

# Understanding the Cognitive and Genetic Underpinnings of Procrastination: Evidence for Shared Genetic Influences With Goal Management and Executive Function Abilities

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Previous research has suggested that individual differences in procrastination are tied to everyday goal-management abilities, but little research has been conducted on specific cognitive abilities that may underlie tendencies for procrastination, such as executive functions (EFs). In this study, we used behavioral genetics methodology to investigate 2 hypotheses about the relationships between procrastination and EF ability: (a) that procrastination is negatively correlated with general EF ability, and (b) that this relationship is due to the genetic components of procrastination that are most related to other everyday goal-management abilities. The results confirmed both of these hypotheses. Procrastination was related to worse general EF ability at both the phenotypic and genetic levels, and this relationship was due to the component of procrastination shared with self-report measures of everyday goal-management failures. These results were observed even after controlling for potential self-report biases stemming from the urge to respond in a socially desirable manner. Together, these findings provide strong evidence for growing theories of procrastination emphasizing the importance of goal-related cognitive abilities and further highlight important genetic influences that underlie procrastination.

**Keywords:** procrastination, executive control, goal management, individual differences, heritability

People vary greatly in their tendency to procrastinate, that is, voluntarily yet irrationally delaying an intended course of action. The correlates and etiology of such individual differences in procrastination have received growing attention in psychological research (e.g., Gustavson, Miyake, Hewitt, & Friedman, 2014; Pychyl & Flett, 2012; Steel, 2007; Steel & Ferrari, 2013) and in the realm of popular science and self-help books (e.g., Ferrari, 2010; Pychyl, 2013a; Steel, 2010). The tendency to procrastinate is persistent across the life span (Steel, 2007), is correlated with many personality traits such as conscientiousness and impulsivity (Schouwenburg & Lay, 1995; Steel, 2007; van Eerde, 2003), and is correlated with demographic variables such as age, education, and marital status (Steel & Ferrari, 2013). Additionally, recent work has also demonstrated that variation in procrastination reflects roughly equally genetic and environmental influences (Gustavson et al., 2014). Despite such advances in understanding the correlates and etiology of this pervasive and problematic

behavioral trait,<sup>1</sup> what has been lacking in the literature is systematic attempts to specify crucial cognitive abilities underlying individual differences in procrastination. Such attempts are necessary to attain a more complete, mechanistic model of procrastination.

One particularly promising set of cognitive abilities in this regard is executive functions (EFs), a set of higher-level, goal-driven cognitive processes that help control and regulate one's thought and behavior (Miyake & Friedman, 2012; Miyake et al., 2000). EFs are considered to play an important role in the effective management of goals and subgoals (Miyake & Friedman, 2012), which in turn has been theorized to be crucial for successfully avoiding procrastination (e.g., Gropel & Steel, 2008; Gustavson et al., 2014; Krause & Freund, 2014; Steel & Konig, 2006). In fact, there is even some initial evidence that self-report measures of EF ability significantly predict individual differences in procrastination (Rabin, Fogel, & Nutter-Upham, 2011). To the best of our knowledge, however, no prior research has investigated the relationship between procrastination and performance-based measures of EF ability, such as those typically used in laboratory settings, in a rigorous, large-scale study.

In this multivariate twin study, we examined how individual differences in procrastination are associated with EF ability at the phenotypic and genetic levels. Young adult twins completed mul-

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<sup>1</sup> Throughout this article, we discuss procrastination as a stable *trait*-like construct. Although procrastination could also be construed as a *state* variable reflecting situation-specific behaviors or phenomena that arise at particular points in time, conceptualizing procrastination as a stable trait is not only consistent with leading perspectives on procrastination but also supported by existing evidence (e.g., Schouwenburg & Lay, 1995; Steel, 2007, 2010; Steel & Ferrari, 2013).

multiple measures of procrastination as well as well-established performance-based measures of EFs. Using structural equation modeling and behavioral genetics methodology, we tested two specific, *a priori* hypotheses about the relationship between procrastination and EF ability: (a) that individual differences in procrastination are related to worse general EF ability (i.e., common EF ability); and (b) that this relationship is due to the genetic component of procrastination shared with the ability to effectively manage goals in everyday situations.

