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Common Genetic Influences on Impulsivity Facets are Related to Goal Management, Psychopathology, and Personality

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Abstract

In this study, we characterized the genetic/environmental commonality and heterogeneity of impulsivity facets and tested the hypothesis that goal-management is central to their common variance. 764 young-adult twins completed the UPPS-P Impulsive Behavior Scale and measures of goal management, personality, and psychopathology. We found common genetic influences across all impulsivity facets except sensation seeking. These impulsivity genetic influences explained 40.0% of variance in goal-management ability. Other results supported three hypotheses concerning heterogeneity: that sensation seeking is independent of other facets, that urgency is more related to psychopathology than lack of premeditation, and that lack of perseverance is more similar to urgency than lack of premeditation. Thus, impulsivity facets show considerable heterogeneity in addition to common variation related to goal-management abilities.

Keywords

impulsiveness; heritability; self-control; cognitive control; SEM

Introduction

Higher impulsivity is associated with a wide range of negative outcomes, including greater procrastination, substance use, and internalizing and externalizing psychopathology (Coskunpinar, Dir, & Cyders, 2013; Dick et al., 2010; Grano et al., 2007; Steel, 2007;

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Swann, Bjork, Moeller, & Dougherty, 2002), and poor goal management (Gustavson, Miyake, Hewitt, & Friedman, 2014). Although its definition has varied (Evenden, 1999), impulsivity generally refers to a tendency to act without premeditation and without control (Cloninger, Svrakic, & Przybeck, 1993; Eysenck & Eysenck, 1985; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; Tellegen, 1982). However, impulsivity is also multifaceted (Smith, Fischer, Cyders, Annus, & Spillane, 2007). These facets are not always correlated with other psychological traits to the same degree (Cyders et al., 2007; Fischer, Smith, & Cyders, 2008; Johnson, Carver, & Joormann, 2013), and some are not even correlated with one another (Cyders & Smith, 2007; Whiteside & Lynam, 2001). Thus, there is commonality and heterogeneity in impulsivity, but it is still somewhat unclear what unites and distinguishes the array of traits that index impulsivity. Here, we elucidate the structure of impulsivity by examining the interrelations among impulsivity facets and their associations with other constructs, such as goal management, within a genetically informative design.

The construct of impulsivity features prominently in many theories of personality (Evenden, 1999), and there are many models of human impulsive behavior (Shulman, Smith, et al., 2016; Stanford et al., 2009; Whiteside & Lynam, 2001). Table 1 summarizes these models as well as constructs of sensation seeking and self-control, as they are sometimes, but not always, considered closely related to impulsivity (Lecrubier, Braconnier, Said, & Payan, 1995; Shulman, Harden, Chein, & Steinberg, 2016; Tangney, Baumeister, & Boone, 2004; Whiteside & Lynam, 2001). In summary, most theories of impulsivity emphasize its multidimensionality, and although different terms are used, some aspects are consistent across many theories (e.g., acting without thinking, giving into temptations or desires, sensation seeking). Impulsivity facets are also often classified under a range of personality traits including conscientiousness, extraversion, and novelty seeking (Sharma, Markon, & Clark, 2014).

Evaluating hypotheses in the context of a genetically-informative twin sample can extend our understanding of impulsivity facets in novel ways. First, we can examine basic research questions such as whether there are genetic influences that are common across all facets. Meta-analytic estimates suggest that half of the variance in various impulsivity measures can be explained by genetic influences (heritability = .50; Bezdjian, Baker, & Tuvblad, 2011), but few studies have examined the genetic and environmental overlap between impulsivity facets (Hur & Bouchard, 1997; Niv, Tuvblad, Raine, Wang, & Baker, 2012). Similarly, although there are few genome-wide associations studies of impulsivity to date (Bevilacqua & Goldman, 2013; Ebejer et al., 2013; Linnér et al., 2018), those studies also have not thoroughly considered the multifaceted nature of impulsivity. To the extent that there is heterogeneity among measures and distinct genetic influences among facets, this will hurt statistical power and make it difficult to interpret results.

It will also be useful to examine evidence for environmental influences underlying multiple impulsivity facets, including whether the genetic and environmental structures are similar or distinct. Environmental influences can include measurement error, but if environmental influences common to multiple impulsivity facets relate to other constructs like psychopathology, this would suggest that they index true environmental risk factors.

Moreover, to the extent that some facets are more strongly accounted for by common genetic versus environmental influences, they may be more useful as an index of genetic risk for impulsivity than other facets, especially in studies that are not genetically informative.

Finally, analyses of twin data can inform how impulsivity is related to other important constructs. Regression-based approaches are limited in that they emphasize the unique contribution of predictor variables, and do not explicitly model the contribution of variance shared across multiple predictors. In addition to providing information about the genetic and environmental covariance, multivariate genetic models can provide fine-grained estimation of how common and unique sources of variance across impulsivity facets relate to other psychological outcomes. For example, the genetic relations between impulsivity and internalizing psychopathology could be due to common genetic influences on all facets, or genetic influences specific to emotionally-triggered impulsivity (Johnson et al., 2013).

Hypotheses of the Current Study

The central goal of the current work is to better understand the genetic and environmental commonality across impulsivity facets and their genetic associations with other psychological constructs such as goal management. Here we use the UPPS-P (Lynam, Smith, Whiteside, & Cyders, 2006; Whiteside & Lynam, 2001), a commonly used, reliable, and valid measure of impulsivity with five facets (i.e., positive and negative urgency, lack of premeditation, lack of perseverance, and sensation seeking). After fitting a genetic and environmental model of the five facets (and testing whether these genetic/environmental structures are similar or distinct), we examine two hypotheses aimed to elucidate the factors that account for the commonality (Hypothesis 1) and heterogeneity (Hypotheses 2a-2c) of these facets. We evaluate these hypotheses by examining genetic/environmental associations between common and unique sources of variance on impulsivity facets and six psychological constructs that have been previously found to be associated with impulsivity: goal-management failures, procrastination, neuroticism, extraversion, internalizing psychopathology symptoms, and externalizing psychopathology symptoms. Each construct (except extraversion) was assessed at the level of latent variables, improving power and generalizability of the findings.

Goal-management abilities underlie common variance among impulsivity facets (Hypothesis 1).

We hypothesize that an inability to effectively engage goal-management processes is a common thread linking the five facets of impulsivity in the UPPS-P (Hypothesis 1). In other words, facets of impulsivity should be united to the extent that they capture goal-management abilities (or lack thereof). Giving in to one's temptations (negative and positive urgency), acting without thinking (lack of premeditation), and giving up on difficult tasks (lack of perseverance) each may represent a situation where a goal is neglected in favor of a short-term desire, and this may be true even in sensation seeking (e.g., if an individual seeks exciting environments or situations at the cost of long-term goals). Thus, we predict that goal management abilities will be most strongly associated with the genetic and environmental factors that capture the common variance among impulsivity facets.

Goal-management ability is also a complex construct with a long history (e.g., Latham & Locke, 1991; Locke & Latham, 2006). Here, we focus on failures to engage in goal management in everyday situations using two self-reported measures that were strongly correlated with urgency and lack of premeditation in earlier work on this sample (Gustavson et al., 2014). These measures incorporate a range of abilities including failures to retrieve task goals and maintain task representations, tendencies to experience attentional lapses, memory and psychomotor slips, and “everyday task failures” (Wallace, 2004). Previous work demonstrated that everyday goal-management failures accounted for most of the phenotypic (74%) and genetic (68%) covariance between procrastination and impulsivity (a latent factor comprising urgency and lack of premeditation only), suggesting the two constructs are associated through their ability to use goals to regulate actions (Gustavson et al., 2014). These goal-management failures were in turn genetically associated with poor cognitive executive functions, including response inhibition and working memory updating (Gustavson, Miyake, Hewitt, & Friedman, 2015), providing further support for this hypothesis. However, this previous work did not consider how goal-management abilities unite impulsivity facets (including lack of perseverance and sensation seeking).

