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Genetic and Environmental Associations Between Procrastination and Internalizing/Externalizing Psychopathology

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Abstract

Recent work on procrastination has begun to unravel the genetic and environmental correlates of this problematic behavior. However, little is known about how strongly procrastination is associated with internalizing and externalizing psychopathology, and the extent to which shared genetic/environmental factors or relevant personality constructs (e.g., fear of failure, impulsivity, and neuroticism) can inform the structure of these associations. The current study examined data from 764 young adult twins who completed questionnaires assessing procrastination and personality and structured interviews regarding psychopathology symptoms. Results indicated that procrastination was positively correlated with both internalizing and externalizing latent variables, and that these correlations were driven by shared genetic influences. Moreover, the association between procrastination and internalizing was accounted for by fear of failure and neuroticism, whereas the association between procrastination and externalizing was primarily explained by impulsivity. The role of procrastination in psychopathology is discussed using a framework that highlights common and broadband-specific variance.

Keywords

procrastination; internalizing; externalizing; impulsivity; neuroticism

Many individuals struggle with procrastination in their day-to-day lives. Recently, procrastination has been linked to a number of negative health outcomes, including poor sleep quality (Hairston & Shpitalni, 2016; Sirois, van Eerde, & Argiopoulou, 2015), mood dysregulation (Sirois & Kitner, 2015; Sirois & Pychyl, 2013), and symptoms of depression and anxiety (Beutel et al., 2016). Moreover, there are well-studied links between procrastination and personality variables such as impulsivity and neuroticism (Ferrari, 1993; Schouwenburg & Lay 1995; Steel, 2007; Van Eerde, 2000) that have also been identified as substantial predictors of psychopathology (Hink et al., 2013; Johnson, Caver, & Joormann,

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Author Contributions

Daniel Gustavson developed the study design, conducted the data analysis, and drafted the manuscript under the supervision of Naomi Friedman. All authors contributed to the study design, provided critical revisions, and approved the final version of the paper for submission.

2013). Although this body of work suggests that procrastination may play an important role in mental health (Tice, Bratslavsky, & Baumeister, 2001; Wohl, Pychyl, & Bennett, 2010), little is known about how procrastination behaviors are associated with internalizing and externalizing psychopathology. Moreover, it is unclear whether procrastination accounts for some of the general risk shared across internalizing and externalizing psychopathology (i.e., common variance across both domains of psychopathology) or variance unique to specific psychopathologies (i.e., broadband-specific variance in internalizing or externalizing psychopathology).

In the current study, we examine the association between procrastination and general liabilities to internalizing and externalizing psychopathology using a genetically informative twin study. We test three hypotheses regarding the association between procrastination and internalizing and externalizing psychopathology to investigate the phenotypic and genetic/environmental architecture of these associations. First, we test the hypothesis that procrastination is related to greater internalizing and externalizing psychopathology at the latent construct level. Second, we test the hypothesis that these relations between procrastination and psychopathology operate through different personality correlates (fear of failure, impulsivity, and neuroticism): specifically, that procrastination is associated with internalizing through fear of failure, with externalizing through impulsivity, and with both internalizing and externalizing through neuroticism. Finally, we test the hypothesis that these relations are primarily due to common genetic influences. To our knowledge, this is the first study to examine the association between procrastination behaviors, personality constructs, and psychopathology symptoms at the latent construct level and in a genetically informative sample.

Existing Theoretical Perspectives on Procrastination

Procrastination has been defined as a tendency to intentionally delay action on short- and long-term goals, despite expecting to be worse off for that delay (Gustavson, Miyake, Hewitt, & Friedman, 2014, 2015; Schouwenburg & Lay 1995; Steel, 2007). Although procrastination tendencies can be unique to certain situations or domains (e.g., academic procrastination), individual differences in procrastination are generally stable, trait-like, and associated with a number of negative psychological and financial consequences (Jaffe, 2013; Pychyl & Flett, 2012; Schouwenburg & Lay 1995).

Existing theoretical models of procrastination describe how procrastination tendencies are explained by individual differences in motivation, self-regulation, goal management, and personality (Gustavson et al., 2014, 2015; Krause & Freund, 2014; Schouwenburg & Lay 1995; Steel & König, 2006). Furthermore, individual differences in procrastination are explained roughly equally by genetic influences and nonshared environmental influences,¹ but genetic influences primarily account for its association with personality, goal

¹Throughout the text, we refer to shared and nonshared environmental influences. Shared environmental influences (typically, *C* in twin models) are influences that make twins similar to one another (e.g., family socio-economic status) whereas nonshared environmental influences (*E* in twin models) are environmental influences that make twins uncorrelated (e.g., unique peer groups). Both shared and nonshared environmental influences can correlate across traits (e.g., the nonshared environmental influences for procrastination are correlated with the nonshared environmental influences for impulsivity; Gustavson et al., 2014)

management, and cognitive control (Gustavson et al., 2014, 2015). Finally, the recent procrastination-health model has focused on the role of procrastination and stress in physical health outcomes including sleep quality, hypertension, and immune functioning (Sirios, 2015; Sirois & Kitner, 2015; Sirois et al., 2015; Tice & Baumeister, 1997).

Despite these advancements in understanding the motivational, cognitive, and physical health correlates of procrastination, research has yet to extensively apply these models of procrastination to psychopathology, especially at the level of internalizing or externalizing factors that explain covariation across multiple disorders (Lilienfeld, 2003; Weiss, Süsser, & Catron, 1998). This lack of research on procrastination and psychopathology is somewhat surprising, because perhaps the largest subset of work on procrastination has focused on its correlations with personality traits (for meta-analysis and reviews, see Steel, 2007; Klingsieck, 2013) that have been highlighted as important predictors of psychopathology (Hink et al., 2013; Johnson et al., 2013). For example, procrastination is associated with higher levels of fear of failure, impulsivity, and neuroticism, among other traits (Steel, 2007), all of which are relevant to psychopathology. In the following sections, we summarize the existing research that directly examines procrastination and psychopathology, and describe how personality variables may be relevant to these links given their associations with both procrastination and psychopathology.

Procrastination and Internalizing Psychopathology

Common psychological disorders often cluster into internalizing and externalizing factors. The internalizing domain typically encompasses disorders related to anxiety and depression (e.g., generalized anxiety disorder, major depressive disorder, separation anxiety disorder, Krueger, McGue, & Iacono, 2001; Weiss et al., 1998). Although specific internalizing disorders may have their own unique causes, symptoms, and risk factors, they are substantially correlated and often co-occur (Kendler & Myers, 2014; Krueger et al., 2002; Weiss et al., 1998). Focusing on the variation shared across these multiple specific internalizing disorders has been fruitful, both in terms of understanding which personality traits are associated with general internalizing behaviors (e.g., negative emotionality and neuroticism, Hink et al., 2013; Krueger et al., 2002; Tackett et al., 2013), and identifying shared genetic and environmental risk factors across internalizing disorders (Tackett et al., 2013).

Although few, if any, studies have directly examined how individual differences in procrastination are associated with broad internalizing behaviors, many have examined associations between procrastination, depression, and anxiety. For example, one study examining over 2,500 participants from a community sample (age 14 – 95 years) revealed that everyday procrastination (measured with the General Procrastination Scale) was positively correlated with the depression ($r = .36$) and anxiety ($r = .32$) subscales of the Patient Health Questionnaire (Beutel et al., 2016). These estimates are consistent with recent meta-analytic estimates of the association between procrastination and depression symptoms ($r = .28$, $k = 56$, Steel, 2007) and fear of failure, which included scales such as evaluation anxiety ($r = .18$, $k = 57$). Based on these findings that procrastination is associated with both

anxiety and depression, it is likely that procrastination is associated with internalizing psychopathology more generally.