### Existing Goal-Management Accounts of Procrastination and Our Theoretical Framework

Although previous empirical research on procrastination has focused largely on the personality and demographic correlates of procrastination (Steel, 2007; Steel & Ferrari, 2013), some recent theories have noted the importance of goal-related abilities that may contribute to individual differences in procrastination (Krause & Freund, 2014; Kuhl, 1994; Steel & Konig, 2006). Temporal-motivation theory (Steel & Konig, 2006), for example, proposes that an individual's goal-setting ability underlies his or her tendency to procrastinate (Steel & Konig, 2006). This hypothesis has received some support in a large-sample study, although the assessment of the goal-setting ability is limited in that it is based on a single 4-item self-report questionnaire (Gropel & Steel, 2008). Others have proposed that different types of goal focus may help reduce procrastination depending on situational factors related to the specific goal (Krause & Freund, 2014). Specifically, Krause and Freund (2014) have proposed that a focus on process, or *how* a goal should be pursued, may reduce procrastination after individuals experience failure or problems, whereas a focus on outcome, or *why* a goal should be pursued, may be more effective when task aversiveness is high, although this hypothesis has not been empirically tested. Finally, Kuhl's (1994) theory of state and action orientation suggests that individuals high in procrastination (i.e., state-oriented individuals) have difficulty discriminating between goals and often attribute goals to the self even when they are unrealistic or incompatible with their self-identity (Kuhl, 1994, 2000). Combined with personal projects analysis—a methodological approach involving goal generation—this theory has motivated research linking procrastination to task aversiveness, boredom, uncertainty, and guilt (Blunt & Pychyl, 2000, 2005; Pychyl & Binder, 2004). As these examples illustrate, there is increasing theoretical emphasis on the crucial role of goal management in procrastination, even though further evidence is needed to substantiate the respective claims.

Our own previous work provides more direct evidence for the role of goal-management abilities in procrastination (Gustavson et al., 2014). This prior study investigated the relationship between procrastination and one of its well-studied correlates, impulsivity (Ferrari, 1993; Steel, 2007), at the level of latent variables. Impulsivity is typically conceptualized as the tendency to think and act quickly according to one's desires (Whiteside & Lynam, 2001). Thus, on the surface, it may seem counterintuitive to think that those two traits are positively associated with each other (i.e., a greater tendency to procrastinate is significantly associated with a higher degree of impulsivity). From the perspective of goal management, however, this positive association makes sense in that procrastination is all about failing to act on one's long-term goals,

whereas impulsivity is about giving into one's immediate desires and cravings in the face of more important long-term goals. An important result of this earlier study was that as much as three quarters of the shared variation between procrastination and impulsivity at the latent-variable level was accounted for by self-report measures of goal-management failures, namely, failures to keep track of short-term and long-term goals in everyday life (Gustavson et al., 2014). These findings provide empirical support for the suggestion that goal management is an important factor underlying individual differences in procrastination.

Our theoretical framework draws on a goal-based evolutionary account proposed by Steel (2010). This account posits that procrastination has evolved as a by-product of the more ancient trait, impulsivity, which had evolutionary advantages in early (preagricultural) human history when humans needed to satisfy their basic survival needs quickly, without long-term planning. Impulsivity, however, has become problematic in modern societies where humans must juggle many different short-term and long-term goals, while simultaneously resisting their previously beneficial impulsive natures. When modern humans fail to overcome their temptations and desires, they lose track of their important long-term goals and, therefore, procrastinate.

This evolutionary account of procrastination suggests that procrastination has a genetic origin (Steel, 2010), and that the genetic variance in procrastination should highly overlap with that for impulsivity. These predictions were both supported. Specifically, the Gustavson, Miyake, Hewitt, and Friedman (2014) study revealed that procrastination and impulsivity share identical genetic influences (i.e., a genetic correlation of 1.0<sup>2</sup>), and that goal-management abilities account for most of this shared variation at the genetic level as well (about 68%). These results are not only in line with the proposal that procrastination may be an evolutionary by-product of impulsivity (Steel, 2010) but also extend the goal-management accounts of procrastination by suggesting that these traits are connected due to their shared genetic influences.

Together, these theoretical perspectives and empirical findings reviewed here support the notion that goal-management abilities underlie procrastination not only at the phenotypic level but also at the genetic level. This goal-management account of procrastination, however, has yet to be tested using performance-based measures of goal-management abilities, such as those assessed by EF tasks. If goal-management abilities truly underlie individual differences in procrastination, then procrastination should also be related to worse performance on goal-based cognitive tasks. More-

