posterior distributions over group-level delay-discounting rates estimated using the base descriptive model. Note that the distributions contain uncertainty in parameter estimates and can therefore be directly compared across groups.

anticipated direction. Students had lower BIS-NP scores than both the MTurk group and the SUD group (student – MTURK: 95% HDI = [–2.67, –0.87]; student – SUD: 95% HDI = [–11.36, –7.61]). The MTurk group also had lower BIS-NP scores than the SUD group (MTURK – SUD: 95% HDI = [–9.42, –5.92]). Results were similar for state anxiety (see Fig. 2a); students had lower STAI-S scores than both the MTurk group and the SUD group (student – MTURK: 95% HDI = [–6.16, –2.23]; student – SUD: 95% HDI = [–19.82, –11.36]). The MTurk group also had lower STAI-S scores than the SUD group (MTURK – SUD: 95% HDI = [–15.29, –7.31]). Frequentist t tests offered the same conclusions.[6] In the base descriptive model, discounting rates varied as predicted across groups (Fig. 2b). The SUD group showed the steepest discounting, followed by the MTurk group, then students. Taken together, results indicate that our selection criteria effectively produced three different groups with varying levels of trait impulsivity, state anxiety, and impulsive decision-making during the DDT.

Descriptive models. Model comparison of the base as opposed to the trait descriptive models showed that the trait descriptive model more effectively accounted for student (Base LOOIC – Trait LOOIC = 2.5; *SE* difference = 6.9), MTurk (Base LOOIC – Trait LOOIC = 24.8; *SE* difference = 25.9), and SUD (Base LOOIC – Trait LOOIC = 68.4, *SE* difference = 74.5) participants' DDT performance.[7] This suggests that main or dependent effects of trait impulsivity and state anxiety accounted for meaningful variance in individual-level decision-making.[8] The difference in LOOIC among models for students was lowest relative to the standard error of the difference, which may be due to a lack of dependency between BIS-NP and STAI-S among students. In fact, the 95% HDI on β_3, the interaction term for students indicates weak evidence for no moderating effects of BIS-NP and STAI-S on discounting rates (95% HDI on β_3 = [–0.34, 0.29]), whereas both the MTurk (95% HDI on β_3 = [–0.25, 0.00]) and SUD (95% HDI on β_3 = [–0.98, –0.27]) samples showed evidence of moderating effects (see Fig. S1 in the Supplemental Material).

In addition, both student (95% HDI on β_1 = [0.16, 0.78]) and MTurk (95% HDI on β_1 = [0.11, 0.40]) groups showed strong correspondences between nonplanning impulsivity (BIS-NP) and discounting rates, conditioned on state anxiety and their interaction (Fig. S1). Conversely, the SUD (95% HDI on β_1 = [–0.66, 0.45]) group showed weak evidence for no conditional effect of BIS-NP on delay discounting. Conditional effects of state anxiety (STAI-S) on discounting rates were weaker; there was some evidence for a negative association among students (95% HDI on β_2 = [–0.57, 0.04]) and some evidence for a positive relationship in the MTurk (95% HDI on β_1 = [–0.03, 0.26]) and SUD (95% HDI on β_1 = [–0.05, 0.89]) groups. Figure 3a shows discounting rates for each group, predicted by the trait descriptive model, at varying levels of BIS-NP and STAI-S, which makes the moderating effect of anxiety on the association between impulsivity and discounting clearer. In Figure 3a, interactions are evident for both the MTurk and SUD groups such that discounting rates were highest when individuals endorsed both low levels of state anxiety and high levels of trait impulsivity. In contrast, the student group showed no interaction; discounting rates were best characterized by independent main effects of trait impulsivity and state anxiety. These results suggest that impulsive decision-making is multiply determined by both trait impulsivity and state