If procrastination is related to internalizing psychopathology, this association may be in part due to fear of failure – the belief that aversive consequences will follow failures (Conroy, Willow, & Metzler, 2002). For example, individuals who are highly concerned over making mistakes may be especially likely to exhibit symptoms of anxiety when anticipating difficult exams. This anxiety may result in the delay of action to avoid confronting and experiencing that failure. Delay of action may, in turn, lead to a further increase in anxiety (e.g., due to having little time left to study or prepare for an exam). Although fear of failure is most often studied in the context of anxiety (i.e., evaluation anxiety), it may be related to depression as well, given that it is associated with devaluing of the self and shame or embarrassment (Conroy et al., 2002). Therefore, the association between procrastination and general internalizing psychopathology may be explained, in part, by individual differences in the tendency to fear failure.

Procrastination and Externalizing Psychopathology

Externalizing psychopathology also encompasses multiple psychiatric disorders, including substance use disorders, antisocial personality disorder, conduct disorder, and, in many models, attention-deficit/hyperactivity disorder (Caspi et al., 2014; Kreuger et al., 2001; Weiss et al., 1998). Externalizing psychopathology is positively associated with neuroticism, novelty seeking, impulsivity, and lack of constraint (Casillas & Clark, 2002; Hink et al., 2013; Krueger, et al., 2001). Externalizing is also negatively associated with cognitive abilities such as executive functions and intelligence (Caspi et al., 2014; Young et al., 2009). Furthermore, twin studies have identified genetic and environmental factors that influence general externalizing behaviors (Kendler & Myers, 2014; Kendler, Prescott, Myers, & Neale, 2003).

The association between procrastination and externalizing behaviors is less well established than the association between procrastination and internalizing. Nevertheless, there is some evidence that procrastination tendencies are positively associated with externalizing psychopathology, such as greater self-reported inattention symptoms of attention deficit hyperactivity disorder (Niermann & Scheres, 2014). Further, among adult marijuana users in a relapse-prevention support group, 94% reported procrastination as a problem related to their marijuana use (Stephens, Roffman, & Curtin, 2000). Although the existing studies on the direct associations between procrastination and externalizing behaviors are somewhat limited, this evidence can be leveraged with other known personality variables associated with both procrastination and externalizing.

Indeed, one of the most well studied correlates of procrastination is impulsivity (Ferrari, 1993; Gustavson, et al., 2014; Steel, 2007), which is strongly associated with externalizing behavior (Berg, Latzman, Bliwise & Lilienfeld, 2015; Casillas & Clark, 2002; Johnson, et al., 2013; Whiteside & Lynam, 2009). Therefore, we expect procrastination to be associated to externalizing through impulsivity. Procrastinators may be likely to engage in externalizing behaviors (e.g., using substances, engaging in antisocial behaviors) because they are more

likely to give into their temptations and/or act spontaneously and without thinking about the consequences of their actions, rather than restraining themselves and pursuing more productive long-term tasks and goals. This possibility is consistent with our work suggesting that procrastination and impulsivity may be genetically identical ($r_g = 1.0$; Gustavson et al., 2014), suggesting that any shared genetic risk factors underlying the association between impulsivity and psychopathology will also account for individual differences in procrastination.

Common Variance

A popular framework has emphasized a transdiagnostic approach that identifies sources of common and specific variance in psychopathology (Lilienfeld, 2003; Weiss, et al., 1998). Variation in psychopathology can be decomposed into three sources: (a) *narrowband-specific* features that are unique to a given disorder (e.g. physiological arousal in anxiety disorders), (b) *broadband-specific* features that distinguish internalizing from externalizing (e.g., novelty seeking in externalizing but not internalizing), and (c) common features that are shared across internalizing and externalizing psychopathologies (but differ from normality). This framework of internalizing and externalizing has been useful in examining the correlates of broad liability for psychopathology (Caspi et al., 2014; Tackett et al., 2013), as well as personality traits that are correlated with only internalizing or externalizing (Hink et al., 2013), while distinguishing these associations from those that are disorder-specific.

We have described two cases in which procrastination may be associated with broadband-specific variation in internalizing (i.e., through fear of failure) and externalizing (i.e., through impulsivity), but procrastination may also be a common feature through its association with neuroticism ($r = .24$, $k = 59$, Steel, 2007). Neuroticism is associated with both internalizing and externalizing, and importantly, their overlap (Hink et al., 2013; Kotov, Gamez, Schmidt, & Watson, 2010; Ormel, Rosmalen, & Farmer, 2004). For example, Hink et al. (2013) showed that genetic and nonshared environmental influences on neuroticism influenced the covariation between internalizing and externalizing disorders (as well as the covariation among internalizing disorders, and among externalizing disorders). Furthermore, after accounting for neuroticism and negative emotionality, there were no significant genetic/environmental influences shared between internalizing and externalizing disorders. Therefore, procrastination may also relate to the common variance between internalizing and externalizing (due to its association with neuroticism), and these associations may be present in addition to those broadband-specific associations described through separate mechanisms earlier (i.e., fear of failure and/or impulsivity).

Finally, it is worth noting that recent work has also linked impulsivity to internalizing (Granö et al., 2007; Johnson et al., 2013), suggesting that impulsivity may also account for common variance. However, given that the associations between impulsivity and internalizing may be considerably weaker than the associations between impulsivity and externalizing (Berg, et al., 2015), we expect that impulsivity primarily explains the association between procrastination and externalizing.

Genetic and Environmental Etiology

Decomposing the genetic and environmental etiology of the associations between procrastination and internalizing or externalizing may also provide more information about the transdiagnostic predictive value of procrastination and the proposed roles of personality traits in these associations. For example, if these associations are driven by genetic influences, this would suggest that levels of procrastination share a common genetic liability with the development of internalizing and/or externalizing symptoms. In contrast, if these associations are environmental in origin, this may grant some insight into the environmental and situational factors that lead to both procrastination and psychopathology. Finally, it is possible that procrastination may be associated with one construct primarily through genetic influences and the other through environmental influences, which would also be informative to these constructs' development, progression, and differentiation.

Our previous work has revealed that individual differences in procrastination are explained relatively equally by genetic influences ($h^2 = .46$) and nonshared environmental influences ($e^2 = .54$; Gustavson et al., 2014). Internalizing and externalizing are also heritable (Kendler & Myers, 2014; Kendler et al., 2003; Kreuger et al., 2002), and their correlations with personality constructs are often observed through shared genetic influences (Hink et al., 2013; Tackett et al., 2013). Procrastination, internalizing, and externalizing are also explained by nonshared environmental influences (Gustavson et al., 2014; Hink et al., 2013; Kendler & Myers, 2014), with limited evidence for shared environmental influences (Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler et al., 2003), especially for procrastination (Gustavson et al., 2014). As discussed earlier, the associations between procrastination and other traits such as impulsivity and goal management are primarily due to shared genetic factors rather than environmental influences, though some nonshared environmental correlations were observed (Gustavson et al., 2014).

