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. Longstanding theories of personality and decision-making predict that neurally mediated individual differences in sensitivity to reward cues versus punishment cues (frustrative non-reward) interact to affect behavioral tendencies. Such interactions obscure 1:1 correspondences between single personality traits and task performance. We use 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 moderates 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

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* based on common genetic, neural, cognitive, and behavioral processes observed across disorders (e.g., Beauchaine, Zisner et al., 2017; Gatzke-Kopp et al., 2009; Gizer, Otto, & Ellingson, 2017). Notably, those 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 et al., 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 laboratory

task performance (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 like most human behavioral traits, impulsivity 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 consider 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 et al., 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). Notably, many adult measures assess multiple facets of impulsivity (Sharma, Markon, & Clark, 2014; Whiteside & Lynam, 2001). For example, the Barratt Impulsiveness Scale (BIS-11) assesses non-planning, motor, and attentional impulsivity (Patton et al., 1995). High scores on non-planning (BIS-NP), which captures preferences for immediate over delayed rewards, are observed consistently among those 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 use here, assess how individuals assign value to delayed rewards by presenting them with sequences of choices between smaller magnitude, sooner (SS) rewards and larger magnitude, later (LL) rewards (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 are observed among those 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 show average 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 examine 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 reward cues (trait impulsivity) vs. frustrative non-reward/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), rather than measures of impulsivity alone, might better account for performance on specific tasks.

RST and similar perspectives are supported behaviorally by consistent evidence that trait anxiety mollifies externalizing risk among vulnerable children and adolescents (see Beauchaine, Zisner, et al., 2017; Schatz & Rostain, 2006). For example, anxiety symptoms predict better responses to certain treatments among externalizing children (Jensen et al., 2001). Furthermore,

youth with CD and comorbid anxiety are less aggressive, experience less peer rejection, and face fewer police contacts than youth 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 however, 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, where 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 controls, 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 (vmPFC) when encoding rewards, yielding 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). Additionally, computational models

derived from prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992) reveal that those who meet criteria for generalized anxiety disorder show stronger risk aversion relative to healthy controls when making choices among both certain and probabilistic rewards/punishments (Charpentier, Aylward, Roiser, & Robinson, 2017). Similarly, individual differences in social anxiety, trait anxiety, and worry in both clinical and non-clinical 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 among reward magnitude and delay). Thus, anxiety and associated indecision allows 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). More importantly, 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 find that anxiety decreases impulsive decision-making through

increased risk-aversion, whereas others show 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 affect 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) then use regression to estimate relations between 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/effects, particularly when numbers of observations for a measure are not fixed across participants and/or

[1]We explain mathematical details underlying these assumptions in the Supplementary Text (see Model Parameterizations and Fitting Procedures *Base Descriptive model*).

within conditions. In classic test theory terms, such estimates are *non-portable* (see Rouder & Haaf, 2019). Of note, 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 answers the same questions, etc.). Summary measures from behavioral rarely meet these standards (e.g., Hedge, Powell, & Sumner, 2017).

A solution to these problems is to construct a single model that simultaneously pools behavioral data within and across participants to estimate both individual- and group-level summary statistics, and 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 can jointly estimate relations between 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 we also demonstrate, HBA allows 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 use an adaptive version of the delay discounting task (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 (total *N*=967) with low to severe substance use patterns. The descriptive models we developed reveal 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 those who report concurrently high trait impulsivity *and* state anxiety. To better explain our pattern of findings, we develop 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 between 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).

Results offer potential insight into mechanisms through which anxiety serves a protective role among impulsive individuals, yet *potentiates* impulsive decision-making among those without elevated trait impulsivity. We conclude that (1) main effects of single biobehavioral systems are often insufficient to describe task performance among those with psychopathology (see Beauchaine & Hinshaw, 2020; Beauchaine et al., 2018); (2) methods such as HBA offer principled means of testing complex theories of psychopathology that span levels of analysis; and (3) future research should gravitate away from searching for 1:1 correspondences between traits and task performance toward constructing statistical models that link levels of analysis in theoretically motivated ways (see Beauchaine & Constantino, 2017).

Method

Participants

Date were collected from three independent samples. Demographic characteristics of each sample appear in Table 1. The first sample comprised adult undergraduates ($n_{student}$ = 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 or not state anxiety shows moderating effects on trait impulsivity when both are within normal ranges (cf. Corr, 2004, 2008; Corr & McNaughton, 2017).