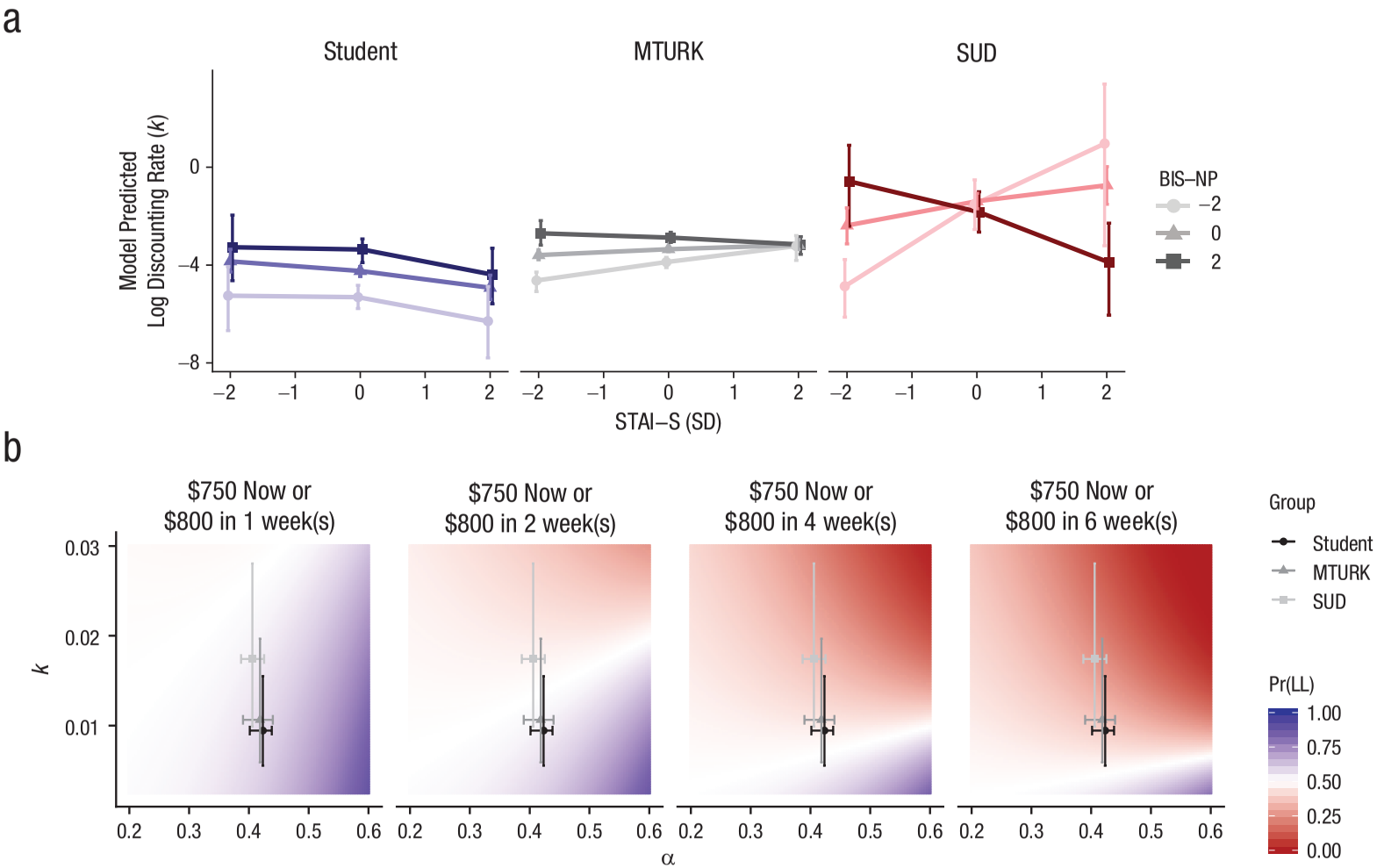

Fig. 3. Interaction of the nonplanning subscale of the Barratt Impulsiveness Scale (BIS-NP) and state-anxiety measure of the State-Trait Anxiety Inventory (STAI-S) in predicting discounting rates for both trait models. (a) Model-predicted discounting rates for different combinations (standard deviations from mean) of trait impulsivity (BIS-NP) and state anxiety (STAI-S) within each group given parameter estimates from the trait descriptive model. Points indicate modes of model predictions, and uncertainty intervals (vertical bars) reflect 80% highest density intervals (HDIs) of model-predicted discounting rates (posterior predictive distributions over group-level discounting rates), which help to visualize how uncertainty in the trait descriptive-model parameters affects estimates of discounting rate. Note that Bayesian intervals indicate probabilities. Although low on the BIS-NP, highly anxious participants in the SUD group appear to have steeper discounting rates relative to others on average; there is a nonnegligible probability that they have lower rates (the HDIs span both above and below others). When accounting for such uncertainty, Amazon Mechanical Turk (MTurk) and SUD groups showed a very similar pattern. (b) Model-estimated effects of STAI-S and BIS-NP on parameters of the trait explanatory model for different example choices from the DDT (LL = larger, later choice). Individual plots are "zoomed-in" versions of the same plot from Fig. 1b, which we chose for interpretative purposes. Points indicate predicted group-level estimates (μ_k and $\mu\alpha$ from Equation 5; see also Equation S3 in the Supplemental Material available online for more details) for individuals with sample-average levels of STAI-S and BIS-NP within each group. Uncertainty intervals highlight the same estimates but for individuals at the 5th and 95th in-sample quantiles of STAI-S and BIS-NP within each group. Therefore, uncertainty intervals represent variation in α and k across participants that is attributable to individual differences in state/trait measures, in which α and k are negatively and positively associated with STAI-S and BIS-NP scores, respectively.

anxiety, although there is some apparent discrepancy among groups (i.e., main effects with no interaction in the student sample). Below, we expand to provide an explanatory account of the impulsivity-anxiety interactions and develop a more robust model across all groups.

Explanatory models. Model comparison of the base and trait explanatory models showed that the trait explanatory model—in which α and k are assumed to relate to state anxiety and trait impulsivity, respectively—provided the best out-of-sample predictions (LPPD = −4,098) compared with the base explanatory model (LPPD = −5,011) and the trait incongruent explanatory model (LPPD = −4,441).[9] In addition to best predicting performance across the whole SUD group, the trait explanatory model outperformed competing models for individual participants in the SUD group (see Fig. S3 in the Supplemental Material). Such results provide relatively strong evidence that state anxiety and trait impulsivity are linked to mechanisms of reward/risk and delay valuation (captured by α and k, respectively) in a way that generalizes across qualitatively different groups.