Based on these earlier results, we expect that the association between procrastination and internalizing and externalizing psychopathology will be due to genetic influences. Although we expect genetic overlap between procrastination and internalizing and externalizing psychopathology, we acknowledge that procrastination may also be associated with internalizing and externalizing psychopathology through correlated nonshared environmental influences. However, our previous work has suggested that procrastination does not have any shared environmental influences, at least in the sample tested here (Gustavson et al., 2014). Therefore, we do not test for the presence of shared environmental correlations between procrastination and internalizing/externalizing in the context of the current study (though we still estimate shared environmental influences on internalizing and externalizing behaviors).

The Current Study

The current study investigates whether procrastination is associated with symptoms of internalizing and externalizing psychopathology, using data from a longitudinal twin study of same-sex twins from the normal population. Because we have multiple measures of each construct, we use structural equation modeling to estimate the phenotypic and genetic/

environmental correlations between these constructs at the latent variable level. Although endorsement of symptoms for any given psychopathology are low in this population sample (see Rhea, Gross, Haberstick, & Corley, 2013), we are able to examine internalizing and externalizing at the level of latent variables because twins were asked about psychopathology symptoms across many domains.

The three hypotheses tested in this study concern whether procrastination is associated with both internalizing and externalizing psychopathology, and if so, inform the extent to which these associations are associated with broadband-specific features of internalizing and externalizing psychopathology versus common variance across both tendencies. First, we expect that procrastination will be positively correlated with both internalizing and externalizing factors, such that individuals who procrastinate will endorse more internalizing symptoms and more externalizing symptoms. Second, we expect that three personality variables (fear of failure, impulsivity, and neuroticism) will account for the associations between procrastination and internalizing and externalizing psychopathology in different ways. Specifically, we predict that fear of failure will account for some of the shared variance between procrastination and internalizing, but not the association between procrastination and externalizing. Conversely, we expect that impulsivity will explain some of the association between procrastination and externalizing, but not internalizing. Finally, we expect that neuroticism will at least partially account for the associations between procrastination and both internalizing and externalizing.

Third, we examine the genetic and environmental associations between procrastination and internalizing and externalizing psychopathology. Specifically, we expect that both associations will be driven by shared genetic influences, but do not discount the possibility that environmental factors will also account for some of their covariation. To the extent that procrastination is genetically and/or environmentally associated with both constructs, we will conduct follow-up analyses to examine whether the personality variables that account for the association between procrastination and psychopathology do so through genetic and/or environmental influences.

Method

Subjects

Analyses were conducted on data from 764 individuals (402 females, 362 males) from 392 same-sex twin pairs (210 monozygotic [MZ] and 182 dizygotic [DZ]) from the ongoing Colorado Longitudinal Twin Study. Twins were born in Colorado between 1984 and 1990, were recruited through the Colorado Department of Health, and are representative of the general population of Colorado. Most of the sample identified themselves as White (92.6%), with additional individuals identifying as “more than one race” (5.0%), American Indian/Alaskan Native (< 1%), Pacific Islander (< 1%), or did not report their race (1.2%). Hispanic individuals comprised 9.1% of the sample. For more information on this sample, see Rhea et al. (2013).

The total sample included all individuals who completed questionnaire or interview measures at approximately age 23 ($M = 22.81$, $SD = 1.28$), except for neuroticism, which

was completed at age 12 ($M=12.42$, $SD=.38$) and age 17 ($M=17.24$, $SD=.64$). In some cases, individual observations were missing (e.g., if they did not complete at least 80% of the items on an individual questionnaire), but almost all subjects completed all aspects of the study. An additional 114 twins completed measures of neuroticism at age 12 and/or 17, but they were not included in these analyses because they did not complete the primary measures of procrastination, internalizing, or externalizing at age 23.

Measures

Procrastination—Procrastination was assessed using the same three questionnaires used in our earlier work using this sample (Gustavson et al., 2014, 2015). The measures were (a) the General Procrastination Scale (GPS; Lay, 1986), (b) the average of the external control, goal neglect, and effort avoidance subscales of the Volitional Components Inventory (VCI; Kuhl & Fuhrmann, 1998), and (c) the prospective and decision-related action orientation vs. hesitation subscale of the Action Control Scale (ACS; Kuhl, 1994). These questionnaires asked about tendencies for domain-general, non-academic procrastination in everyday life.

Fear of failure—We assessed fear of failure with two subscales of the Multidimensional Perfectionism Scale (Frost, Marten, Lahart, & Rosenblate, 1990): (a) the concern for mistakes subscale (CoM), and (b) the parental criticism subscale (PC). These subcomponents of perfectionism were chosen because they are most relevant to the fear of failure (e.g., fearing punishment from parents if their expectations are not met). They are also distinct from other aspects of perfectionism in that they have been shown to correlate positively with procrastination, whereas personal perfectionism (e.g., high standards or expectations) is uncorrelated with procrastination ($r = -.03$, $k = 24$, Steel, 2007)).

Impulsivity—Impulsivity was also measured using the same questionnaires described in our previous work (Gustavson et al., 2014), which assess two components of impulsivity that are most related to procrastination: the tendency to give into one's cravings or to act based on emotion (i.e., impulsive urgency) and the tendency to act spontaneously or without thinking. The measures were (a) the average of the negative urgency, positive urgency, and lack of premeditation subscales of the UPPS-P Impulsive Behavior Scale (UPPS; Lynam, Smith, Whiteside, & Cyders, 2006), and (b) the average of 20 (out of 36) items of the Self-Control Scale (SCS; Tangney, Baumeister, & Boone, 2004). As described in our previous work, this 20-item impulsivity subscale of the SCS was validated using exploratory factor analysis, and excluded items related to health, organization, and procrastination (Gustavson et al., 2014).

Neuroticism—Neuroticism was assessed with 9 items from the Eysenck Personality Inventory (EPQ; Eysenck & Eysenck, 1975). Although we did not have a measure of neuroticism at the same age as the other measures of this study (mean age 23 years), we used neuroticism subscale scores from earlier assessments in early adolescence (mean age 12 years) and late adolescence (mean age 17 years) to create the latent variable for neuroticism described here.

Internalizing—Internalizing psychopathology was measured using lifetime symptom endorsement on the major depressive disorder (MDD) and generalized anxiety disorder (GAD) modules of the Diagnostic Interview Schedule– IV (DIS-IV; Robins et al., 2000). The DIS-IV is a structured interview that assesses symptoms and diagnoses of common psychiatric disorders from the fourth edition of the Diagnostic and Statistical Manual of Mental Health Disorders (American Psychiatric Association, 1994). Because symptom counts were significantly skewed, subject symptom counts were then binned into ordinal variables for each disorder (Derks, Dolan, & Boomsma, 2004). Bins were created to ensure at least 30–50 subjects per bin while maximizing the amount of bins per dependent measures (i.e., to preserve as much variance in the measure as possible). For phenotypic analyses including GAD and MDD, a score of zero was assigned to subjects who endorsed no symptoms ($n = 676$ for GAD, $n = 574$ for MDD), a score of one 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 two was assigned to subjects who met criteria for a diagnosis ($n = 35$ for GAD, $n = 99$ for MDD). We further collapsed the last two bins in genetic analyses (i.e., to reflect one or more symptoms) due to the further reduction in endorsement after separating twins into MZ and DZ groups.