The second group (n_{MTURK} = 800) was recruited through Amazon Mechanical Turk (MTURK), an online platform through which people participate in various tasks and/or surveys for money. Prior research demonstrates 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 pre-screening. Only those who (1) believed they had a problem, or (2) reported having a relative or friend who was concerned with their substance use were enrolled. After pre-screening, MTURK participants were excluded if they failed more than 1 of 4 attention check questions randomly dispersed among questionnaires (e.g., "*Most people would rather lose than win*" is failed if a participant selects *True*). Additionally, we excluded MTURK participants who completed the DDT but failed to complete the trait impulsivity and/or state anxiety questionnaires described below (8 total). MTURK participants were paid $10/hr. We anticipated this group would show higher levels of trait impulsivity than students given pre-screening criteria.

The third group (n_{SUD} = 35) comprised current patients at a local inpatient alcohol and drug treatment clinic (SUDs group). Participants were eligible if they met *DSM-5* (American Psychiatric Association, 2013) criteria for any alcohol or substance use disorder according to the Structured Clinical Interview for *DSM-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 SUDs participants would show the highest levels of trait impulsivity.

Table 1. *Demographic Characteristics by Group*.

	Group			F	η^2
	Student (*n*=132)	MTURK (*n*=800)	SUD (*n*=35)		
Age (*SD*)	20.1 (4.6)	35.1 (10.8)	35.8 (10.3)	124.8	.2
Sex (male/female)	61/71	363/437	25/10	–	–
AUDIT score	4.9 (3.3)	9.6 (7.3)	14.9 (11.7)	25.4	.06
DAST-10 score	0.6 (0.9)	2.4 (2.1)	7.7 (2.9)	141.6	.23

Notes. Due to experimenter error, participants recruited near the beginning of the study were not shown a portion of the AUDIT questionnaire. Summary statistics/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, MTURK, and SUD groups, respectively. AUDIT scores ≥ 7 in women (8 in men) indicate harmful/hazardous alcohol use. 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, $\chi^2 = 9.1$

Measures

Barratt Impulsiveness Scale

The BIS-11 is a 30-item self-report questionnaire that assesses three facets of impulsivity including non-planning, motor, attentional impulsivity (Patton, Stanford, & Barratt, 1995). We used the non-planning subscale (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 one-month test-retest reliability (*r*) of the BIS-NP both exceed .7 (Stanford et al., 2009).

State-Trait Anxiety Inventory

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), as 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 the known casual effect that state anxiety has on risk sensitivity/reward valuation (e.g., Engelmann, Meyer, Fehr, & Ruff, 2015). Test-retest reliability of the STAI-S ranges from $r = .16$ to .83 for time periods spanning one week to many months (Barker, Wadsworth, & Wilson, 1976; Spielberger, 1983). Internal consistency (Cronbach's α) exceeds .80 (Spielberger, 1983). See Table S1 in the Supplementary Text for the bivariate correlations between all impulsivity and anxiety subscales.

Alcohol Use Disorder Identification Test

The 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 a strong likelihood of hazardous/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 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 >

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).

Structured Clinical Interview for the DSM-5, Research Version *(SCID).*

The SCID (First et al., 2015) was used to assess eligibility for the substance use treatment clinic group, primarily to assess which substances caused the most dysfunction for participants. All SCIDs were conducted by either: (1) trained graduate students in a clinical psychology Ph.D. program, or (2) by trained research assistants. Final diagnostic decisions were rendered by W.-Y. A. using a combination of SCID assessments and patient medical records to ensure patients did not meet exclusion criteria.

Behavioral Task

Delay Discounting 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) or larger-later (LL) reward (e.g., would you rather have *$10 now* or *$20 in one week*). After collecting choice data, 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 similar alternatives (e.g., exponential, power) (but see Cavagnaro, Aranovich, McClure, Pitt, & Myung, 2016). Steeper discounting rates are observed among those with a wide range of externalizing conditions (ADHD, CD, ASPD), and among those who abuse various substances (Beauchaine, Ben-David et al., 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., 2019) that uses a version of Bayesian active learning, adaptive design optimization to improve task efficiency and the precision of parameter estimation (ADO, 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 3-8 times more rapidly and 3-5 times more precisely than traditional staircase approaches (Ahn et al., 2019). Although each participant's parameters were estimated as they 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. Student and SUDs 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 exceeds r = .95 after 20 or fewer ADO trials) and to minimize off-task behavior (Ahn et al., 2019).

Procedure

All participants provided informed consent before completing questionnaires (including the BIS-11 and STAI). They then completed two sessions of ADO for the DDT. Following the DDT, participants were debriefed and either given course credit or paid.