We focus on self-reported measures in the current study in part to be most consistent with the assessment of impulsivity and other constructs. There are other paradigms that putatively assess motor impulsivity using the matching familiar figures test (or prepotent response inhibition tests). However, there has been a long debate about whether these tasks actually assess impulsivity (Block, Block, & Harrington, 1974), especially given the low correlations between self-report and behavioral measures (Duckworth & Kern, 2011; Sharma et al., 2014; Toplak, West, & Stanovich, 2013), even at the level of latent variables (MacKillop et al., 2016). Thus, we focus on self-reported measures that are most likely to capture a range of goal-management processes that we hypothesize underlie much of the common variance among impulsivity facet.

Understanding the heterogeneity of impulsivity (Hypotheses 2a-2c).

We also explore three hypotheses examining factors that may account for separability of impulsivity facets as well as their overlap with personality and psychopathology.

Sensation seeking versus other facets (Hypothesis 2a).—Sensation seeking is the least correlated with other UPPS-P subscales (Lynam et al., 2006; Whiteside & Lynam, 2001) and is often uncorrelated with other traits that are associated with impulsivity (Fischer et al., 2008; Steel, 2007), suggesting that its inclusion within the domain of impulsivity may represent the “jingle” fallacy (Block, 1995; Whiteside & Lynam, 2001) wherein two very different constructs are often referred to with the same label. Thus, sensation seeking and impulsivity may represent entirely independent processes or dual systems (Shulman, Harden, et al., 2016; Shulman, Smith, et al., 2016; Steinberg et al., 2008), with sensation seeking capturing bottom-up processing of rewards and impulsivity reflecting the inability to exert cognitive control (i.e., top-down processing). This is consistent with our view that goal-management processes link other impulsivity facets, but are not as readily related to the tendency to seek out exciting situations (unless this is at the expense of long-term goals), although there is some evidence for genetic associations between sensation seeking and the

control (vs. impulsiveness) scale of the Multidimensional Personality Questionnaire (Hur & Bouchard, 1997).

We hypothesize that sensation seeking will have the smallest factor loadings on the common genetic and environmental factors, thus bearing little resemblance to the other facets of impulsivity (Hypothesis 2a). Even if sensation seeking is phenotypically associated with the same constructs as other facets, we expect these associations to remain significant in a regression model controlling for other facets (thus representing distinct sources of variance).

Emotional versus non-emotional impulsivity (Hypothesis 2b).—Urgency and (lack of) premeditation are perhaps the most commonly studied facets of impulsivity, but they have differential relations to psychopathology (for meta-analysis, see Berg, Latzman, Bliwise, & Lilienfeld, 2015). Specifically, emotionally-triggered impulsivity (urgency) is associated with both internalizing and externalizing psychopathology (Berg et al., 2015; Grano et al., 2007; Whiteside & Lynam, 2003), but non-emotional aspects of impulsivity (lack of premeditation) may be only weakly associated with internalizing traits (Berg et al., 2015; Johnson et al., 2013). Similarly, a meta-analysis including the UPPS and other self-reported measures (Sharma et al., 2014) indicated that only negative urgency loaded on a neuroticism-related factor (with negative urgency, lack of premeditation, and lack of perseverance loading on a conscientiousness factor and sensation seeking on an extraversion-related factor).

We hypothesize that emotional context is the central feature distinguishing urgency from premeditation (Hypothesis 2b). Thus, we predict that urgency (positive and negative) will be more strongly related to internalizing psychopathology and neuroticism than premeditation will be. We also predict that urgency will show unique genetic or environmental associations with these constructs after controlling for common genetic and environmental factors across all facets. Externalizing and procrastination may also demonstrate unique associations with urgency to the extent that they are influenced by one's ability to cope with negative emotions and regulate mood (Eisenberg et al., 2009; Sirois & Kitner, 2015; Sirois & Pychyl, 2013; Zeman, Shipman, & Suveg, 2002), but we expect these associations with externalizing and procrastination to be more similar across urgency and premeditation facets, given previous work (Grano et al., 2007; Gustavson et al., 2017; Whiteside & Lynam, 2003).

Incorporating lack of perseverance (Hypothesis 2c).—Lack of perseverance is studied less frequently than other facets. Understanding how it fits in with other facets may hinge on its similarity with lack of premeditation and on whether it too has an emotional component, leading to two alternate hypotheses. In one sense, lack of premeditation and perseverance are two sides of the same coin, where individuals who act spontaneously may do so at the expense of giving up on other tasks. This is similar to long-held views on procrastination and impulsivity (Ferrari, 1993; Gustavson et al., 2014), as important tasks are delayed in favor of short-term temptations. By this view, lack of perseverance is akin to procrastination (both reflecting avoiding or giving up on difficult tasks), and should therefore be more strongly associated with procrastination than other impulsivity facets, but otherwise demonstrate similar associations with other constructs as lack of premeditation. Indeed, lack of premeditation and lack of perseverance have been modeled together in a

“Deficits in Conscientiousness” factor in the same model as an urgency factor (positive and negative) and sensation seeking (Cyders & Smith, 2007), providing some evidence that premeditation and perseverance have some additional genetic and/or environmental covariance beyond that shared with other facets.

Alternatively, lack of perseverance may have a stronger emotional component than premeditation. Whereas lack of premeditation does not necessarily depend on one’s emotional state, an individual’s ability to persist on difficult tasks likely depends on his/her ability to deal with frustration and failure. This alternative hypothesis predicts that to the extent that urgency (especially negative urgency) diverges from lack of premeditation, so will lack of perseverance. These hypotheses are not entirely mutually exclusive. To the extent that procrastination has an emotional component (Sirois & Kitner, 2015; Sirois & Pychyl, 2013), perseverance may be closely related to procrastination and yet still be more similar to urgency than lack of premeditation).

Method

Participants

Analyses were based on 754 individuals (398 females, 356 males) from same-sex twin pairs (193 full monozygotic [MZ] twin pairs, 171 full dizygotic [DZ] twin pairs, and 26 unpaired twins) in the Colorado Longitudinal Twin Sample ($M = 22.80$ years, $SD = 1.27$, Range = 21.10 to 28.00) who completed UPPS-P Impulsive Behavior Scale (Lynam et al., 2006). An additional 10 individuals (4 females, 6 males) were included in the analyses involving other constructs (all completed at least part of the diagnostic interviews and about half had data for other relevant questionnaires). Although they did not have UPPS-P data (and thus do not contribute to estimates of the genetic/environmental correlations between impulsivity and these constructs) their inclusion informs the thresholds of psychopathology variables, as well as the factor loadings and heritability estimates for other latent variables. The sample was recruited through the Colorado Department of Health based on twins born between 1984 and 1990 as is representative of the population of Colorado at that time. For more information on the sample characteristics, see Rhea, Gross, Haberstick, and Corley (2013).

Measures

Development of latent variables of goal-management failures, procrastination, neuroticism, internalizing psychopathology, and externalizing psychopathology in this sample has been detailed in previous work (Gustavson et al., 2017; Gustavson et al., 2014), but is summarized here. Genetic/environmental models of each construct alone are displayed in Figure 1. Although previous work described some associations between impulsivity and these constructs, analyses focused on associations between procrastination and other constructs. This work also did not examine measures of lack of perseverance, sensation seeking, or extraversion, and did not consider genetic/environmental influences unique to individual impulsivity facets whatsoever.