<sup>2</sup>Loehlin and Martin (2014) recently reported a much smaller genetic correlation between procrastination and impulsivity ( $r_g = .30$ ). However, their analyses were methodologically limited in that this estimate was based on a single questionnaire item of procrastination with a binary response and a single scale of impulsivity (a sum of nine binary-response items from two personality questionnaires). When we reanalyzed the Gustavson et al. (2014) data using a comparable method (using each of the 12 items from the Action Control Scale [ACS] that required binary responses and an impulsivity scale constructed from impulsivity items in the Self-Control Scale), the genetic correlations between procrastination and impulsivity across the 12 ACS items ranged from .14 to 1.0, with a median of  $r_g = .59$ , much closer to Loehlin and Martin's (2014) estimate. This reanalysis suggests that the smaller estimate obtained by Loehlin and Martin (2014) stemmed primarily from the limitations of the measures used in their analyses and highlights the importance of examining these relationships at the level of latent variables.

over, this shared variation should be observed at both the phenotypic and genetic levels. Therefore, in this study, we examined the extent to which the phenotypic and genetic overlap between procrastination and everyday goal-management failures is also predictive of variation in performance-based measures of goal-management abilities.

Perhaps the most obvious candidate for a set of goal-related cognitive abilities linked with procrastination are EFs. EFs are a set of general-purpose control mechanisms that are implicated in many types of goal-related abilities and are often measured with objective performance-based tasks (Miyake et al., 2000). They are considered an important component of self-control (Hofmann, Schmeichel, & Baddeley, 2012; Miyake & Friedman, 2012) and have also been linked to individual differences in various clinically and societally relevant domains (e.g., the expression and control of implicit racial biases, Ito et al., 2015; the vulnerability to externalizing behavior problems, Young et al., 2009). Although there are multiple types of EFs (i.e., prepotent response inhibition, mental-set shifting, working memory updating) that are separable at the level of individual differences (the diversity of EFs), it has been shown repeatedly that these different types of EFs also share some underlying commonality (the unity of EFs). According to a recently outlined unity/diversity framework (Miyake & Friedman, 2012), one's goal-management ability—namely, activating and maintaining appropriate goals and thereby effectively guiding lower-level processes—is hypothesized to underlie this general EF ability, termed common EF.

This conceptualization of the common EF factor in the unity/diversity framework predicts that common EF should be a substantial predictor of individual differences in both procrastination and everyday goal-management abilities. In fact, in line with this proposal, a prominent researcher who has studied procrastination extensively has speculated on the potential relationship between EFs and procrastination: “Although there is limited previous research that implicates the frontal system network in the self-regulatory failure of procrastination, no previous research has examined which aspects of EF were most strongly related to procrastination” (Pychyl, 2013b). In a later post, he continued: “Future twin studies that include measures of executive function and conscientiousness will take the emphasis off the risk factor of impulsivity alone in an understanding of the evolutionary etiology of procrastination” (Pychyl, 2014). In this study, we extend our previous work (Gustavson et al., 2014) by relating individual differences in procrastination to those in EFs, as assessed with objective performance-based measures that have been validated in our own previous research (Friedman et al., 2008, 2015; Ito et al., 2015; Miyake & Friedman, 2012; Miyake et al., 2000).

An additional reason to focus on EFs is that self-report measures of procrastination and goal-management failures may be systematically biased in some way. For example, it is not socially desirable to report high levels of procrastination, or frequent tendencies to lose track of important short-term and long-term goals. Thus, these social desirability factors may account, at least in part, for the correlations between procrastination and goal-management failures. In fact, self-reported measures of self-control and impulsivity, which correlate strongly with procrastination and goal-management ability (Gustavson et al., 2014; Steel, 2007), have been shown to correlate substantially with measures of social desirability in multiple studies ( $r = .54-.60$ ; Tangney, Baumeister,

& Boone, 2004). Thus, it is possible that some of the common variation linking self-reported procrastination and self-reported goal-management abilities (as well as other self-regulatory traits like impulsivity) is due to the subjects' biased reports of their own behavior to present themselves in a socially desirable light. Because performance-based measures of cognitive abilities, such as EFs, should be relatively free from potential self-report biases such as social desirability, using those objective performance-based measures of EFs in addition to the self-report measures of goal-management abilities should also help better quantify the contribution of goal-management abilities to procrastination tendencies.

### Hypothesizing the Relationship Between Procrastination and Executive Functions

The guiding theoretical framework of EF for this study is the unity/diversity framework (Friedman et al., 2008, 2015; Miyake & Friedman, 2012). Since our group's initial work (Miyake et al., 2000), EFs have typically been discussed and interpreted from the perspective of a correlated-factors framework, in which three different facets of EFs (e.g., inhibition, shifting, and updating) are considered to be distinct but moderately correlated abilities. However, our most recent conceptualization of EFs has more directly focused on the fact that these different EF abilities share common variance (unity) and have some unique components as well (diversity). In this unity/diversity framework, individual differences in the three correlated EFs can be decomposed into three other latent factors: common EF, updating-specific, and shifting-specific abilities (Friedman et al., 2008; Miyake & Friedman, 2012).