Externalizing—Externalizing psychopathology was measured using lifetime symptom endorsement from the Composite International Diagnostic Interview– Substance Abuse Module (CIDI-SAM; Cottler, Robins, & Helzer, 1989) and the antisocial personality disorder (ASPD) module of the DIS-IV. Like the DIS-IV, the CIDI-SAM is a structured clinical interview used to assess symptoms of substance abuse and dependence alcohol, tobacco, cannabis, and illicit drugs. Like internalizing, these symptom responses were also binned due to the non-normality of the data using similar binning procedures.

ASPD was binned using the same three bins as the phenotypic analyses for GAD and MDD (but ASPD was not reduced to two bins for the genetic analyses): no symptoms ($n = 417$), symptoms but no diagnosis ($n = 199$), and diagnosis ($n = 147$). 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 ($n = 55$; the final two bins were also collapsed for genetic analyses). Tobacco and cannabis use disorder symptoms were binned into three bins for both phenotypic and genetic analyses: 0 symptoms ($n = 509$ for tobacco, $n = 588$ for cannabis), 1 symptom ($n = 112$ for tobacco, $n = 76$ for cannabis), and 2 or more symptoms ($n = 141$ for tobacco, $n = 98$ for cannabis). Finally, given a low prevalence of problematic illicit drug use, we used the substance with the highest number of symptoms endorsed across all illicit substances used by a given individual. Problematic illicit substance use was binned into two bins for both phenotypic and genetic analyses: 0 symptoms ($n = 692$), and 1 or more symptoms ($n = 70$).

Data Analysis

All analyses were conducted using Mplus Version 7.2 (Muthén & Muthén, 2010). Model fit was determined to be good if the root-mean-square error of approximation (RMSEA) was less than .06, and the comparative fit index (CFI) was greater than .95 (Hu & Bentler, 1998). All parameters were deemed significant using chi-squared difference tests (χ^2_{diff}). In models

that included ordinal psychopathology variables, we used the Mplus default estimator of weighted least squares, mean and variance adjusted (WLSMV, with delta parameterization), which uses pairwise deletion for missing data. These models assume an underlying normal distribution for the ordinal variables using a probit model. In phenotypic analyses only, standard errors and chisquares were adjusted using the “type=complex” command to account for the familial clustering of the data and chi-squared difference tests were appropriately scaled using the scaling factors provided in the output (Satorra & Bentler, 2001).

All behavioral genetic analyses were based on the typical assumptions of twin models. Additive genetic influences (A) are assumed to correlate at 1.0 for MZ and 0.5 for DZ twins, because MZ twins share 100% of their alleles identical by descent and DZ twins share, on average, 50% of their alleles identical by descent. Shared environmental influences (C) are assumed to correlate at 1.0 for both MZ and DZ twins because both sets of twins are reared together. Finally, nonshared environmental influences (E) do not correlate, by definition. Twins were assigned to twin 1 vs. twin 2 based on the same random assignment used in our prior work (first used by Friedman et al., 2008).

Preliminary analyses—The latent variables and heritability estimates for procrastination, fear of failure, and impulsivity were reported in previous work (Gustavson et al., 2014), so they are not described in detail here. Preliminary analyses involving individual constructs (e.g., procrastination only) did not reveal substantially different factor loadings from those presented in the larger models presented in the phenotypic analyses below. Further, the heritability estimates of each construct alone did not differ from those presented in the multivariate genetic analyses below.

To account for the sex differences for internalizing and externalizing behaviors (e.g., Eaton et al., 2012), we regressed each indicator of internalizing and externalizing on sex for all phenotypic and genetic/environmental models presented here (preliminary analyses did not reveal any mean sex differences for any measures of procrastination or personality). The effects of sex on each indicator of psychopathology in the full phenotypic model are shown in the supplement (Table S1) and were consistent with previous literature (i.e., females having more endorsement of internalizing and males of externalizing; Eaton et al., 2012).

However, because these analyses still do not account for sex differences in the latent variable correlations, we also examined the phenotypic latent variable correlations separated by sex. In this sex differences model, factor loadings, residual variances, and latent variances on Internalizing and Externalizing could not be collapsed across sex (they were largely comparable but factor loadings were generally slightly lower in males for both Internalizing and Externalizing). Importantly, the results of this model suggested that there were generally no sex differences at the level of latent variable correlations.² Moreover, we note that

²There were three significant sex differences in the sex differences model. The correlation between Procrastination and Impulsivity was greater for females than males, $r_{\text{female}} = .78$, $r_{\text{male}} = .54$, $\chi^2_{\text{diff}}(1) = 5.18$, $p = .023$, as was the correlation between Procrastination and Externalizing, $r_{\text{female}} = .42$, $r_{\text{male}} = .20$, $\chi^2_{\text{diff}}(1) = 4.68$, $p = .031$, and the correlation between Fear of Failure and Internalizing, $r_{\text{female}} = .37$, $r_{\text{male}} = .20$, $\chi^2_{\text{diff}}(1) = 5.54$, $p = .019$. Because all of these effects were in the same direction (i.e., greater correlations for females), and all of the correlations for males remained in the same positive direction, we note this limitation but did not conclude that these sex differences affected the overall interpretation of the results.

splitting the analysis up by sex did not change the conclusions of the structural models shown in Figure 1. Genetic analyses could not be performed separated by sex because of the low endorsement of psychopathology in this sample (i.e., some bins would be very small or empty after separating by MZ/DZ and male/female). Thus, for the purposes of this paper, we used the model collapsing across sex and accounting for mean sex differences only.

Finally, in all genetic analyses involving internalizing, the residual genetic variance on GAD was set to be zero to aid in model convergence. This was done because of the unusually small MZ twin correlation for GAD ($r_{MZ} = .13$, $r_{DZ} = .47$), likely due to the low endorsement of anxiety in this sample. Although these results suggest that we cannot estimate the heritability of GAD in this sample, the cross-twin, cross-trait correlations between GAD and MDD were much more consistent with genetic influences ($r_{MZ} = .45$ – $.47$, $r_{DZ} = .20$ – $.23$), and allowed us to estimate the significant genetic influences on the latent Internalizing factor ($A = 28\%$, see Genetic Analyses). The MZ and DZ twin correlations for other psychopathology measures were consistent with genetic influences (MDD: $r_{MZ} = .35$, $r_{DZ} = .29$; ASPD: $r_{MZ} = .52$, $r_{DZ} = .30$; Tobacco: $r_{MZ} = .66$, $r_{DZ} = .48$; Alcohol: $r_{MZ} = .58$, $r_{DZ} = .37$; Cannabis: $r_{MZ} = .53$, $r_{DZ} = .40$; Illicit substances: $r_{MZ} = .62$, $r_{DZ} = .47$, all $p < .05$).

Results

Descriptive statistics and twin correlations for all of the continuous variables used in the study are displayed in the supplement (Table S2). Throughout the analyses, we refer to latent variables in the structural models using capital letters (i.e., Procrastination), and the constructs that they represent using lower case (i.e., procrastination).

Phenotypic Analysis

The first hypothesis was that individual differences in procrastination are positively associated with both internalizing and externalizing. To examine these associations, we fit a full measurement model involving all of the constructs involved in this study, $\chi^2(98)=150.91$, $p<.001$, RMSEA=.027, CFI=.980. These latent variable correlations are displayed in Table 1 (below the diagonal).