Impulsivity.—Impulsivity was assessed with 35 of the 59 items of the UPPS-P Impulsive Behavior Scale (Lynam et al., 2006). This shortened UPPS-P scale included five subscales: 7

items assessing negative urgency, 7 items assessing positive urgency, 8 items assessing lack of premeditation, 6 items assessing lack of perseverance, and 7 items assessing sensation seeking. The UPPS-P was condensed to accommodate time restrictions. As described below, the intercorrelations between subscales remained highly consistent with those from previous work. Example items for each subscale are displayed in Table 2.

Goal management.—Goal-management failures, or the tendency to forget to activate, maintain, and retrieve task goals in everyday situations, were assessed with (a) the 25-item Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, FitzGerald, & Parkes, 1982) and (b) the log-transformed average of items from the short-term (14-items), long-term (14-items), and internally cued (10-items) subscales of the Prospective Memory Questionnaire (PMQ; Hannon, Adams, Harrington, FriesDias, & Gipson, 1995). Higher scores indicated more goal-management problems.

Personality.—Trait levels of procrastination were assessed with (a) the 20-item General Procrastination Scale (GPS; Lay, 1986); (b) the average of the external control, goal neglect, and effort avoidance subscales (7 items each) of the Volitional Components Inventory (VCI; Kuhl & Fuhrmann, 1998), and (c) the 12-item prospective and decision-related action orientation versus hesitation subscale of the Action Control Scale (ACS; Kuhl, 1994). Higher scores reflect more procrastination.

Assessments of other personality variables were not completed at the same time as other constructs. Neuroticism was assessed at age 17 ($M = 17.25$, $SD = .64$, Range = 16.13 to 20.08) with the average of 9 items from the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975) and at age 21 ($M = 21.09$, $SD = 2.05$, Range = 18.00 to 28.03) with the average of the 12-item Revised Eysenck Personality Questionnaire (Francis, Brown, & Philipchalk, 1992). Extraversion was assessed with the average of 9 items from the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975) as part of the same assessment as neuroticism (mean age 17). This was the only available assessment of extraversion, so it could not be modelled using a latent variable. Higher scores reflected more neuroticism or extraversion.

Psychopathology.—Internalizing and externalizing psychopathology were measured using structured interviews from the Diagnostic Interview Schedule-IV (Robins et al., 2000), which assesses symptoms and diagnoses of common psychiatric disorders from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (1994). Because summed symptom counts were positively skewed, all symptom count measures were binned into ordinal variables for each disorder, described in previous work (Gustavson et al., 2017) and summarized here. This method is preferred to transformations because it produces unbiased estimates of censored data in twin models (Derks, Dolan, & Boomsma, 2004).

Internalizing was measured with symptom endorsement of generalized anxiety disorder (GAD) and major depressive disorder (MDD) of the DIS-IV. A score of 0 was assigned to subjects who endorsed no symptoms ($n = 676$ for GAD, $n = 574$ for MDD), a score of 1 was assigned to subjects who reported symptoms but did not reach the criteria for diagnosis ($n =$

52 for GAD, $n = 90$ for MDD), and a score of 2 was assigned to subjects who met criteria for diagnosis ($n = 35$ for GAD, $n = 99$ for MDD).

Externalizing psychopathology was measured using lifetime symptom endorsement for antisocial personality disorder (ASPD), and lifetime symptom endorsement from the Composite International Diagnostic Interview-Substance Abuse Module (Cottler, Robins, & Helzer, 1989) for four different substances: alcohol, tobacco, cannabis, and illicit substance use (the substance with the highest number of symptoms endorsed across all illicit substances used by a given individual). For ASPD, three bins were created: no symptoms ($n = 417$), symptoms but no diagnosis ($n = 199$), and diagnosis ($n = 147$). Diagnosis of ASPD did not include the criterion of conduct disorder before age 15. Alcohol use disorder symptoms were binned into five bins: 0 symptoms ($n = 327$), 1 symptom ($n = 138$), 2-3 symptoms ($n = 164$), 4-5 symptoms ($n = 78$), and 6 or more symptoms ($n = 55$). Tobacco use disorder symptoms were binned into three bins: 0 symptoms ($n = 509$), symptoms but no diagnosis ($n = 112$), and diagnosis ($n = 141$). Cannabis use disorder symptoms used the same bins as tobacco use: 0 symptoms ($n = 588$), 1 symptom ($n = 76$), and 2 or more symptoms ($n = 98$). Illicit substance use symptoms were binned into two bins: 0 symptoms ($n = 692$) and 1 or more symptoms ($n = 70$).

The final two bins for GAD, MDD, and alcohol use disorder were collapsed in genetic analyses due to reduction in bin sized after separating into MZ and DZ groups.

Data Analysis

Analyses were conducted using Mplus (Muthén & Muthén, 2010-2012), which accounts for missing observations using full-information maximum likelihood. Model fit was determined based on chi-square tests (χ^2), the root mean error of approximation (RMSEA), and the Comparative Fit Index (CFI). Good-fitting models had χ^2 values less than two times the degrees of freedom, RMSEA values less than .06, and CFI values greater than .95 (Hu & Bentler, 1998). Significance of individual parameter estimates were established with χ^2 difference tests by fixing that parameter to zero. For all phenotypic models, standard errors and chi-squares were adjusted for clustering within families, and the χ^2_{diff} tests were appropriately scaled based on scaling factors provided in the output (Satorra & Bentler, 2001).

In all phenotypic and genetic/environmental models presented here, we accounted for sex differences by regressing all measures on sex. Sex differences for internalizing and externalizing psychopathology, for example, were reported in previous work on this sample and consistent with previous literature (Eaton et al., 2012; Gustavson et al., 2017).

Assumptions in genetic analyses.—Genetically informed models were based on standard assumptions in twin designs. Additive genetic influences (A) are correlated at 1.0 in MZ twin pairs and 0.5 in DZ twin pairs because MZ twins share 100% of their alleles identical-by-descent and DZ twins share, on average, 50% of their alleles identical-by-descent. Common/shared environmental influences (C) correlate 1.0 for both MZ and DZ twins because they are environmental factors that affect both pairs of twins in a family equally. Nonshared environmental influences (E) do not correlate, by definition. We also

assume equal means and variances within pairs and across zygosity, and assume that identical and fraternal twins experience similar environments (i.e., the “equal environments assumption”). These standard assumptions for univariate twin analyses extend to the multivariate analyses described here.

Although previous work has identified evidence for non-additive genetic influences on impulsivity (Bezdjian et al., 2011), we did not estimate non-additive influences due to a lack of power (Martin, Eaves, Kearsley, & Davies, 1978). Therefore, our estimates of additive genetic influences should be interpreted as a combination of additive and non-additive genetic influences (broad-sense heritability) rather than only additive genetic influences (narrow-sense heritability).

Model fitting approach.—First, we identified the best-fitting genetic and environmental model of the five impulsivity facets by comparing independent pathway and common pathway models (for examples, see supplement Figure S1). In both models, there are genetic (A_{Imp}), shared environmental (C_{Imp}), and nonshared environmental factors (E_{Imp}) that capture common variance between all facets of impulsivity. There are also unique sets of genetic and environmental influences on each facet (e.g., A_{NU} , E_{NU} for negative urgency) that capture variance not explained by the common factors. The key difference is that the common pathway model constrains the genetic and environmental correlations between facets of impulsivity to be proportional to factor loadings on an Impulsivity latent factor, whereas the independent pathway model allows for different genetic and environmental structures. We also compared these models with a genetic/environmental version of the three-factor model (urgency, deficits in conscientiousness, and sensation seeking) described by Cyders and Smith (2007).