Data Analysis

First, we conducted Bayesian *t-test*s 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 between groups as described by Kruschke (2015). *BEST* estimates parameters for means, *SD*s, and normality within groups, and differences between estimated means are used to infer group differences. We used the default, non-informative 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 term 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 between state anxiety, trait impulsivity, and delay discounting. We term 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 linear 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:

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

[2]Note that we use the term *explanatory* because the model offers a specific explanation for how anxiety and impulsivity interact through their relations with different cognitive processes. We note, however, that the model is still descriptive because it does not identify a direct, causal mechanism.

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, the time delay (t) leads to greater decreases in the value of delayed rewards (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:

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

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) versus randomly (c closer to 0) participants make choices according to differences in V_{LL} and V_{SS}.

We used hierarchical Bayesian analysis (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 on parameters in complex hierarchical models (e.g., Ahn et al., 2011). Details on the prior distributions and on the detailed fitting procedures (including all the models overviewed below) are in the Supplementary Text.

Trait Descriptive Model

To test our hypothesis of an impulsivity-anxiety dependency in affecting discounting, we implemented Bayesian regression by re-parameterizing 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 *SD* separately within each group. Standardizing measures within each group allowed us to test if 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 (see Equation S1 in the Supplementary Text for more details):

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

Here, β weights are interpreted similarly as in a standard multiple regression. Intuitively, β_0 is now interpreted as the group average discounting rate (i.e., μ_k from Eq. S1 in the Supplementary Text), 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 Eq. 3 for simplicity. Use of personality/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 computational neuroscience, decision-making, and translational research on delay discounting (e.g., Cools et al., 2011; Doya, 2002; 2008; Ho, Mobini, Chiang, Bradshaw, & Szabadi, 1999; Luckman, Donkin, & Newell, 2017). Specifically, we made a simple extension to

[3]We also tested the traditional frequentist version of the two-stage approach, which showed evidence for an interaction only in the SUDs group. We discuss these results in detail in the Supplementary Text (see Traditional Two-stage Approach from the Supplementary Text).

the traditional hyperbolic model which assumes that reward magnitudes (e.g., $10) and delays (e.g., in two weeks) are valued independently and then combined in a way that naturally gives rise to an interactive effect:

$$V = \frac{A^{\alpha}}{1 + kt} \tag{4}$$

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. Importantly, changes in α can lead to similar behaviors compared to changes in the traditional discounting rate k. Specifically, as $\alpha \to 0$, 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 $\alpha \to +\infty$, 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), with the major difference being that we assume a power function for reward valuation as opposed to a hyperbolic saturating function.