As expected, Procrastination was associated with both Internalizing, $r=.33$, $\chi^2_{diff}(1)=29.02$, $p<.001$, and Externalizing, $r=.31$, $\chi^2_{diff}(1)=34.78$, $p<.001$. These results suggest that individuals who procrastinate more in everyday life are more likely to struggle with internalizing and/or externalizing psychopathology, and suggest that procrastination may account for some of the common variation across these domains of psychopathology.

Both Internalizing and Externalizing were significantly associated with all three of the personality variables included here. Internalizing was moderately associated with higher levels of Fear of Failure, $r=.32$, $\chi^2_{diff}(1)=27.10$, $p<.001$, Impulsivity, $r=.32$, $\chi^2_{diff}(1)=24.20$, $p<.001$, and Neuroticism, $r=.40$, $\chi^2_{diff}(1)=32.70$, $p<.001$. Externalizing was associated strongly with Impulsivity, $r=.62$, $\chi^2_{diff}(1)=147.13$, $p<.001$, and weakly to moderately with Fear of Failure, $r=.18$, $\chi^2_{diff}(1)=9.12$, $p=.004$, and Neuroticism, $r=.26$, $\chi^2_{diff}(1)=24.62$, $p<.001$.

Structural analyses involving procrastination—The second set of hypotheses concerned whether the associations between procrastination and internalizing and externalizing psychopathology are accounted for by personality constructs. To examine these hypotheses, we fit a structural model in which internalizing and externalizing variables were predicted by procrastination and all three personality variables (controlling for the influences of one another). This structural model (Figure 1) had identical fit to the measurement model, and is essentially a multiple regression with four latent independent variables (Procrastination, Fear of Failure, Impulsivity, and Neuroticism) and two latent dependent variables (Internalizing and Externalizing). Although the arrows in Figure 1 may suggest causation, we are not claiming that procrastination causes internalizing or externalizing. These models simply allowed us to partition the covariance between procrastination and psychopathology into variance unique to procrastination and variance shared with other personality constructs.

As we expected, controlling for the other constructs, both Fear of Failure and Neuroticism were still significantly associated with Internalizing, and they accounted for 15% and 31% of the association between Procrastination and Internalizing, respectively.³ In contrast, Impulsivity was not significantly associated with Internalizing after controlling for the other constructs, $\beta=.06$, $\chi^2_{diff}(1)=.43$, $p=.512$. After accounting for these personality constructs, the association between Procrastination and Internalizing was no longer significant, $\beta=.14$, $\chi^2_{diff}(1)=2.78$, $p=.095$.

The findings for Externalizing were also largely in line with our predictions, with some caveats. First, Impulsivity explained all of the positive correlation between Procrastination and Externalizing. The predicted correlation between Procrastination and Externalizing through Impulsivity was $r=.48$, but the total phenotypic correlation was only $r=.31$, resulting in a significant negative association between Procrastination and Externalizing after controlling for Impulsivity (and the other constructs), $\beta=-.19$, $\chi^2_{diff}(1)=6.98$, $p=.008$. Although these results suggest that unique variation in procrastination may relate to lower externalizing psychopathology after controlling for impulsivity, we do not strongly interpret this resulting negative correlation as there was no a priori reason to suspect that procrastination is related to less externalizing psychopathology.

Second, as expected, there was no association between Fear of Failure and Externalizing after controlling for Impulsivity. Finally, exploratory analyses examining Neuroticism indicated that Neuroticism also accounted for about 18% of the variation between Procrastination and Externalizing, $\chi^2_{diff}(1)=10.10$, $p=.002$. However, this effect was not significant in the full model controlling for Impulsivity and Fear of Failure, suggesting that, when controlling for these other traits, the unique variance in Neuroticism primarily explains Internalizing rather than common variance.

³The percent of the correlation between Procrastination and Internalizing that is explained by Fear of Failure (15%), for example, can be computed by dividing the predicted correlation between Procrastination and Internalizing through Fear of Failure (i.e., the correlation of .24 with Fear of Failure multiplied by the path of .21 from Fear of Failure to Internalizing) by the total correlation (.33).

Genetic Analyses

The within-trait and cross-trait cross-twin correlations for the latent constructs are presented in Table 1 (along and above the diagonal). The lack of evidence for shared environmental influences in these cross-twin correlations (i.e., r_{DZ} is not greater than $1/2 * r_{MZ}$ between Procrastination and psychopathology or personality and psychopathology) confirmed our decision to not model shared environmental associations between procrastination and psychopathology, or between personality and psychopathology in all genetic analyses.

Procrastination and internalizing/externalizing—Our third hypothesis concerned the genetic and environmental etiology of the associations between procrastination and psychopathology. We hypothesized that the positive associations between procrastination and psychopathology would be at least partially explained by genetic influences. To test this hypothesis, we fit two biometrical twin Cholesky decompositions, which allowed us to decompose the phenotypic correlations into genetic and environmental components. As shown in Figure 2, a Cholesky decomposition partitions the variance in the second variable in the model into variance shared with the first variable and variance independent of the first variable. The ACEs for the first variable (Procrastination) capture all of the variance in that variable (i.e., they are equivalent to the univariate ACEs for Procrastination). The paths from these ACEs to the second variable indicate the extent to which each biometric component for Procrastination also predicts Internalizing or Externalizing. The second set of ACEs then captures the unique genetic and environmental variance in Internalizing or Externalizing.

The model involving Procrastination and Internalizing is shown in Figure 2A, $\chi^2(116)=120.29$, $p=.374$, RMSEA=.014, CFI=.992. Genetic influences explained about 40% of the variation in Procrastination at the latent level ($.63^2$) and about 28% of the variation in Internalizing ($.46^2 + .26^2$). Importantly, most of the genetic influences on Internalizing were shared with the genetic influences on Procrastination (A_{Proc}), explaining about 21% of the total variation in Internalizing and resulting in a strong genetic correlation between the two constructs, $r_g=.87$, $\chi^2_{diff}(1)=11.02$, $p=.001$. After accounting for the shared genetic influences with Procrastination, the genetic influences unique to Internalizing (A_{Int}) were not significant, $\chi^2_{diff}(1)=.02$, $p=.904$.

Nonshared environmental influences accounted for 61% of the variation in Procrastination and 21% of the variation in Internalizing, but these influences were not significantly correlated, $r_e=.24$, $\chi^2_{diff}(1)=.96$, $p=.326$. Shared environmental influences accounted for the remaining 53% of the variation in Internalizing, but because shared environmental influences do not account for variation in Procrastination (Gustavson et al., 2014), they could not account for the covariation between Procrastination and Internalizing. In summary, the phenotypic correlation between Procrastination and Internalizing ($r=.33$) was primarily due to common genetic influences ($r_g=.87$).

Similar results were observed for the model of Externalizing and Procrastination, shown in Figure 2B, $\chi^2(289)=310.50$, $p=.184$, RMSEA=.019, CFI=.989. Externalizing was explained primarily by genetic influences, explaining 77% of the variation. Genetic influences on Procrastination (A_{Proc}) explained about 14% of the variation in Externalizing, with a moderate genetic correlation between the two constructs, $r_g=.43$, $\chi^2_{diff}(1)=13.37$, $p<.001$,

but there were also substantial unique genetic influences on Externalizing (A_{Ext} , explaining 62% of Externalizing). The nonshared environmental correlation between Procrastination and Externalizing was nonsignificant, $r_e=.18$, $\chi^2_{diff}(1)=2.33$, $p=.117$, despite the fact that nonshared environmental influences accounted for about 23% of the variation in Externalizing in this sample. Shared environmental influences on Externalizing were estimated at 0% for Externalizing (though residual c^2 influences were estimated at .01 for ASPD and .10-.23 for substance use variables). Therefore, as with Internalizing, the phenotypic correlation between Procrastination and Externalizing ($r=.31$) was largely due to shared genetic influences ($r_g=.43$); however, the remaining variation in Externalizing was explained primarily by unique genetic influences, whereas the remaining variation in Internalizing was explained by unique shared environmental influences.