After identifying the best-fitting model of impulsivity facets, this model was combined with the genetic model of each of the other constructs (in separate analyses for each construct). In these analyses, variance in a given construct was decomposed, using Cholesky paths, into variance explained by genetic/environmental factors common to all facets of impulsivity (A_{Imp} , E_{Imp}), genetic/environmental paths unique to some impulsivity facets (e.g., A_{NU} , E_{NU}), and unique genetic/environmental influences on that construct (A_{Unique} , E_{Unique}). An example of this model is displayed in Figure 3 (for goal-management failures) and described further below.

In these models, shared environmental paths were estimated only for internalizing and externalizing psychopathology because these were the only traits to demonstrate significant shared environmental influences (Gustavson et al., 2017). We also present corresponding phenotypic models where the five impulsivity facets are included in a model with each other psychological construct (also in separate analyses for each construct). Correlational models and regression models are presented, but the regression models must be interpreted with caution as some impulsivity facets are strongly correlated with one another, and the regression model does not explicitly model the contribution of common versus unique sets of variance as in the genetically-informative model (Figure 3).

Sample size and power considerations.—We analyzed data from an existing twin study, including all subjects with available data. Based on prior work (e.g., Cyders & Smith, 2007), we expected moderate to strong phenotypic correlations among most impulsivity facets and similar correlations between impulsivity facets and the other latent constructs ($r = .20$ to $.50$). We estimate that we had $>80\%$ power to detect phenotypic correlations of $r = .10$ or greater (Soper, 2018), and genetic correlations of $r = .30$ or greater (Neale et al., 2016). Power for genetic correlations with other constructs is further increased when shared environmental correlations can be assumed to be zero (all constructs except psychopathology) and is always greater for nonshared environmental correlations. Thus, this study was well-powered to examine the relations among the examined constructs.

Results

Descriptive Statistics and Preliminary Analyses of Associations Among Impulsivity Facets

Descriptive statistics for all measures are displayed in Table 3. The phenotypic correlations between the five UPPS-P subscales are displayed in Table 4 and were similar to those described in previous work (Cyders & Smith, 2007; Whiteside & Lynam, 2001). All facets of impulsivity were positively correlated with one another ($r_s = .08$ to $.61$) except that sensation seeking was weakly negatively correlated with lack of premeditation ($r = -.10$). In the supplement, these correlations are decomposed into genetic, shared environmental, and nonshared environmental correlations using a basic Cholesky decomposition (Table S1).

Genetic and Environmental Model of Impulsivity

The best-fitting genetic model of impulsivity is displayed in Figure 2. There are a few noteworthy results from this independent pathway model. First, the genetic impulsivity factor (A_{Imp}) explained a significant portion of variance in all facets of impulsivity except sensation seeking. The nonshared environmental impulsivity factor (E_{Imp}) explained a significant portion of variance in all impulsivity facets, but accounted for only 4.4% of the variance in sensation seeking. These findings confirm that there are overlapping genetic and environmental influences across most impulsivity facets. There was also evidence for shared environmental influences on some facets of impulsivity (positive urgency, lack of premeditation, and sensation seeking). In total, shared environmental influences explained 25% of the variance in sensation seeking, but explained less than 7% of the variance in any of the other facets.

This independent pathway model fit well, $\chi^2(113) = 152.72$, $p = .008$, RMSEA = .043, CFI = .958, and did not fit worse than the Cholesky decomposition (a baseline model), $\chi^2(18) = 8.86$, $p = .963$. Importantly, a common pathway model fit significantly worse than this independent path model, $\chi^2(8) = 25.72$, $p = .001$, suggesting that the genetic and environmental structures on impulsivity are not identical. Similar results were observed even when sensation seeking was excluded from the common factor.¹ The three-factor model

¹In this model, all facets except sensation seeking loaded onto a common factor, with genetic/environmental covariance paths between the common factor and sensation seeking. This model still fit worse than the independent pathway model displayed in Figure 2, $\chi^2(6) = 24.05$, $p = .001$, suggesting that the genetic and environmental structures of impulsivity facets are not identical even when sensation seeking is excluded from the common factor.

described by Cyders and Smith (2007) did not have acceptable fit (see Hypothesis 4), $\chi^2(112) = 207.63, p < .001$, RMSEA = .066, CFI = .899. In all genetic models, we detected two additional paths necessary to improve model fit, capturing nonshared environmental correlations between negative urgency and positive urgency ($r_e = .52$) and between lack of perseverance and sensation seeking ($r_e = -.35$).² In these analyses, we included both genetic and nonshared environmental correlations, but the genetic correlations were nonsignificant, both $\chi^2(1) < 3.64, p > .056$.

Associations Between Facets of Impulsivity and Other Constructs

Table 5 shows the phenotypic correlations between the facets of impulsivity and other constructs. Each row represents a single model where that construct is correlated with all five facets of impulsivity. Table 5 also displays the regression model where that construct is predicted by the five impulsivity subscales (in parentheses). Model fit of the correlational and regression models were identical. Factor loadings on latent factors are displayed in the supplement (Table S2), as are the phenotypic intercorrelations among these other constructs (Table S3).

The genetic and environmental associations are displayed in Table 6. Again, each row represents a unique analysis combining the genetic/environmental model of impulsivity from Figure 2 with that construct. In the first row, for example (depicted in Figure 3), variance in goal-management failures is decomposed into variance explained by genetic/environmental factors common to all facets of impulsivity (A_{Imp}, E_{Imp}), and unique genetic/environmental influences on goal-management failures (A_{Unique}, E_{Unique}). Additionally, genetic/environmental paths were included for impulsivity facets that were significant in the regression model (Table 5). In the case of the model with goal management, we included genetic/environmental paths from negative urgency (A_{NU}, E_{NU}), positive urgency (A_{PU}, E_{PU}), and lack of perseverance (A_{Persev}, E_{Persev}). In the final model displayed in Table 6, these additional paths were included only when statistically significant (for a version of this model that includes nonsignificant paths, see supplemental Table S4). Variance in goal-management failures explained by each factor can be computed by squaring these factor loadings from Table 6. Model fit and factor loadings on latent variables are displayed in the supplement (Table S2).

Goal management underlies commonality among impulsivity facets

(Hypothesis 1).—Our first hypothesis was that goal-management failures were central to the common variance among impulsivity facets. As shown in Table 5, goal-management failures demonstrated the strongest or second-strongest phenotypic correlation with each facet ($r_s > .33, p_s < .001$) except sensation seeking ($r = .06, p = .152$) compared to the other constructs examined here. Regression analyses suggested that, controlling for other facets, there were unique associations between (poor) goal management and negative urgency ($\beta = .34$), positive urgency ($\beta = .10$), and lack of perseverance ($\beta = .30$), but this multiple

²It was not surprising that it was necessary to include an additional path between the two urgency facets. Although it is somewhat surprising to include a negative environmental correlation between lack of perseverance and sensation seeking, their weak negative association has been well documented (Cyders & Smith, 2007; Whiteside & Lynam, 2001), and it makes sense that this negative association reflects an association unique to perseverance and sensation seeking given that all other facets are positively correlated with one another.

regression does not model the common and unique variance among impulsivity facets as explicitly as the independent path model.

In the genetic model (Table 6), the common genetic factor for impulsivity (A_{Imp}) was strongly predictive of goal-management failures, explaining 40.0% of its total variance. The nonshared environmental influences on impulsivity (E_{Imp}) explained an additional 5.8% of the variance in goal-management failures. Both estimates were larger than for any other construct examined here, suggesting that a considerable portion of the common genetic variance among impulsivity facets is associated with goal-management abilities. Additionally, there was a small path from the environmental influences unique to negative urgency to goal-management failures, explaining 4.4% of its total variance.

Understanding the heterogeneity of impulsivity (Hypotheses 2a, 2b, and 2c).