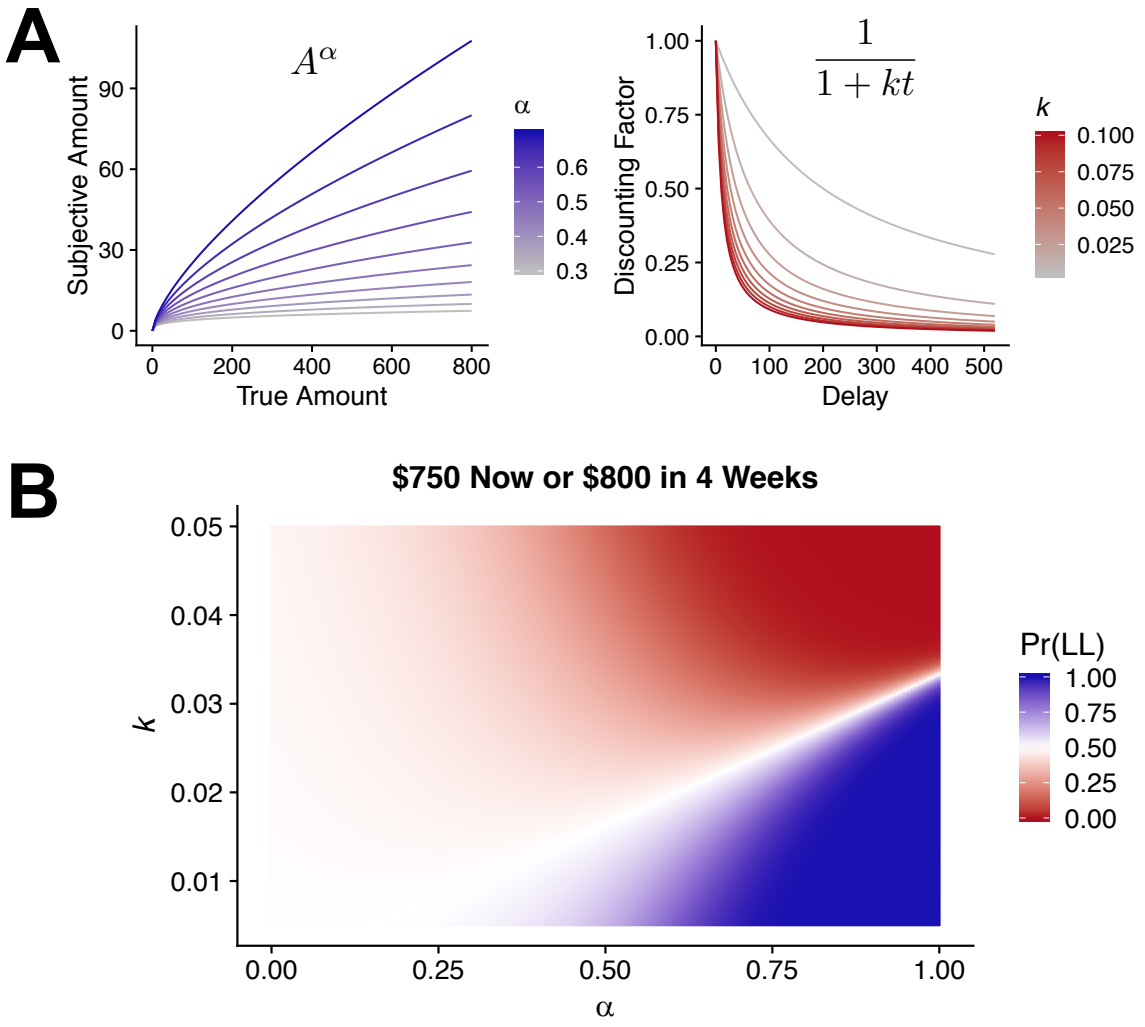
As defined mathematically in Eq. 4, α corresponds to the "risk sensitivity/aversion" parameter from prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992), as is leads to an increase in risk aversion at the behavioral level of analysis when $\alpha < 1$. Although our DDT does not involve risky decision-making, model comparison studies offer strong evidence that the risk aversion parameter (i.e. α) is preserved within-participants across risky- and inter-temporal choice paradigms (Luckman, Donkin, & Newell, 2017). Therefore, although we do 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 (see

Trait Explanatory model below for details). Specifically, RST predicts that anxiety leads to risk assessment (see Corr, 2004, pg. 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" due to 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 co-linearity between parameters[4]. More importantly, when $c = 1$ for all participants, the model described by Eq. 4 produces better interactive effects between α and k, which are described in more detail below (see *Trait Explanatory* model). See Fig. 1 for graphical depiction of independent and interactive effects of α and k.

[4]We conducted an additional sensitivity analysis to determine if setting $c = 1$ affected our inference, as describes in the Supplementary Text (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).

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 $+\infty$, rewards become increasingly discounted with time, and *vice-versa*. (B) The *Explanatory* model assumes that both valuation mechanisms described shown in panel A are combined such that they give rise to interactive effects (we constrained the parameter ranges for visualization purposes). Specifically, when reward sensitivity is low (i.e. as $\alpha \to 0$), 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 (i.e. as $\alpha \to +\infty$), the effect of k becomes increasingly strong, such that the larger later (LL) or shorter sooner (SS) choice becomes strongly preferred dependent on the specific choices and discounting rate. Assuming that reward magnitude and delay sensitivity are related to state anxiety and trait impulsivity, respectively (see *Trait Explanatory model*), the model offers a more formal account of how anxiety and impulsivity may interact to produce (non)impulsive decisions.

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 (i.e., α) is related to anxiety/excessive avoidance. Although k has traditionally been thought to capture impulsivity, correlational and experimental studies reveal a correspondence between 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:

$$\begin{aligned} \mu_\alpha &= \beta_{\alpha_0} + \beta_{\alpha_1} \cdot \text{STAI-S} \\ \mu_k &= \beta_{k_0} + \beta_{k_1} \cdot \text{BIS-NP} \end{aligned} \tag{5}$$

As in Eq. 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" (β_{α_0}) and an "effect" (β_{α_1}) of individual differences in state anxiety (and similarly 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 Supplementary Text). In general, results held across subscales, with the model presented in main text showing the strongest hypothesized relations (see Fig. S6).