Common EF represents abilities that underlie successful performance on all types of EF tasks. Specifically, common EF ability is hypothesized to reflect the ability to actively maintain task goals and use this goal information to guide lower-level processes (Miyake & Friedman, 2012). Thus, it is the component of EF that should be most closely related to general goal-management ability. The other two abilities are specific to performance on set-shifting tasks (shifting-specific) and working memory updating tasks (updating-specific), respectively, and explain additional variance common to these types of tasks that are not captured by common EF factor. Shifting-specific ability is hypothesized to reflect aspects of cognitive flexibility (e.g., the speed with which goals or mental sets can be replaced), because set-shifting tasks require subjects to flexibly switch between task representations. In contrast, updating-specific ability may reflect effective gating of information or the controlled retrieval from long-term memory. In multiple independent samples, there is no evidence for a separate inhibition-specific factor (Friedman et al., 2008; Ito et al., 2015; Miyake & Friedman, 2012), suggesting that individual differences in prepotent response inhibition can be entirely captured by the common EF factor.

The unity/diversity model of EF was first estimated in a sample of adolescent twins (Friedman et al., 2008), and later replicated in a second wave of EF assessment performed by the same group of twins approximately 6 years later (Friedman et al., 2015) as well as in an independent sample of undergraduate students (Ito et al., 2015). The unity/diversity model, estimated for the twin sample used in this study and based on the second wave of EF assessment, is displayed in Figure 1. As discussed, common EF ability predicts performance on all of the EF tasks, whereas shifting-specific and

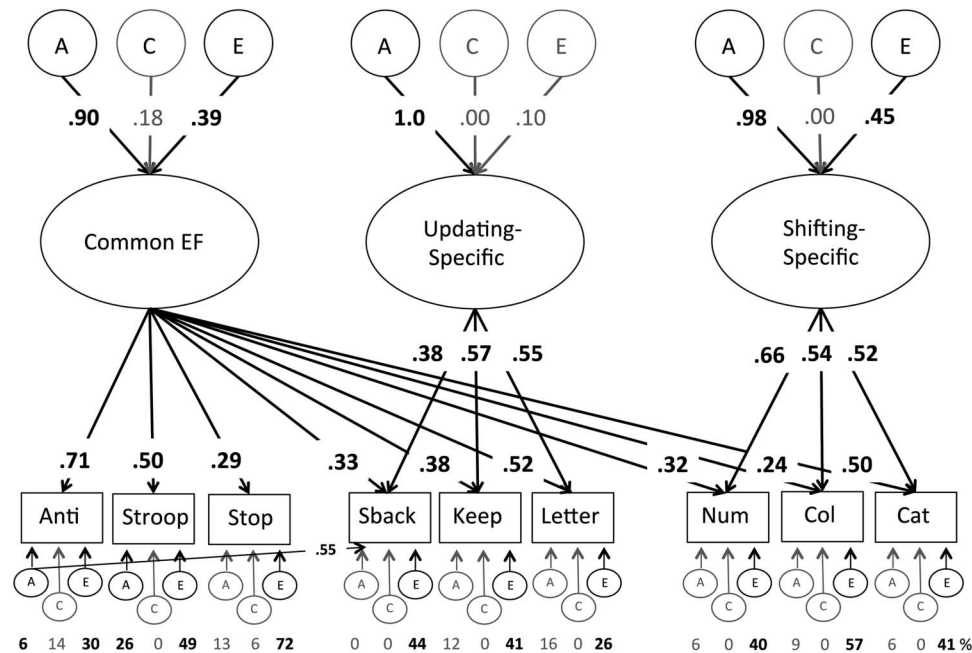


Figure 1. Genetic models for the measures of executive function. Numbers on arrows are standardized factor loadings. Significant factor loadings ( $p < .05$ ) are indicated in bold and with solid black arrows. Anti = antisaccade; Stroop = Stroop task; Stop = Stop Signal task; Sback = Spatial  $n$ -back; Keep = keep track; Letter = letter memory; Num = number-letter; Col = color-shape; Cat = category switch; A = genetic influences; C = shared environmental influences; E = nonshared environmental influences.

updating-specific abilities predict additional variation in the measures of task switching and working memory updating, respectively (although some of the factor loadings for the individual EF tasks are low, those loadings are all statistically significant). Because this sample is genetically informative (i.e., it is comprised of identical and fraternal twins), we know that all three EF factors show a high degree of genetic influences (heritability = 80%–99%). The remaining variation in common EF and shifting-specific abilities are largely influenced by nonshared environmental factors (15%–20%), or environmental factors that are unique to one twin in a pair. There are no significant shared environmental influences (i.e., environmental influences that influence both twins in a pair) on any of the EF abilities.