Sensation seeking versus other facets (Hypothesis 2a): Consistent with the hypothesis that sensation seeking is largely distinct from other facets, it was not significantly explained by the common genetic factor in impulsivity (A_{Imp} ; Figure 2). Although some variance in sensation seeking was explained by the shared environmental (C_{Imp} , 25.0%) and nonshared environmental (E_{Imp} , 4.4%) common factors, the shared environmental factor did not explain much variance in any other facet ($<7\%$, suggesting it primarily captured sensation seeking), and the nonshared environmental factor accounted for little variance.

The phenotypic results for sensation seeking also differed from those of other facets of impulsivity (Table 5). Sensation seeking was the only facet of impulsivity not associated with procrastination ($r = -.03$) or goal-management failures ($r = .06$) and was also the only facet-negatively correlated with neuroticism ($r = -.20$). Furthermore, all associations between sensation seeking and other constructs remained significant in regression analyses, suggesting their associations with sensation seeking are distinct from other facets. These distinct associations between sensation seeking and neuroticism, extraversion, and externalizing were nonsignificant in the genetic model (Table 6) but appeared to be explained by genetic influences (Table S4).

Emotional versus non-emotional impulsivity (Hypothesis 2b): We expected that positive and negative urgency would be more strongly related to internalizing psychopathology and neuroticism than lack of premeditation. Consistent with that hypothesis, negative and positive urgency were associated with greater neuroticism ($r = .56$ and $.29$, respectively), and with more internalizing psychopathology ($r = .32$ and $.17$, respectively), but lack of premeditation was not associated with either neuroticism ($r = .04$) or internalizing ($r = .05$). All associations were significantly larger for positive or negative urgency than for lack of premeditation, all $\chi^2(1) > 6.51$, $ps < .011$. Similarly, all three constructs were associated with greater procrastination and more externalizing psychopathology, but negative urgency was more strongly correlated with both than was lack of premeditation, both $\chi^2(1) > 14.32$, $ps < .001$.

In the regression model, there was some evidence for unique associations between lack of premeditation and externalizing psychopathology ($\beta = .14$), neuroticism ($\beta = -.12$), and extraversion ($\beta = .17$). Interestingly, these associations with neuroticism and extraversion

were in the opposite direction than those for negative urgency, and driven by genetic influences (see Table 6). Nonshared environmental influences on negative urgency (E_{NU} , which were correlated with those for positive urgency), demonstrated unique associations with all constructs except extraversion (explaining 1.7 to 13.0% of their variance), suggesting there are distinct environmental associations between urgency and other psychological constructs (E_{NU}) beyond the common environmental influences linking impulsivity facets (E_{Imp}).

Incorporating lack of perseverance (Hypothesis 2c): We expected that lack of perseverance would demonstrate similar associations as lack of premeditation or diverge in a similar manner as negative urgency. The results were consistent with the latter possibility. Phenotypically, lack of perseverance was associated with all the same constructs as negative urgency (greater procrastination, neuroticism, internalizing, and externalizing problems, and lower extraversion). Like the previous results for urgency, lack of perseverance was more positively correlated with all constructs than lack of premeditation (or more negatively for extraversion), all $\chi^2(1) > 3.90$, $ps < .048$, except for externalizing, $\chi^2(1) = 2.21$, $ps = .137$.

The key distinguishing feature of lack of perseverance was its association with procrastination ($r = .71$; $\beta = .63$). Much of this correlation was driven by genetic influences common to all facets of impulsivity (A_{Imp}), but there were also genetic and environmental influences unique to lack of perseverance that accounted for 7.3% and 14.4% of its variance, respectively. Finally, as noted above, the three-factor model from Cyders and Smith (2007), in which lack of perseverance and lack of premeditation loaded on a common factor, fit poorly, providing further evidence against this scenario.

Discussion

The goal of the study was to elucidate the commonality (Hypothesis 1) and heterogeneity (Hypotheses 2a-2c) of impulsivity facets and their genetic and environmental associations with important psychological constructs during late adolescence/early adulthood. Results were consistent with our primary hypothesis that goal-management abilities are central to the common variance underlying impulsivity facets, and supported three other hypotheses aimed to illuminate the factors contributing to the differences between impulsivity facets. Multivariate twin analyses revealed that common variance in impulsivity facets was associated with other constructs primarily through genetic influences, whereas the unique associations between impulsivity facets and other constructs were explained by a mix of genetic and environmental factors.

Implications for Goal Management and Impulsivity (Hypothesis 1)

These findings are consistent with the view that goal-management abilities are a large component of the commonality among facets of impulsivity. This ability to activate and maintain goals accounts for most of the genetic covariance between procrastination and impulsivity (Gustavson et al., 2014). The present findings demonstrate that lack of perseverance also plays a large role in this same genetic covariance, but that sensation seeking is genetically distinct. Although environmental influences common across impulsivity facets had a different structure than the genetic influences, these influences were

also associated with goal-management failures. In fact, the nonshared environmental influences common among impulsivity facets were only significantly associated with goal-management failures and extraversion. Thus, goal-management abilities appear to underlie much of the genetic influences uniting impulsivity facets, and at least some of its environmental influences.

Important to note, the items assessing goal-management abilities in this study did not draw on an individual's tendency to give into their temptations or act spontaneously. Instead, they draw on whether goals are not activated at the appropriate time, lost track of, or forgotten (e.g., *Do you drop things*, *Do you daydream when you ought to be listening to something*, *I forgot what I came into a room to get*, and *I forgot to tip when I finished dinner at a restaurant*). Nevertheless, genetic influences common across facets of impulsivity explained 40% of the total phenotypic variance in goal-management failures, suggesting genetic influences that support the activation and maintenance of goal representations are tightly linked with those that allow one to resist giving into their temptations (urgency), acting without thinking (premeditation), and giving up on difficult or boring tasks (perseverance).

To fully understand these genetic influences on impulsivity and their overlap with goal management (and cognition more broadly), it will be important for future work to tease apart which aspects of goal-management failures examined here account for these associations. The factor examined here likely relates to multiple executive function processes including objective measures of sustained attention and working memory, as well as other processes such as encoding and retrieval of long-term memory representations. Indeed, the small but significant associations between executive functions and the goal-management failures factor described here are explained primarily by genetic influences (Gustavson et al., 2015), suggesting that they may overlap (at least partially) with those on impulsivity revealed here. Moreover, these associations with goal management were explained by general variance common to multiple executive function tasks (response inhibition, set shifting, and working memory updating) rather than variance unique to one set of tasks (e.g., working memory; Gustavson et al., 2015), providing further evidence they capture general processes tapping the active maintenance and pursuit of goal representations (Friedman & Miyake, 2017).

Interestingly, there was an additional environmental association between negative urgency and goal-management failures. These findings suggest that some aspects of goal-management ability are not related to all impulsivity facets. Because urgency captures tendencies to control urges and resist temptations, this environmental association may capture a greater reliance on some aspects of executive function or attentional control in urgency. However, this association was small (explaining only 4.4% of the variance in goal-management failures, compared to 45.5% of the variance explained by genetic/environmental influences common among facets), suggesting the vast majority of the association between goal-management and impulsivity is due to common variance shared among all facets except sensation seeking.

Although we focused on goal-management failures, each of the psychological constructs investigated here was associated to some degree with genetic influences common to impulsivity. Particularly, it is well known that different facets of impulsivity are associated

with psychopathology symptoms (Berg et al., 2015) and these associations are driven by higher-order variance in internalizing and externalizing (Gustavson et al., 2017; Whiteside & Lynam, 2003). Here, genetic variance in impulsivity (A_{Imp}) explained 30.3% of the variance in externalizing and 27.0% of the variance in internalizing psychopathology. Because goal-management abilities accounted for a large portion of the common genetic influences on impulsivity, these findings are consistent with models of psychopathology that highlight the role of cognitive control, measured with either impulsivity or cognitive executive functions (Caspi et al., 2014; Gustavson et al., 2017; Young et al., 2009). However, it will also be important to understand whether psychopathology's associations with impulsivity and cognition represent the same underlying genetic influences, or if one or both has unique genetic/environmental associations.