Personality, procrastination, and internalizing/externalizing—Although the focus of our second hypothesis concerning the associations among procrastination, personality, and internalizing/externalizing was accomplished through structural models of the phenotypic data, we also fit some genetic models to further understand how much of the genetic covariance between procrastination and psychopathology was related to these personality traits (as well as the genetic and environmental etiology of the relations between personality and Procrastination).

Trivariate genetic Cholesky decompositions are displayed in Table 2. These are similar to the decompositions displayed in Figure 2, but they also include one personality construct as the first variable in each model (six models total). For example, the top model of Table 2 displays the genetic/environmental associations between Fear of Failure, Procrastination, and Internalizing. The first two columns represent factor loadings from the genetic/environmental influences on Fear of Failure on all three constructs (A_{Person} , E_{Person}). That is, the genetic influences on the personality trait of Fear of Failure are allowed to also predict Procrastination and Internalizing. The next two columns provide factors loadings for the remaining genetic/environmental influences on Procrastination not already accounted for by Fear of Failure (A_{Proc} , E_{Proc}), which are also allowed to predict Internalizing. That is, the path from A_{Proc} to Internalizing captures the semipartial genetic correlation between Procrastination and Internalizing, controlling for genetic variance in Fear of Failure. Finally, as in Figure 2, there are genetic/environmental influences unique to Internalizing (A_{Int} , C_{Int} , E_{Int}). Therefore, to the extent that genetic influences on a given personality variable account for the association between Procrastination and Internalizing (or Externalizing), we expect to see significant factor loadings from that personality variable (A_{Person}) predicting personality, Procrastination, and Internalizing, and residual genetic influences on Procrastination (A_{Proc}) no longer significantly predicting Internalizing.

For Internalizing (Table 2a), genetic influences on Neuroticism accounted for 69% of the variation in Neuroticism ($.83^2$), 12% of the variation in Procrastination ($.34^2$), and 37% of the variation in Internalizing ($.61^2$). Importantly, after accounting for these influences, the unique genetic influences on Procrastination remained significant ($.53^2$, explaining 28% variation) but these influences no longer explained a significant amount of variation of Internalizing ($.16^2$, or 3%), $\chi^2_{diff}(1)=1.06$, $p=.302$. There was no evidence that Internalizing was explained by environmental influences on Neuroticism (<1% variation), suggesting that

Neuroticism accounted for the association between Procrastination and Internalizing solely due to genetic influences. Similar results were obtained for Impulsivity (i.e., the association between Procrastination and Internalizing was accounted for by genetic but not environmental influences on Impulsivity), but these associations are not described in detail, as Impulsivity did not account for this phenotypic association after controlling for Neuroticism and Fear of Failure.

The association between Internalizing and Procrastination with respect to Fear of Failure was the only one with some evidence (albeit weak) for environmental influences. Nonshared environmental influences explained 48% of the variation in Fear of Failure, and these influences also explained 4% of the variation in Procrastination, $\chi^2_{diff}(1)=7.92$, $p=.005$, and 7% of the variation in Internalizing, $\chi^2_{diff}(1)=3.78$, $p=.052$, though this latter effect was only marginally significant.⁴ Genetic influences on Fear of Failure did not account for a significant portion of variation in Procrastination, 3%, $\chi^2_{diff}(1)=3.47$, $p=.063$, nor Internalizing, 5%, $\chi^2_{diff}(1)=2.65$, $p=.103$. In other words, Fear of Failure accounted for 26% of the covariation between Procrastination and Internalizing in this genetic model, and this covariance may be due to environmental (15%) more so than genetic sources (11%). However, most of the association between Procrastination and Internalizing was due to shared genetic influences on Procrastination not associated with Fear of Failure (71%), and results in the previous paragraph suggest that some of this genetic covariance is related to Neuroticism.

Similar results were observed for the models involving Externalizing (Table 2b), suggesting that Impulsivity largely accounted for the association between Procrastination and Externalizing through genetic factors. Genetic influences on Impulsivity ($A = 50\%$) explained 37% of the variation in both Procrastination and Externalizing. Although there was little genetic variance unique to Procrastination in this model (4%), it was negatively associated with Externalizing, $\chi^2_{diff}(1)=12.71$, $p<.001$. Finally, although not as strong as the genetic influences, the environmental influences on Impulsivity ($E = 50\%$) also accounted for some variation in Procrastination, 10%, $\chi^2_{diff}(1)=25.57$, $p<.001$, and Externalizing, 7%, $\chi^2_{diff}(1)=17.23$, $p<.001$. In summary, Impulsivity accounted for all of the positive correlation between Procrastination and Externalizing ($r=.31$), and this was mostly due to genetic factors (81%) but partially to environmental influences (19%). The remaining variation in Procrastination predicted a small negative association with Externalizing ($r=-.15$), also mostly due to genetic influences (86%) rather than environmental influences (14%).

Discussion

The current study tested three hypotheses regarding the etiology of the association between individual differences in procrastination and internalizing and externalizing

⁴In fact, there was also evidence for a nonshared environmental association between Fear of Failure, Procrastination, and Externalizing. In the model with Externalizing, the nonshared environmental influence on Fear of Failure significantly explained 3% of the variation in Procrastination, $\chi^2_{diff}(1)=10.60$, $p<.001$, and 2% of the variation in Externalizing, $\chi^2_{diff}(1)=4.90$, $p=.027$. However, we do not discuss these results in detail because Fear of Failure did not account for any of the association between Procrastination and Externalizing after controlling for Neuroticism and Impulsivity.

psychopathology. First, the results confirmed that at the latent variable level, higher levels of procrastination were associated with more internalizing symptoms ($r=.33$) and externalizing symptoms ($r=.31$). Second, we used relevant personality variables to explore whether the associations between procrastination and internalizing/externalizing were due to broadband-specific variation (i.e., due to fear of failure and impulsivity) and/or common variation between internalizing and externalizing (i.e., due to neuroticism). Phenotypic results from the model examining the three personality variables simultaneously (Figure 1) were consistent with two of our hypotheses: (a) that the association between procrastination and internalizing occurs through fear of failure, and (b) that the association between procrastination and externalizing occurs through impulsivity. The third hypothesis, that neuroticism would be a common feature for the relations between procrastination and both internalizing and externalizing was supported in a model with neuroticism alone. However, neuroticism only accounted for the association between procrastination and internalizing after controlling for the other personality variables. Third, we confirmed the hypothesis that the phenotypic correlations between procrastination and internalizing/externalizing would be primarily due to shared genetic influences, which were moderate to strong ($r_g = .87$ for internalizing, $r_g = .43$ for externalizing). However, we also found some evidence for environmental covariance with fear of failure and impulsivity.