Posterior distributions for parameters of the trait explanatory model are shown in Figure S4 in the Supplemental Material. We found strong evidence for a negative relation between the STAI-S and α such that increases in state anxiety predicted attenuated reward valuation (95% HDI on $\beta_{\alpha 1} = [-0.045, -0.011]$), consistent with both (a) relations between anxiety and indecision/increased risk sensitivity predicted by RST (Corr, 2004, 2008) and (b) previous studies showing that state anxiety increases risk aversion. In addition, we found a positive association between BIS-NP and k such that increases in trait impulsivity predicted increases in delay discounting (95% HDI on $\beta_{k1} = [0.20, 0.40]$). These results corroborate the interaction revealed by the trait descriptive model and offer an explanation for how state anxiety and trait impulsivity interact to produce impulsive decisions. For example, Figure 3b demonstrates estimated group-level effects of state anxiety and trait impulsivity on four different example choices from the DDT.

Discussion

Psychopathology research continues to shift from discrete syndromal conceptualizations of mental illness toward transdiagnostic trait approaches that specify complex interactions among multiple vulnerabilities (e.g., Beauchaine & Cicchetti, 2019; Beauchaine & Constantino, 2017; Beauchaine & Hinshaw, 2020; Robbins et al., 2012). Trait impulsivity is one such vulnerability (Beauchaine & McNulty, 2013; Beauchaine, Zisner, & Sauder, 2017; Ersche et al., 2010; Lombardo et al., 2012; McCloskey et al., 2009). Our findings demonstrate a clear functional dependency between trait impulsivity and state anxiety such that high state anxiety decreases rates at which trait-impulsive individuals discount delayed rewards. Our trait explanatory model suggests that this pattern of behavior is better explained by a delay-discounting model assuming that impulsivity and anxiety reflect delay/time and reward/risk valuation, respectively. Furthermore, given evidence that reward/risk sensitivity is preserved across intertemporal and risk decision-making paradigms within participants (Luckman et al., 2017) and that anxiety inductions increase risk aversion (Engelmann et al., 2015), our model provides an explanation for why anxiety has differential effects on impulsive decisions across both different paradigms and levels of trait impulsivity. Decreases in reward/risk sensitivity (α) in response to anxiety lead to more random responding during delay-discounting paradigms, which can be interpreted as either an increase or decrease in impulsivity depending on individuals' discounting rates (see Fig. 1). However, in risky decision-making paradigms (e.g., \$3 with certainty or \$4 with probability .8), the same decrease in α leads to a higher likelihood of choosing the safe ("nonimpulsive") option. Future studies might manipulate state anxiety experimentally among individuals who are low as opposed to high on trait impulsivity. Experimental manipulations, combined with alternative forms of delay discounting (e.g., cigarette discounting) may reveal novel strategies for decreasing reward values of drug cues among individuals with SUDs.