Implications for the Heterogeneity of Impulsivity (Hypothesis 2)

Sensation seeking versus other facets.—The findings were consistent with recent proposals that suggest sensation seeking and impulsivity represent early independent processes, or dual systems (e.g., top-down control vs. bottom-up reward; Shulman, Harden, et al., 2016; Shulman, Smith, et al., 2016; Steinberg et al., 2008). There were some environmental associations between sensation seeking and other facets (Figure 2), but it was entirely genetically distinct from the other facets, and its associations with other constructs remained significant in the regression model controlling for other facets (Table 5). Thus, the inclusion of sensation seeking within the construct of impulsivity may be largely misleading (i.e., the “jingle fallacy”), and studies seeking to further elucidate the specific genes underlying impulsivity (e.g., genomewide association studies) should exclude sensation seeking as a candidate measure.

Emotional versus non-emotional impulsivity.—Consistent with Hypothesis 2b, lack of premeditation was not as strongly associated with psychopathology or neuroticism as positive or negative urgency. A growing body of work suggests that both positive and negative urgency are strongly predictive of internalizing psychopathology (Berg et al., 2015; Johnson et al., 2013), whereas positive urgency, negative urgency, and lack of premeditation are associated with externalizing problems (Grano et al., 2007; Whiteside & Lynam, 2003). Although more work is necessary to unpack the relations between personality variables and psychopathology (Durbin & Hicks, 2014), especially at the level of individual disorders, the associations between urgency and internalizing psychopathology demonstrated here may represent multiple factors. These may include heightened emotional reactivity, poor emotion regulation, and poor distress/stress tolerance. However, there is some work suggesting that it is not solely an artifact of current symptoms, and that it is not limited to negative emotional experiences (Carver & Johnson, 2018; Carver, Johnson, & Joormann, 2013). Lack of premeditation was associated with procrastination and externalizing psychopathology; these associations were also weaker than those for urgency (and lack of perseverance), likely due to the fact that both also involve an inability to cope with negative emotions (Eisenberg et al., 2009; Sirois & Kitner, 2015; Sirois & Pychyl, 2013).

Furthermore, genetic influences unique to lack of premeditation (A_{Prem}) were associated with less neuroticism and greater extraversion, associations that were observed in the

opposite direction as the genetic influences across impulsivity facets (A_{Imp}). This may explain why premeditation did not load on the neuroticism or externalizing factors in recent meta-analysis incorporating the UPPS and other self-reported measures (Sharma et al., 2014). Namely, there is substantial genetic covariance of urgency and lack of premeditation that links both with high neuroticism and low extraversion, but genetic influences unique to lack of premeditation may be protective against neuroticism and low extraversion, cancelling out the overall association. This finding highlights the strengths of multivariate genetic models.

Finally, one of the most consistent results was that the environmental influences unique to negative urgency (ENU) were positively associated with all other constructs (except extraversion). Like the genetic associations, these likely reflect the contribution of many independent environmental influences (possibly including those related to emotional reactivity, emotion regulation, and distress/stress tolerance noted above). This particular environmental association also indicates that there may be some causal or bi-directional associations between urgency and neuroticism or psychopathology beyond genetic liability shared with other facets. Other applications of the twin design (e.g., co-twin control) can be useful in examining such causal models or specific environmental factors. Although these associations appeared to be due to negative, rather than positive, urgency, it is important to recall that these influences were positively correlated with positive urgency. Thus, they likely represent unique environmental influences on impulsivity urgency as a whole, rather than only negative urgency.³

Incorporating lack of perseverance.—Lack of perseverance has not been studied as frequently as other impulsivity facets, and it was unclear if it would be more similar to lack of premeditation or urgency. Results were consistent with the latter possibility, suggesting that the emotional control needed to deal with frustration on difficult tasks is quite similar to that involved with controlling impulses when in a negative mood. Although our data did not support a model where lack of premeditation and perseverance load on a common factor (Cyders & Smith, 2007), these facets were captured by the nonshared environmental influences on impulsivity (E_{Imp}) more strongly than other facets. Thus, the two may be more *environmentally* similar than other facets. Finally, lack of perseverance also demonstrated unique genetic and environmental associations with procrastination. This finding suggests that the tendency to give up on difficult tasks is similar to the tendency to avoid the unpleasant tasks altogether, even after considering the well-known association between other facets of impulsivity and procrastination (Ferrari, 1993; Gustavson et al., 2014; Steel, 2007), and that this stronger similarity with procrastination may be the key distinguishing feature between perseverance and urgency.

³As shown in Figure 3, the residual association between negative and positive urgency was not estimated directly in the models with other constructs (displayed in Table 6), but rather included as a Cholesky path (common in structural twin models). Negative urgency was included first in the Cholesky ordering, but if positive urgency had been included first, these environmental associations would have likely been driven by that factor (E_{PU}).

Methodological Implications

Multivariate genetic analytic models allowed us to examine the commonality and heterogeneity of impulsivity in multiple novel ways. The independent path model demonstrated that there are genetic and environmental influences accounting for most of the covariance between impulsivity facets. The existence of common genetic and environmental factors is important for ongoing efforts to identify specific genetic influences on impulsivity (using genome wide association studies); namely, that it will be important to consider whether genes identified as related to impulsivity are common or unique to specific facets, affecting the likelihood of their relevance to personality, psychopathology, and cognition. Genome wide associations must be conducted using extremely large sample sizes (Visscher et al., 2017), yet it will be useful to confirm that genetic correlations based on measured genes (single nucleotide polymorphisms) show similar associations with the constructs we examined here.

Second, the findings that there are some unique genetic and environmental associations between impulsivity facets and other psychological constructs suggests that it will be important to be careful about which impulsivity measures are aggregated in a given analysis. Most of the associations between impulsivity and other psychological constructs could be explained by the common genetic factor (A_{Imp}), but for every construct there were unique associations with one or more impulsivity facets. Because some of these associations with unique genetic/environmental components were observed in the opposite direction as the common genetic factor, aggregating measures may obscure the true associations between impulsivity and other constructs.

Limitations and Future Directions

Several limitations should be noted. First, we examined these associations in early adulthood, but these associations with impulsivity may differ depending on the stage of life. Genetic influences on impulsivity may be more strongly predictive of some of the constructs examined here in younger adolescents or older adults when control abilities are still developing or beginning to exhibit age-related decline. Moreover, genetic influences underlying personality, including impulsivity, are only moderately stable until at least young adulthood (Briley & Tucker-Drob, 2014; Niv et al., 2012), and neuroticism and extraversion were not measured concurrently with impulsivity here. The correlations between impulsivity, neuroticism, and extraversion would likely have been larger if examined at the same time. Furthermore, reliability estimates for these other personality variables were low, especially at age 17 (.58 to .66), so the results should be interpreted with caution. Nevertheless, the fact that we observed moderate correlations between both constructs and impulsivity indicates some stability of these relationships, at least between late adolescence and young adulthood.

Second, although we identified sets of common and unique genetic influences on impulsivity facets, we did not have the power to distinguish between additive and dominant genetic influences. Meta-analytic work has suggested that the genetic influences identified here reflect a combination of both additive and dominant genetic influences (Bezdjian et al., 2011), thus these results should be interpreted in the context of broad-sense heritability.