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 $\alpha \to 0$, the effect of the discounting rate becomes increasingly attenuated, which leads to (near) indifference between the SS and LL options, irrespective of the magnitude of k. Conversely, as $\alpha \to +\infty$, the effect of k becomes increasingly strong, 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 Eq. 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, SUDs). In contrast, to compare *Explanatory* models, which were fit to all groups simultaneously, we used a leave-one-group-out measure (termed LPPD). We fit *Explanatory* models simultaneously to the student and MTURK groups, and then made predictions on individual-level choices for each participant in the SUDs group using their state anxiety and trait impulsivity scores alone. Further details on the model comparison measures are included in the Supplementary Text.

Interpreting Bayesian Models

[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 Eq. 3.

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, where every value falling inside the interval is more probable than any value falling outside of the interval. Using the *Trait Descriptive* model as an example, a 95% HDI = [0.15, 0.3] on β_1 would indicate that the most probable 95% of values for β_1 fall between .15 and .3. Intuitively, it is useful to imagine the behavior of the HDI as we use a smaller and smaller x%. As $x \to 0$, the interval converges to the single most probable parameter value (i.e., the mode of the distribution). As $x \to 100$, 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 only make probability statements 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?*", etc.). Again using the *Trait* model as an example, a 95% HDI = [0.15, 0.30] on β_1 would indicate strong evidence for a positive effect, given that the range of 95% most probable values are well above 0, and the 95% range is itself relatively narrow (i.e., the estimate is precise). Conversely, a 95% HDI = [-0.3, 0.4] on β_1 would indicate 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/differences with respect to frequentist confidence intervals, see chapter 11 of

Kruschke (2015).