Our findings also have broader implications for traditional methods used to test hypotheses in psychopathology research. For example, psychopathology research continues to shift away from single-level analyses and toward multiple-level analyses in development and validation of theories of mental illness (e.g., Beauchaine & McNulty, 2013; Cicchetti, Ackerman, & Izard, 1995; Cicchetti & Dawson, 2002). Often, researchers assume one-to-one links between constructs across levels of analysis. As in the two-stage approach, this assumes that behavioral measures are unidimensional and portable. However, behavior observed on seemingly single-dimension tasks (the DDT here) is often determined by multiple, competing mechanisms (see also Ahn et al., 2014; Beauchaine & Constantino, 2017; Beauchaine & Hinshaw, 2020; Finucane, Challman, Martin, & Ledbetter, 2016; Haines, Vassileva, & Ahn, 2018). We demonstrated this across self-report and behavioral measures, but similar effects were observed when linking behavior to neural data (Turner et al., 2018). Consequently, main effects analyses using summary statistics derived from behavioral data alone are insufficient for identifying latent cognitive, emotional, and neural mechanisms

underlying complex behaviors. Furthermore, the assumption of portability is rarely considered for data collected from anything other than self-report measures (e.g., behavioral, physiological, and neural), which can lead to biased inferences and overconfidence in the wrong parameter values (e.g., β weights from multiple regression). HBA offers a flexible statistical framework to solve such problems and construct interpretable, complex models of psychopathology that can be formally compared (see Boehm et al., 2018; Rouder & Haaf, 2019).

Several limitations should be considered. First, the student sample and especially the SUD sample were smaller than the MTurk sample. Smaller samples are underpowered relative to larger samples and may be influenced more by outliers or sample-specific characteristics. However, HBA is less sensitive to small sample sizes than traditional methods, which often do not pool information across participants in a principled manner to estimate effects (Kruschke, 2015; Lee & Wagenmakers, 2013; Rouder & Lu, 2005; Shiffrin et al., 2008). In addition, our explanatory model was fit simultaneously to all 967 participants, and we identified patterns consistent with the descriptive models that were fit to each sample. Still, findings specific to the SUD sample in particular should be replicated in future research.

Second, the student, MTurk, and SUD groups likely differ in ways not measured, which may have contributed to finding no impulsivity–anxiety interaction within the student group using the trait descriptive models. Executive function/self-control is one possible explanation. In theory, strong executive control could modulate competition between impulsivity and anxiety, consistent with RST (see Fig. 2a and State, Trait, and Behavioral Differences section; Beauchaine & Hinshaw, 2020; Corr, 2004, 2008). However, there is significant overlap among posterior distributions for the interaction terms in the student and MTurk models (see Panel 3 of Fig. S1), and it is possible that a larger student sample could reveal an interaction similar to the MTurk group. Therefore, we caution overinterpretation of the interaction from the trait descriptive model in the student group given large uncertainty intervals. Future studies may address these points by incorporating additional relevant measures such as executive function into the trait descriptive model we developed.

Furthermore, our study was cross-sectional, and we are not claiming that links between trait impulsivity and state anxiety are causal. Use of anxiety manipulations in future studies may identify potential causal effects. Finally, although our explanatory model takes a step in this direction, use of neurally inspired computational models that account for dynamics among choices, response times, and neural activation might allow for more precise inferences on joint effects of state anxiety and trait impulsivity on impulsive decision-making (Turner et al., 2018; Turner, Van Maanen, & Forstmann, 2015). Although we did not collect reaction-time measures, future studies may leverage such models to more precisely determine separable effects of impulsivity, anxiety, and executive function on impulsive decision-making and behavior.