Similarly, although the residual genetic influences on all impulsivity facets were significant, we may have lacked power to detect genetic correlations between these residual influences and the other psychological constructs. Nevertheless, some associations were significant in Table 5, and results of the more conservative version of this model (Table S4) suggested that the environmental associations were not upwardly biased by removing the nonsignificant genetic associations.

Third, we did not adjust the associations between impulsivity facets and other constructs (Table 5) for multiple comparisons because there is extensive literature linking impulsivity facets with each of the constructs examined here, including meta-analyses and reviews (Berg et al., 2015; Coskunpinar et al., 2013; Dick et al., 2010; Grano et al., 2007; Steel, 2007; Swann et al., 2002). However, the decomposition of these associations into common and unique sets of genetic and environmental influences should be replicated in a larger sample, particularly those weaker associations between unique genetic/environmental influences on impulsivity facets that we may not have power to detect (Table 5 vs. Table S4). We also report exact *p* values for the phenotypic (Table 5) and genetic analyses (Table 6) in the supplement (Tables S5 and S6), which can be used to examine which results remain significant after adjusting for multiple comparisons.

Fourth, the associations between impulsivity and psychopathology may differ in clinical samples with higher proportions of individuals diagnosed for the disorder, or in samples with more measures of internalizing (e.g., social anxiety disorder or panic disorder). Finally, although we focused here on self-reported measures, it will be important to examine how performance-based measures are associated with the same covariance. There is low correspondence between self-reported measures and behavioral measures of self-control (Duckworth & Kern, 2011; Sharma et al., 2014; Toplak et al., 2013), so it may be necessary to examine these associations using latent variables, and at the level of genetic correlations, to see substantial associations.

Summary and Conclusions

Our results revealed a substantial genetic factor linking variance among impulsivity facets (except sensation seeking) that was strongly associated with individuals' abilities to activate and maintain goal representation in everyday life. However, there were also considerable differences among the facets of impulsivity, with unique genetic and environmental factors on most all factors predicting unique variance in at least one construct examined here. In some cases, the correlations between impulsivity facets and other constructs were stronger than the correlations among facets themselves (especially for negative urgency). These findings support the idea that there is considerable independent variance in impulsivity facets in addition to that common variation that appears to be related to goal management abilities. Future analyses will benefit from similar techniques that highlight the common and specific sources of variance between impulsivity and other important constructs, especially methods that hone in on the genetic risk factors that account for most, but not all, of these associations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

Genetic influences uniting impulsivity facets strongly related to goal management

Unique genetic and environmental influences on all facets lead to separability

Sensation seeking was entirely genetically distinct from other impulsivity facets

Urgency was more strongly related to psychopathology than lack of premeditation

Lack of perseverance was more similar to urgency than lack of premeditation

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Figure 1:

Genetic and environmental models of the other constructs of the study. In some models (A and C) it was necessary to equate factor loadings to identify the model (these loadings were freed in later models involving impulsivity). Internalizing and externalizing psychopathology were included in the same model to aid in model identification. In all models, we accounted for sex differences by regressing all measures on sex. Shared environmental influences excluded from models A through C based on previous work (Gustavson et al., 2015; 2017). Significant paths are displayed with black text and solid black lines ($p < .05$). A = genetic influences, C = shared environmental influences, E = nonshared environmental influences. Model fit: (A) $\chi^2(19) = 29.04$, $p = .065$, RMSEA = .052, CFI = .981; (B) $\chi^2(48) = 52.04$, $p = .320$, RMSEA = .021, CFI = .995; (C) $\chi^2(23) = 28.03$, $p = .215$, RMSEA = .033, CFI = .976; (D) $\chi^2(9) = 15.81$, $p = .071$, RMSEA = .064, CFI = .757; (E/F) $\chi^2(193) = 198.75$, $p = .373$, RMSEA = .012, CFI = .997

Figure 2:

Independent pathway model of the genetic (A), shared environmental (C), and nonshared environmental (E) covariance between facets of impulsivity. Percent variance explained by each path can be explained by squaring the factor loading. Significant factor loadings and correlations between residual genetic/environmental influences are displayed with black text and solid lines ($p < .05$).

Figure 3:

Example of the genetic analysis with other constructs displayed in Table 6. In this example, a latent factor representing goal-management failures is decomposed into variance shared with genetic/environmental influences common among impulsivity facets (A_{Imp} , E_{Imp}), genetic/environmental influences unique to some impulsivity facets (e.g., A_{NU} , E_{NU}), and unique genetic/environmental influences on goal management (A_{Unique} , E_{Unique}). The thick arrows representing these paths correspond to the values displayed in Table 6. Shared environmental influences on impulsivity (C_{imp} in Figure 2) were estimated but not shown here for simplicity.

Table 1

Classifications of Impulsivity in Theories of Personality

Source:	Facets of Impulsivity	Brief Description	Instrument:
Whiteside & Lynam, 2001; Lynam et al., 2006	Impulsive urgency Lack of premeditation Lack of perseverance Sensation seeking	Giving in to temptations or desires Acting without thinking or forethought Giving up on boring or difficult tasks Enjoying and searching for exciting experiences	UPPS, UPPS-P
Buss and Plomin (1975)	Inhibitory control Lack of persistence Sensation seeking/boredom Decision time	Ability to resist giving into temptations The tendency to give up quickly Enjoying and searching for exciting experiences Acting on the spur of the moment	Emotionality, Activity, Sociability, and Impulsivity Temperament Scales
Eysenck (1993)	Impulsiveness Venturesomeness	Unconscious risk taking (component of psychoticism) Sensation seeking (component of extraversion)	Eysenck Personality Questionnaire (EPQ)
Tellegen (1982)	Control (vs. impulsiveness)	Acting without thinking or forethought (component of constraint)	Multidimensional Personality Questionnaire (MPQ)
Schalling, Asberg, Edman, and Orelund (1987)	Impulsiveness	Acting without thinking or forethought	Karolinska Scales of Personality
Costa and McCrae (1992)	Impulsiveness Self-discipline Excitement seeking Deliberation	Inability to resist temptations (component of neuroticism) Inability to force oneself to do something desired (component of conscientiousness) Similar to sensation seeking (component of extraversion) Acting without thinking or forethought	Revised NEO Personality Inventory
Cloninger et al. (1993)	Impulsiveness (low) Persistence	Tendency to give in to temptations, thrill seeking (component of novelty seeking) Tendency to give up quickly when frustrated	Temperament and Character Inventory (TCI)
Dickman (1990)	Dysfunctional impulsivity Functional impulsivity	Acting without thinking or forethought Acting with little forethought when it is optimal to do so	Functional and Dysfunctional Impulsivity Scales
Barratt (1993), Patton, Stanford, and Barratt (1995)	Motor impulsivity Cognitive impulsivity Non-planning	Acting without thinking Making quick decisions, racing thoughts Lack of orientation towards the future	Barratt Impulsiveness Scale (BIS-10, BIS-11)
Zuckerman (1994)	Sensation seeking	Enjoying and searching for exciting experiences	Sensation Seeking Scale (SSS)
Lecrubier et al. (1995)	Impulsivity	Control over responses, irritability, impatience, capacity for delay	Impulsivity Rating Scale (IRS)
Tangney et al. (2004)	Self-control	The ability to resist temptations, control thoughts and emotions, and regulate performance and habits	Self-Control Scale (SCS)
American Psychiatric Association (2013)	Impulse control disorders	Disorders characterized by failure to resist temptations or urges (e.g., behavioral addictions, attention deficit hyperactivity disorder, conduct disorder)	Diagnostic and Statistical Manual of Mental Disorders (DSM-V)

Note: This table was revised and updated based on one presented by Evenden (1999).