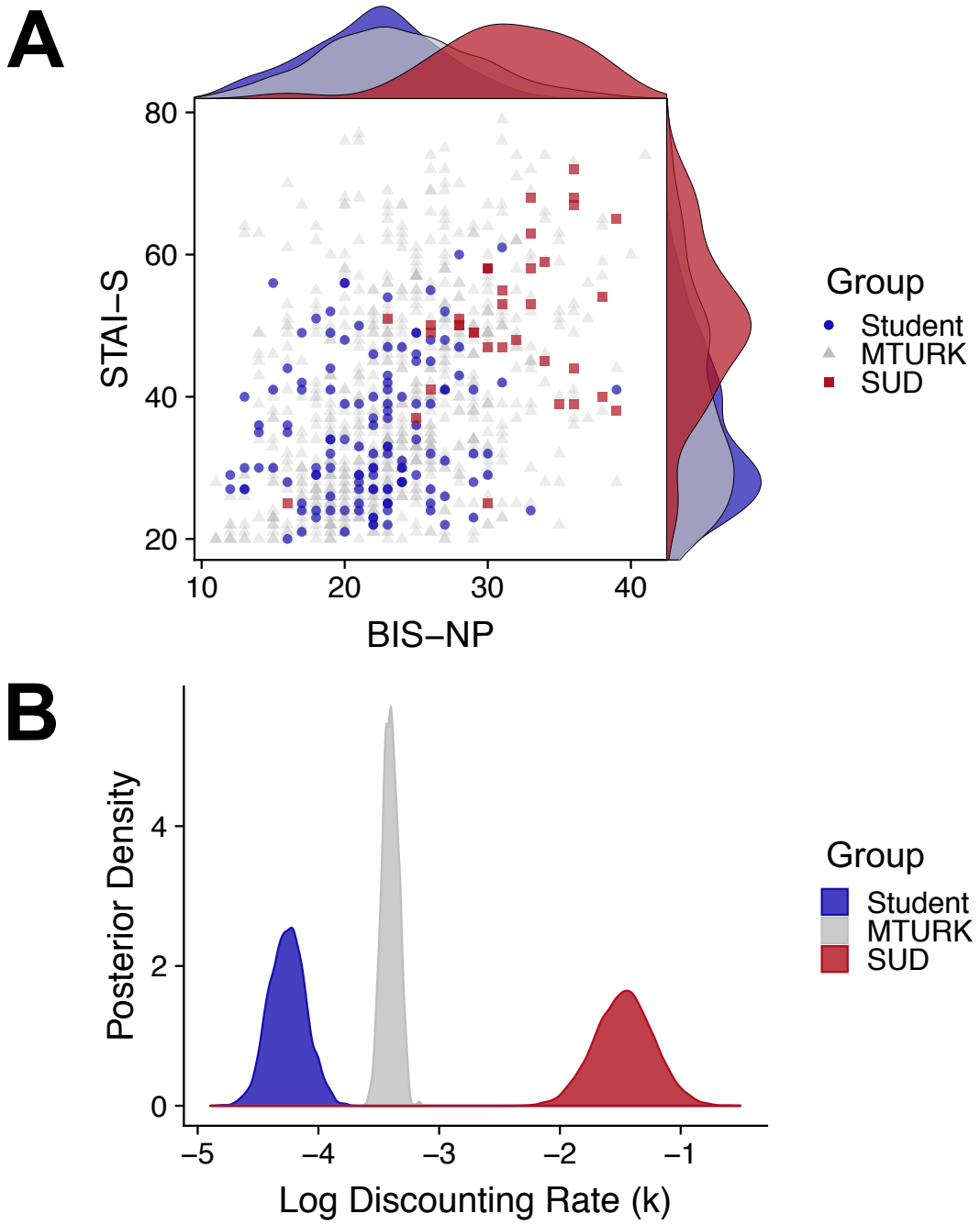
Results

State, Trait, and Behavioral Differences

Here, we report highest density intervals (HDIs) on estimated differences in mean trait impulsivity (BIS-NP) and state anxiety (STAI-S) scores between groups, in addition to estimated group-level discounting rates for each group. As depicted in Fig. 2A, trait impulsivity varied across groups in the anticipated direction. Students had lower BIS-NP scores than both the MTURK, 95% $HDI_{student\text{-}MTURK}$ = [-2.67, -0.87], and SUDs groups, 95% $HDI_{student\text{-}SUD}$ = [-11.36, -7.61]. The MTURK group also had lower BIS-NP scores than the SUDs group, 95% $HDI_{MTURK\text{-}SUD}$ = [-9.42, -5.92]. Results were similar for state anxiety (see Fig. 2A), where students had lower STAI-S scores than both the MTURK, 95% $HDI_{student\text{-}MTURK}$ = [-6.16, -2.23], and SUDs groups, 95% $HDI_{student\text{-}SUD}$ = [-19.82, -11.36]. The MTURK group also had lower STAI-S scores than the SUDs group, 95% $HDI_{MTURK\text{-}SUD}$ = [-15.29, -7.31]. Frequentist *t-test*s offered the same conclusions[6]. In the *Base Descriptive* model, discounting rates varied as predicted across groups (Fig. 2B). The SUDs 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.

[6]Traditional frequentist *t-test*s showed that students had lower BIS-NP scores than both the MTURK, $t(196.7) = -3.91$, $p < .001$, $d = -0.56$, and SUDs groups, $t(51.5) = -10.02$, $p < .001$, $d = -2.79$, and the MTURK group had lower BIS-NP scores than the SUDs 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 SUDs groups, $t(49.5) = -7.40$, $p < .001$, $d = -2.10$, and the MTURK group had lower STAI-S scores than the SUDs group, $t(38.6) = -5.74$, $p < .001$, $d = -1.85$.

Fig. 2 Trait impulsivity, state anxiety, and behavioral impulsivity across groups.



(A) Scatterplot with marginal distributions for summed scores of trait impulsivity (BIS-NP) and state anxiety (STAI-S) across groups. Pearson's correlations between BIS-NP and STAI-S scores for each group were $r_{student} = .17$, $r_{MTURK} = .38$, and $r_{SUD} = .39$. (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.

Descriptive Models

Model comparison of the *Base* versus *Trait Descriptive* models showed that the *Trait Descriptive* model more effectively accounted for student ($LOOIC_{Base}$ - $LOOIC_{Trait}$ = 2.5, $SE_{Difference}$ = 6.9), MTURK ($LOOIC_{Base}$ - $LOOIC_{Trait}$ = 24.8, $SE_{Difference}$ = 25.9), and SUDs ($LOOIC_{Base}$ - $LOOIC_{Trait}$ = 68.4, $SE_{Difference}$ = 74.5) participants' DDT performance[7]. This suggests that main and/or dependent effects of trait impulsivity and state anxiety accounted for meaningful variance in individual-level decision-making[8]. The difference in LOOIC between models for students was lowest relative to the *SE* 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_{β_3} = [-0.34, 0.29]), whereas both the MTURK (95% HDI_{β_3} = [-0.25, 0.00]) and SUDs (95% HDI_{β_3} = [-0.98, -0.27]) samples showed evidence of moderating effects (see Fig. S1).

Additionally, both student (95% HDI_{β_1} = [0.16, 0.78]) and MTURK (95% HDI_{β_1} = [0.11, 0.40]) groups showed strong correspondences between non-planning impulsivity (BIS-NP) and discounting rates, conditioned on state anxiety and their interaction (Fig. S1). Conversely, the SUDs (95% HDI_{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, with some evidence for a negative association among students (95% HDI_{β_2} = [-0.57, 0.04]), and some evidence for a positive relationship in the MTURK (95% HDI_{β_1} = [-0.03,

[7]Because lower LOOIC values indicate better model performance, positive values for the difference of $LOOIC_{Base} - LOOIC_{Trait}$ indicate better performance for the *Trait Descriptive* model.
[8]We fit a main effects only Trait Descriptive model (i.e., no impulsivity-anxiety interaction term) in addition to the full interaction model, which we describe in the Sensitivity Analysis section of the Supplemental Text. Results were consistent with those reported in text.