Together, these results confirm that procrastination tendencies, which many people struggle with on a day-to-day basis, are associated with general liabilities to experience symptoms of internalizing and externalizing psychopathology. These weak-to-moderate associations at the phenotypic level are moderate or strong when focusing on underlying genetic influences. Furthermore, this trait behavioral tendency to delay action is deeply intertwined with psychopathology (i.e., through personality traits and genetic influences), and may give important insight into how these internalizing and externalizing behaviors are related. The primary implications of this work are discussed in three sections: (a) implications for theories of psychopathology and personality, (b) theoretical implications for research on procrastination and mental health, and (c) limitations of the study.

Theoretical Implications for Psychopathology and Personality

These findings help characterize the structure of internalizing and externalizing symptoms and their correlations with commonly studied personality traits (Hink et al., 2013; Krueger et al., 2001; Tackett et al., 2013). The inclusion of personality constructs enabled us to show that the associations between procrastination and internalizing and externalizing psychopathology here may be in part due to broadband-specific factors in addition to common factors for internalizing and externalizing. The full correlational model (Table 1) suggested that procrastination and all personality variables account for common variance in internalizing and externalizing (i.e., all constructs were positively correlated with one another). However, when we controlled for the inter-relations among the personality variables (Figure 2), we also found evidence for broadband-specific associations. Specifically, fear of failure and neuroticism both accounted for the association between procrastination and internalizing, while impulsivity uniquely accounted for the association between procrastination and externalizing (controlling for the other personality variables).

These results are largely consistent with previous work on impulsivity, neuroticism and psychopathology, but revealed some interesting new patterns. First, the results concerning impulsivity are important considering recent work that has suggested that impulsivity is not only related to externalizing, but also to internalizing psychopathology (Granö et al., 2007; Johnson et al., 2013). Although we expected that impulsivity would primarily account for the association between procrastination and externalizing, it is not surprising that impulsivity was also correlated with internalizing given these recent views of impulsivity as a common feature. However, here impulsivity was no longer predictive of internalizing after controlling for other constructs, suggesting that impulsivity's shared variance with internalizing may not exist above and beyond its association with neuroticism, fear of failure, and procrastination. Similarly, neuroticism was no longer correlated with externalizing after controlling for impulsivity, fear of failure, and procrastination, suggesting that it also may be a common feature primarily because of its correlation with other personality constructs. Therefore, these results suggest that the unique variances in impulsivity and neuroticism primarily explain broadband-specific variance in psychopathology.

Our results regarding impulsivity, procrastination, and externalizing give further insight into our previous findings regarding procrastination and impulsivity (Gustavson et al., 2014). In this earlier work on the same sample, procrastination and impulsivity had identical genetic influences ($r_g = 1.0$). However, in genetic models involving externalizing presented here (Table 2b), there was evidence for unique genetic influences on procrastination (that were negatively correlated with externalizing). These results provide some evidence for some genetic separability between procrastination and impulsivity (Loehlin & Martin, 2014). However, even with these potential differences, the genetic correlation between procrastination and impulsivity was still quite high ($r_g = .99$ and $.95$ in Table 2a and 2b, respectively), suggesting that procrastination and impulsivity are largely indistinguishable at the genetic level.

Although the primary results of the genetic/environmental decompositions suggested that procrastination was associated with psychopathology primarily through genetic influences, it is worth noting the environmental associations driven by fear of failure and impulsivity. When examining procrastination and internalizing or externalizing alone, there was no evidence for nonshared environmental influences explaining their phenotypic correlations (Figure 2). However, the nonshared environmental variance in impulsivity explained about 10% of the variation in procrastination and 7% in externalizing. Situational factors may also play some role in the association between procrastination and psychopathology, perhaps such as when risk for upsetting important people is high, when future events are highly unpredictable, or when environments involve tempting distractions (Conway et al., 2002).

Finally, it is important to note the caveats concerning any causal inferences drawn from these correlational data. We expected that fear of failure, impulsivity, and neuroticism would be relevant to the association between procrastination and psychopathology in part because they have been linked to both sets of constructs, but also based on theoretical models that implied causation. For example, we described a potential bi-directional association between fear of failure and procrastination where concern over mistakes leads to procrastination, which in turn heightens anxiety and leads to more procrastination. We could not test for the

bi-directionality of these associations in the context of this study, but it was clear that fear of failure is relevant to the association between procrastination and internalizing psychopathology, and that it exists even after accounting for impulsivity and neuroticism. Similarly, these correlational data cannot prove that procrastination and impulsivity are risk factors for externalizing, but the results of the genetic analyses suggest that underlying shared genetic risk factors predispose individuals for procrastination, impulsivity, and externalizing. Therefore, although future research is needed to verify hypotheses regarding some of the causal associations between these constructs, these findings are generally consistent with the theoretical models proposed in the introduction.

Theoretical Implications for Procrastination

This work also extends the growing literature on the associations between procrastination and mental and physical health (Sirois & Kitner, 2015; Sirois et al., 2015; Tice et al., 2001; Wohl et al., 2010) to symptoms of multiple internalizing and externalizing disorders. Although the sample described here was not drawn from a clinical population, many individuals in the sample reported lifetime symptoms of psychopathology in one or more domains, and we were able to model these individual differences in psychopathology symptoms at the latent internalizing and externalizing levels. These findings extend and support some of the existing work linking procrastination to depression and anxiety symptoms (Beutel et al., 2016), and suggest that procrastination is also associated with some forms of externalizing (e.g., substance abuse and dependence symptoms, antisocial personality disorder symptoms).

These results also advance our understanding of procrastination by showing that it fits in to the well-studied genetic risk underlying both internalizing and externalizing psychopathology (Hink et al., 2013; Kendler & Myers, 2014). In particular, procrastination tendencies seem to be influenced by the same genetic factors that influence multiple forms of psychopathology and associated personality traits. Hence, as we gain understanding of these genetic factors for psychopathology and personality, we will be able to translate that knowledge to an understanding of the biological mechanisms underlying procrastination (and vice versa). As with all complex traits, the genetic influences described here likely reflect the collective contribution of thousands of genetic effects (Chabris, Lee, Cesarini, Benjamin, & Laibson, 2015).

These associations are also relevant to existing theoretical perspectives of procrastination, such as the procrastination-health model. According to this model, procrastination is associated with negative health outcomes primarily because procrastination causes stress, which in turn affects immune functioning and health behaviors (Sirois & Kitner, 2015; Sirois, Melia-Gordon, & Pychyl, 2003). Our results extend these findings on procrastination and health to psychopathology phenotypes, demonstrate how personality variables are relevant to these associations, and suggest that genetic factors underlie the associations between procrastination and health. Psychopathology may cause procrastination, in turn causing stress and poor physical health, but it is also possible that mental health is affected after physical health was been compromised. If both possibilities exist, it would suggest circular causal associations between procrastination, stress, physical health, and mental

health. Again, however, causal inferences cannot be made on the basis of the present correlational analyses.

Finally, these results are also relevant to the recent work on developing successful interventions to treat chronic procrastination. There has been a recent surge in published studies examining the effectiveness of procrastination-related interventions, many of which utilize established clinical treatment approaches (Glick & Orsillo, 2015; Rozental, Forsell, Svensson, Andersson, & Carlbring, 2015; Toker & Avci, 2015). Our results suggest that interventions focused on procrastination could also be of benefit with respect to associated psychopathology.