In sum, state anxiety moderates the association between trait impulsivity and impulsive decision-making such that high trait-impulsive individuals show reduced discounting of delayed rewards when they endorse high levels of state anxiety. Such reduced discounting leads to more optimal, future-oriented decisions in the DDT. Furthermore, our findings from the trait explanatory model reveal a potential mechanism through which anxiety serves as a protective factor against impulsive behavior in individuals with externalizing spectrum disorders but leads to more impulsive behavior for individuals with low trait impulsivity (see above). Future research may use experimental manipulations to determine whether within-participants anxiety inductions decrease the value of drug cues in high trait-impulsive individuals with SUDs. More broadly, HBA offers a principled way to explore how mechanisms at one level of analysis interact to produce effects at another level, which can shed light on dimensional neural-, cognitive-, and trait-level constructs that underlie traditionally discretized behavioral syndromes (see also Ahn, Haines, & Zhang, 2017).

Transparency

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Author Contributions

N. Haines, W.-Y. Ahn, and T. P. Beauchaine developed the study concept. N. Haines, A. H. Rogers, H. Hahn, and W.-Y. Ahn contributed to the study design. M. A. Pitt, J. I. Myung, and W.-Y. Ahn developed and implemented a tool for conducting adaptive design optimization of the delay-discounting task. Testing and data collection were performed by N. Haines, A. H. Rogers, H. Hahn, and W.-Y. Ahn. N. Haines performed all data analyses, and all of the authors provided feedback. All of the authors contributed to interpretation of the results. N. Haines drafted the manuscript, and T. P. Beauchaine provided critical edits and revisions. All of the authors provided revisions and approved the final version of the manuscript for submission.

Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Open Practices

All data have been made publicly available via OSF and can be accessed at https://osf.io/ewzfb/. The complete Open Practices Disclosure for this article can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702620929636. This article has received the badge for Open Data. More information about the Open Practices badges can be found at https://www.psychologicalscience.org/publications/badges.

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Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702620929636

Notes

1. We explain mathematical details underlying these assumptions in the Supplemental Material (see Base Descriptive Model under Model Parameterizations and Fitting Procedures).
2. We use the term *explanatory* because the model offers a specific explanation for how anxiety and impulsivity interact through their interrelations with different cognitive processes. We note, however, that the model is still descriptive because it does not identify a direct, causal mechanism.
3. We also tested the traditional frequentist version of the two-stage approach, which showed evidence for an interaction only in the SUD group. We discuss these results in detail in the Supplemental Material (see Traditional Two-Stage Approach).
4. We conducted an additional sensitivity analysis to determine whether setting $c = 1$ affected our inference, as described in the Supplemental Material (see Sensitivity Analysis). In brief, we fit a model that estimated a single value for c across all participants (akin to the group-level parameters for α and k). Effects of state anxiety and trait impulsivity on α and k, respectively, were consistent with the reported model where $c = 1$ (Fig. S5).
5. We use the term *competition* to refer broadly to the interactive nature of parameters in the model. We use this term instead of *interaction*, which could be misinterpreted to mean a traditional interaction as in Equation 3.
6. Traditional frequentist t tests showed that students had lower BIS-NP scores than both the MTurk, $t(196.7) = -3.91$, $p < .001$, $d = -0.56$, and SUD groups, $t(51.5) = -10.02$, $p < .001$, $d = -2.79$, and that the MTurk group had lower BIS-NP scores than the SUD group, $t(37.9) = -8.79$, $p < .001$, $d = -2.86$. In addition, students had lower STAI-S scores than both the MTurk, $t(220.0) = -4.22$, $p < .001$, $d = -0.57$, and SUD groups, $t(49.5) = -7.40$, $p < .001$, $d = -2.10$, and the MTurk group had lower STAI-S scores than the SUD group, $t(38.6) = -5.74$, $p < .001$, $d = -1.85$.
7. Because lower LOOIC values indicate better model performance, positive values for the difference of Base LOOIC – Trait LOOIC indicate better performance for the trait descriptive model.
8. We fit a main-effects-only trait descriptive model (no Impulsivity × Anxiety interaction term) in addition to the full interaction model, which we describe in the Sensitivity Analysis section of the Supplemental Material. Results were consistent with those reported in text.
9. LPPD closer to 0 indicates better predictive performance within the out-of-sample SUD group. See the Supplemental Material for further details on interpretation of LPPD.

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