0.26]) and SUDs (95% HDI_{β_1} = [-0.05, 0.89]) groups. Fig. 3A shows *Descriptive Trait* model-predicted discounting rates for each group at varying levels of BIS-NP and STAI-S, which makes the moderating effect of anxiety on the association between impulsivity and discounting more clear. In the figure, interactions are evident for both the MTURK and SUDs groups, such that discounting rates are highest when individuals endorse both low levels of state anxiety and high levels of trait impulsivity. In contrast, the student group showed no interaction, with discounting rates 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 anxiety, although there is some apparent discrepancy between 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.

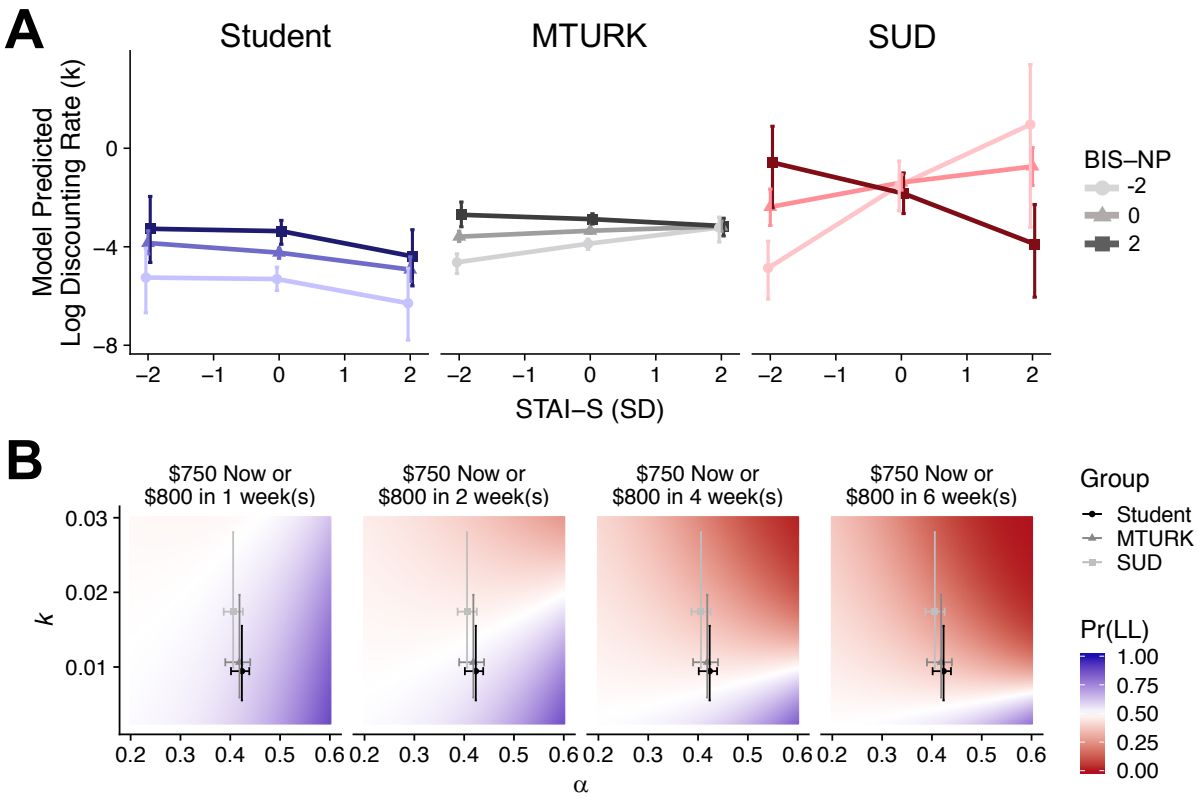
Explanatory Models

Model comparison of the *Base* versus *Trait Explanatory* models showed that the *Trait Explanatory* model—where α and k are assumed to relate to state anxiety and trait impulsivity, respectively—provided the best out-of-sample predictions (LPPD = -4098) compared to the *Base* (LPPD = -5011) and *Trait Incongruent* (LPPD = -4441) *Explanatory* models[9]. In addition to best predicting performance across the whole SUD group, the *Trait Explanatory* model outperformed competing models for individual participants in the SUDs group (see Fig. S3). 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.