Table 2

Example Items from the UPPS-P Impulsive Behavior Subscales

Subscale	Example Items
Negative Urgency	When I feel rejected, I will often say things that I later regret When I am upset I often act without thinking In the heat of an argument, I will often say things that I later regret
Positive Urgency	When I am really ecstatic, I tend to get out of control Others are shocked or worried about the things I do when I am feeling very excited When I am very happy, I feel like it is okay to give in to cravings or overindulge
Lack of Premeditation	My thinking is usually careful and purposeful Before making up my mind, I consider all the advantages and disadvantages ^R I usually think carefully before doing anything ^R
Lack of Perseverance	I tend to give up easily I am a person who always gets the job done ^R I finish what I start ^R
Sensation Seeking	I welcome new and exciting experiences, even if they are a little frightening and unconventional I'll try anything once I would enjoy parachute jumping

Note:

^R indicates reverse-scored items.

Table 3

Descriptive Statistics

Measure	<i>N</i>	<i>M</i>	<i>SD</i>	Range	Skewness	Kurtosis	Reliability
Impulsivity							
Negative Urgency	753	2.13	0.59	1.00, 4.00	0.43	0.01	0.82
Positive Urgency	753	1.88	0.54	1.00, 4.00	0.56	0.35	0.78
Lack of Premeditation	754	1.88	0.48	1.00, 3.88	0.36	0.12	0.80
Lack of Perseverance	754	1.72	0.46	1.00, 3.50	0.59	0.22	0.69
Sensation Seeking	754	2.87	0.66	1.14, 4.00	−0.43	−0.55	0.81
Goal Management							
<i>Goal-Management Failures</i> Cognitive Failures Questionnaire	736	0.63	0.29	0.00, 1.70	0.47	0.08	0.90
Prospective Memory Questionnaire	750	1.39	0.45	0.12, 3.20	0.30	0.54	0.91
Personality							
<i>Procrastination</i>							
General Procrastination Scale	751	2.59	0.57	1.05, 4.60	0.04	−0.03	0.89
Volitional Components Inventory	751	3.03	0.85	1.05, 6.19	0.36	0.04	0.92
Action Control Scale	747	7.90	2.90	0, 12	−0.47	−0.59	0.76
<i>Neuroticism</i>							
Age 17	714	0.39	0.23	0, 1	0.32	−0.65	0.66
Age 21	728	0.31	0.26	0, 1	0.63	−0.50	0.83
<i>Extraversion</i>							
Age 17	714	0.71	0.21	0, 1	−0.68	−0.02	0.58
Psychopathology							
<i>Internalizing Psychopathology</i>							
Generalized Anxiety Disorder	763	0.16	0.48	0, 2	-	-	-
Major Depressive Disorder	763	0.38	0.70	0, 2	-	-	-
<i>Externalizing Psychopathology</i>							
Antisocial Personality Disorder	763	0.65	0.78	0, 2	-	-	-
Tobacco Use Disorder	762	0.52	0.79	0, 2	-	-	-
Alcohol Use Disorder	762	1.21	1.29	0, 4	-	-	-
Cannabis Use Disorder	762	0.43	0.92	0, 4	-	-	-
Illicit Substance Use Disorder	762	0.09	0.29	0, 1	-	-	-

Note: Reliability was computed using Chronbach's alpha. Scores on questionnaires reflect the average response across all items. Psychopathology variables were based on binned symptom counts from diagnostic interviews.

Table 4

Phenotypic Correlations Among Impulsivity Subscales

Impulsivity Subscale	Negative Urgency	Positive Urgency	Lack of Premeditation	Lack of Perseverance	Sensation Seeking
Negative Urgency	1				
Positive Urgency	0.61	1			
Lack of Premeditation	0.32	0.34	1		
Lack of Perseverance	0.32	0.31	0.39	1	
Sensation Seeking	0.08	0.19	0.18	-0.10	1

Note: Correlations are displayed after adjusting each measure for its association with sex. All correlations were significant ($p < .05$).

Table 5

Phenotypic Correlations (and Regression Coefficients) Between Impulsivity Subscales and Measures of Goal Management, Personality, and Psychopathology

Dependent Variable	Negative Urgency	Positive Urgency	Lack of Premeditation	Lack of Perseverance	Sensation Seeking	Model Fit			
						χ ²	df	RMSEA	CFI
Goal Management									
Goal-Management Failures	0.52 (0.34)	0.43 (0.10)	0.33 (0.06)	0.46 (0.30)	0.06 (0.04)	10.89	4	0.048	0.994
Personality									
Procrastination	0.46 (0.26)	0.36 (0.01)	0.32 (−0.01)	0.71 (0.63)	−0.03 (0.02)	39.80	10	0.063	0.983
Neuroticism	0.56 (0.60)	0.29 (0.00)	0.04 (−0.12)	0.20 (0.03)	−0.20 (−0.23)	14.11	4	0.058	0.989
Extraversion	−0.10 (−0.10)	−0.05 (−0.04)	0.10 (0.17)	−0.12 (−0.13)	0.16 (0.14)	0.00	0	0.000	1.000
Psychopathology Symptoms									
Internalizing	0.32 (0.31)	0.17 (0.00)	0.05 (−0.11)	0.17 (0.12)	0.03 (0.04)	13.45	4	0.056	0.986
Externalizing	0.45 (0.36)	0.33 (−0.01)	0.33 (0.14)	0.27 (0.13)	0.21 (0.18)	36.39	25	0.024	0.994

Note: For each dependent variable, correlations with subscales of impulsivity are displayed in the first row, and regression coefficients are displayed on bottom row (in parentheses). Model fit for the correlational and regression models were identical. In all models, the association with sex was regressed out of all measures. Factor loadings on latent factors are displayed in the supplement (Table S2), along with *p*-values for all correlations and regression coefficients displayed here (Table S5). Significant values are displayed in bold ($P < .05$).

Table 6

Genetic and Environmental Influences on Goal Management, Personality, and Psychopathology and Their Overlap with Impulsivity Facets

	a _{Imp}	c _{Imp}	e _{Imp}	a _{NU}	e _{NU}	a _{PU}	e _{PU}	a _{Prem}	e _{Prem}	a _{Pers}	e _{Pers}	a _{SS}	e _{SS}	a _{Unique}	e _{Unique}
<i>Goal Management</i>															
Goal-Management Failures	0.63		0.24		0.21							0.41			0.58
<i>Personality</i>															
Procrastination	0.60		0.16		0.13					0.27	0.38			0.14	0.60
Neuroticism	0.61		0.07		0.35			-0.33						0.00	0.63
Extraversion	-0.37		0.17					0.27						0.21	0.80
<i>Psychopathology Symptoms</i>															
Internalizing	0.52	0.18	-0.23		0.22							0.17	0.72		0.25
Externalizing	0.55	0.15	0.14		0.14							0.67		0.00	0.45

Note: In each model (row), variance in the dependent variable was decomposed into genetic/environmental influences common to all facets of impulsivity (a_{Imp}, c_{Imp}, e_{Imp} from Figure 2), genetic/environmental influences unique to one facet of impulsivity (e.g., a_{NU}, e_{NU} for negative urgency from Figure 2), and genetic/environmental influences unique to that trait (a_{Unique}, c_{Unique}, e_{Unique}). Variance explained by each genetic/environmental factor can be computed by squaring these factor loadings, and significant factor loadings are displayed in bold ($p < .05$). Paths for genetic/environmental influences unique to each facet of impulsivity were tested only when there was evidence for unique associations in the regression analyses, and included in the final model only if they were significant. In all models, the association with sex was regressed out of all measures. Model fit and factor loadings on latent factors are displayed in the supplement (Table S2), along with p -values for all coefficients displayed here (Table S5). NU = Negative Urgency, PU = Positive Urgency, Premed = Lack of Premeditation, Persév = Lack of Perseverance, SS = Sensation Seeking.