Limitations of the Study

These results are informative to understanding how these associations between procrastination, personality, and psychopathology play out in everyday situations and at the trait level. However, although enough individuals from the general population reported psychopathology symptoms to model these associations, we acknowledge that these results may not generalize to a clinically ascertained sample. Similarly, the low endorsement of psychopathology in this sample reduced our ability to examine how procrastination may predict specific psychopathologies. Therefore, we also acknowledge the possibility that narrow-band associations may exist above and beyond those described here (e.g., procrastination associated with the development of anxiety symptoms due to test anxiety and/or aversion to specific situations). We also did not have adult measures of hyperactivity/inattention, which is sometimes included in externalizing models (e.g., Caspi et al., 2014; Weiss et al., 1998).

The restricted range of psychopathology in this sample also made it difficult to quantify the effects of sex on the associations observed here, and made it impossible to examine sex differences in the genetic and environmental associations. However, although sex differences in internalizing and externalizing are well established (Eaton et al., 2012), there are only small sex differences in levels procrastination ($r = -.10$; Steel & Ferrari, 2013), suggesting that they may play little role overall.

Finally, our measure of neuroticism in this sample was limited in that it was assessed about 6 to 11 years earlier than the other assessments in the study (mean ages 12 and 17 vs. mean age 23). Although neuroticism still predicted unique variance in internalizing after accounting for procrastination and other personality variables, the fact that it was not measured at the same time point may have contributed to the lack of association between neuroticism and externalizing after controlling for the other variables (i.e., impulsivity). A related limitation is that we did not also have a measure of conscientiousness, a strong correlate of procrastination that may entirely account for the association between procrastination and neuroticism (Steel, 2007; Steel & Klingsieck, 2016). Therefore, is also possible that the associations between procrastination, neuroticism, and internalizing described here were due to conscientiousness rather than neuroticism.

Concluding Remarks

In summary, this study was the first to show that individual differences in procrastination are associated with both general internalizing and externalizing symptoms, but likely through different mechanisms (i.e., with internalizing symptoms through fear of failure and neuroticism and with externalizing symptoms through impulsivity). The associations described here were primarily due to genetic influences, suggesting that a common set of genetic risk factors predispose individuals to engage in procrastination and experience psychopathology symptoms, but that the environmental influences on procrastination are largely independent of those that influence psychopathology. Further work on these shared genetic influences underlying procrastination and personality may continue to inform the nature of the genetic liability to develop psychopathology, and explain its behavioral antecedents with respect to sub-clinical symptomology in the general population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Structural equation regression model displaying the associations between latent procrastination and personality constructs as simultaneous predictors of psychopathology (controlling for their inter-relations). Factor loadings on each latent variable and effects of sex on psychopathology variables are not shown here, but were similar to the full model presented in Table 1 and supplemental Table S1. Significant pathways and residual correlations (e.g., between Internalizing and Externalizing) are noted with bolded black text and solid arrows ($p < .05$).

Figure 2.

Genetic and environmental associations between (A) Procrastination and Internalizing and (B) Procrastination and Externalizing. Preliminary analyses indicated that the shared environmental influences on Procrastination were estimated at 0, so they were not estimated in these models, nor were the shared environmental covariances between Procrastination and Internalizing or Externalizing. Residual genetic and shared/nonshared environmental influences were estimated on individual measures but not shown here, with the following exceptions: No residual shared environmental influences were estimated for the measures of procrastination, and no residual genetic influences were estimated for GAD. Significant

factor loadings and correlations are displayed with solid lines in black, bolded text ($p < .05$). GPS = General Procrastination Scale, VCI = Volitional Components Inventory, ACS = Action Control Scale, GAD = Generalized Anxiety Disorder, MDD = Major Depressive Disorder, ASPD = Antisocial Personality Disorder, ALC = Alcohol, TOB = Tobacco, CAN = Cannabis, Illicit = Illicit substance with the most symptoms.

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Table 1

Phenotypic Correlations and Cross-Twin Latent Variable Correlations (MZ/DZ)

	1	2	3	4	5	6
1. Procrastination	.48 / .16	.13 / .00	.44 / .23	.31 / .08	.29 / .09	.27 / .01
2. Fear of Failure	.24	.78 / .06	.19 / -.11	.23 / .02	.22 / -.03	.12 / -.10
3. Impulsivity	.65	.34	.54 / .14	.31 / .04	.36 / .16	.45 / .19
4. Neuroticism	.35	.20	.34	.93 / .26	.58 / .10	.34 / .06
5. Internalizing	.33	.32	.32	.40	.85 / .69	.52 / .38
6. Externalizing	.31	.18	.62	.26	.55	.83 / .40

Note: Latent variable correlations from the phenotypic measurement model are displayed below the diagonal. Within-trait cross-twin correlations are displayed along the diagonal, and cross-trait cross-twin correlations are displayed above the diagonal. The number on the left is the cross-twin correlation for MZ twin pairs, and the number on the right is the cross-twin correlation for DZ twin pairs. In the cross-twin model, within-twin correlations were constrained to be equal (e.g., the correlation between twin 1 Procrastination and twin 1 Impulsivity was constrained to equal the same correlation between twin 2 Procrastination and twin 2 Impulsivity), as were the cross-twin correlations (e.g., twin 1 Procrastination with twin 2 Impulsivity was constrained to equal twin 2 Procrastination with twin 1 Impulsivity). Factor loadings on each latent variable are not displayed here, but are presented in the supplement (Table S1) for the correlational model. Significant correlations are displayed in bold ($p < .05$).

Table 2
Cholesky Decompositions Involving Procrastination, Personality, and Internalizing/Externalizing

	Personality*		Procrastination		Psychopathology	
	A _{Person}	E _{Person}	A _{Proc}	E _{Proc}	A _{Int}	E _{Int}
A. Models with Internalizing						
Fear of Failure						
Fear of Failure	.72	.69				
Procrastination	.17	.20	.61	.75		
Internalizing	.23	.27	.41	.03	.23	.35
Impulsivity						
Impulsivity	.69	.72				
Procrastination	.63	.30	.09	.71		
Internalizing	.50	.03	-.34	.10	.00	.36
Neuroticism						
Neuroticism	.83	.57				
Procrastination	.34	.19	.53	.75		
Internalizing	.61	-.01	.16	.12	.00	.37
B. Models with Externalizing						
	A _{Person}	E _{Person}	A _{Proc}	E _{Proc}	A _{Ext}	E _{Ext}
Fear of Failure						
Fear of Failure	.65	.76				
Procrastination	.13	.17	.62	.76		
Externalizing	.10	.14	.36	.06	.79	.46
Impulsivity						
Impulsivity	.71	.71				
Procrastination	.61	.32	.21	.70		
Externalizing	.61	.27	-.63	-.03	.00	.40
Neuroticism						
Neuroticism	.85	.77				
Procrastination	.33	.21	.54	.75		
Externalizing	.34	-.02	.23	.10	.77	.47

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Note: Six Cholesky decompositions involving Procrastination, one of the three personality constructs and (A) Internalizing or (B) Externalizing. Numbers indicate standardized path coefficients from the latent genetic/environmental factors to the latent variables. Variance explained by can be computed by squaring these path coefficients. For example, the first model decomposes variation in Internalizing into genetic and environmental influences shared with Fear of Failure (APers, EPers), genetic/environmental influences shared with Procrastination not already accounted by Fear of Failure (AProc, EProc), and genetic/environmental influences unique to Internalizing (AInt, CInt, EInt). Significant factor loadings are displayed in bold ($p < .05$).