[9]LPPD closer to 0 indicates better predictive performance within the out-of-sample SUD group. See the Supplementary Text for further details on interpretation of LPPD.

Posterior distributions for parameters of the *Trait Explanatory* model are shown in Fig. S4. We found strong evidence for a negative relationship between the STAI-S and α, such that increases in state anxiety predict attenuated reward valuation (95% $\text{HDI}_{\beta_{\alpha 1}}$ = [-0.045, -0.011]), consistent with both (1) relations between anxiety and indecision/increased risk sensitivity predicted by RST (Corr, 2004, 2008), and (2) previous studies showing that state anxiety increases risk aversion. Additionally, we found a positive association between BIS-NP and k, such that increases in trait impulsivity predicted increases in delay discounting (95% $\text{HDI}_{\beta_{k1}}$ = [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, Fig. 3B demonstrates the estimated group-level effects of state anxiety and trait impulsivity on four different example choices from the DDT.

Fig. 3 Interaction of BIS-NP and STAI-S in predicting discounting rates for both *Trait* models.



(A) Model-predicted discounting rates for different combinations (i.e., 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% HDIs of model predicted discounting rates (i.e., 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. Of note, Bayesian intervals indicate probabilities. Although low BIS-NP, high anxious participants in the SUD group appear to have steeper discounting rates relative to others on average, there is a non-negligible probability that they also have lower rates (i.e., the HDI spans both above and below others). When accounting for such uncertainty, MTURK and SUD groups show 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 (i.e., μ_k and μ_α from Eq. 5; see also Eq. S3 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, where α and k are negatively and positively associated with STAI-S and BIS-NP scores, respectively.

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 et al., 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, Donkin, & Newell, 2017), and that anxiety inductions increase risk aversion (Engelmann, Meyer, Fehr, & Ruff, 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 the individual's discounting rate (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 (i.e., "non-impulsive") option. Future studies might manipulate state anxiety experimentally among those who are low vs. 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 those

with substance use disorders.

Our findings also have broader implications for traditional methods used to test hypotheses in psychopathology research. For example, psychopathology research efforts continue to shift away from single-level analyses and toward multiple-level analysis in development and validation of theories of mental illness (e.g., Beauchaine & McNulty, 2013; Cicchetti & Dawson, 2002; Cicchetti, Ackerman, & Izard, 1995). Oftentimes, researchers assume 1:1 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 et al., 2018). We demonstrated this across self-report and behavioral measures, but similar effects are 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 data), which can lead to biased inferences and overconfidence in the wrong parameter values (e.g., *beta* weights from a 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 SUDs sample were smaller than the MTURK sample. Smaller samples are underpowered relative to larger samples, and may also be influenced more by outliers and/or sample-specific

characteristics. Of note, 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). Additionally, 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 SUDs sample in particular should be replicated in future research.

Second, the student, MTURK, and SUDs groups likely differ in other 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 section *State, Trait, and Behavioral Differences*; Beauchaine & Hinshaw, in press; 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 over-interpretation 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 design 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 the joint effects of state anxiety and trait impulsivity on impulsive decision-making (cf. Turner et al., 2018; Turner, Van

Maanen, & Forstmann, 2015). Although we did not collect reaction time measures in this study, 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 concurrent levels of anxiety. Such reduced discounting leads to more optimal, future-oriented decisions in the DDT. Further, our findings from the *Trait Explanatory* model reveal a mechanism through which anxiety may serve as a protective factor against impulsive behavior in those with externalizing spectrum disorders, yet lead to relatively more impulsive behavior for those with low trait impulsivity. Future research may use experimental manipulations to determine if within-subject anxiety inductions can decrease the value of drug cues in high trait-impulsive individuals with substance use disorders. More broadly, hierarchical Bayesian analysis offers a principled way to explore how mechanisms at one level of analysis interact to produce observations at another level, which can shed light on the dimensional neural, cognitive, and/or trait-level constructs that underlie traditionally discretized behavioral syndromes.

Declarations

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Availability of data and materials

Upon publication, all de-identified data along with the R and Stan codes used to reproduce our results and figures will be uploaded to our GitHub repository (https://github.com/CCS-Lab).

Authors' contributions

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

Ethics approval and consent to participate

All participants gave informed consent prior to the study, and the study protocol (#2016H0108) was approved by The Ohio State Biomedical Sciences Institutional Review Board.

Consent for publication

Not applicable.

Competing interests

None declared.

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