Based on this unity/diversity framework of EF and the existing evidence regarding procrastination and goal management, two a priori hypotheses can be proposed regarding the relationship between procrastination and EF ability. First, procrastination should be related to worse common EF ability. The results of the only existing study that investigated the relationship between procrastination and EF support this hypothesis (Rabin et al., 2011). In this study, lower scores on seven of nine self-reported EF domains (including the inhibit, shift, and working memory subscales) on a questionnaire-based EF battery known as the BRIEF were related to higher levels of academic procrastination in a large sample of students ( $N = 243$ ). The finding that procrastination is related to most of the BRIEF subscales suggests that procrastination may be related to abilities common across all EF domains (i.e., the common EF component), rather than those specific to some EF tasks and not others (e.g., updating- and shifting-specific abilities).

In addition to testing this first hypothesis, we are also concerned with understanding the etiology of this relationship. We have already shown that individual differences in EF abilities are highly heritable at the level of latent variables (Friedman et al., 2008, 2015). Moreover, our earlier work on procrastination (Gustavson et al., 2014) has shown that, again at the level of latent variables, about half (46%) of the individual differences in procrastination can be accounted for by genetic influences and that almost three-quarters (72%) of the total genetic variation in procrastination is shared with the ability to manage goals in everyday situations. Together, these findings suggest that there may be substantial genetic influences shared between procrastination, EF abilities, and everyday goal-management failures. Thus, the second hypothesis we tested is that the relationship between procrastination and common EF is due to shared genetic influences. Moreover, these influences should be the same as those shared between procrastination and everyday goal-management failures reported in previous research (Gustavson et al., 2014).

Unlike the relationship between procrastination and common EF, we did not have any specific a priori hypotheses for the relationship between procrastination and the updating-specific or shifting-specific EF abilities. Previous research has revealed that updating-specific ability is sometimes correlated with other traits to the same extent as common EF ability, at least for general intelligence (Friedman et al., 2008), whereas shifting-specific ability is often correlated in the opposite direction as common EF ability for different cognitive, personality, and behavior variables (Herd et al., 2014; Miyake & Friedman, 2012). Thus, it is possible that individual differences in procrastination are associated with



poorer updating-specific ability and/or better shifting-specific ability.

### The Current Study

In the current study, we analyzed the data from a large-scale twin study to test the hypotheses that (a) procrastination is related to worse common EF ability, and (b) that this relationship is due to the genetic component of procrastination shared with the ability to effectively manage goals. Because twins performed multiple measures of performance-based EF abilities and completed self-report questionnaires about procrastination and everyday goal-management ability, we were able to model the relationships between these constructs at the level of latent variables. Latent variables reduce measurement error and may be necessary to observe correlations between EF abilities and self-report measures of everyday self-regulation, especially because individual measures of EF are often impure (i.e., any given EF task reflects not only EF ability, but often many other task-specific processes as well; Miyake et al., 2000). Moreover, a recent meta-analysis has shown that the intercorrelations between performance-based and self-reported measures of EFs and other related constructs such as self-control may be low, especially at the level of individual tasks (Duckworth & Kern, 2011).

In addition to using latent variables, we also created residualized scores for our self-report measures of procrastination and goal-management abilities to remove variance due to social desirability. Thus, our self-reported estimates of procrastination and goal-management failures as well as the relationship between these constructs should not be attributable to individual differences in the tendency to respond in a socially desirable manner.

The latent variable models of EFs have been presented in previous work at both the phenotypic and genetic levels (Friedman et al., 2008, 2015). Similarly, in the context of elucidating the nature and etiology of the relationship between procrastination and impulsivity, our earlier work (Gustavson et al., 2014) also presented the phenotypic and genetic models of the relationship between procrastination and everyday goal-management abilities (without controlling for self-report biases like social desirability). However, the relationships among EFs, procrastination, and goal-management abilities have not been examined within a single study. Understanding how these traits are related to one another and quantifying the extent to which they share genetic and environmental influences is an important research goal: It can not only provide necessary support for growing goal-management accounts of procrastination but also help understand how EF abilities play a role in everyday procrastination and goal management.

The first hypothesis, that procrastination is related to worse common EF ability, predicts that there will be a significant negative correlation between these two traits at the latent level. The second hypothesis, that this negative phenotypic correlation between procrastination and common EF is predominantly due to the genetic component of procrastination shared with everyday goal-management failures, predicts that the genetic influences on common EF ability will be negatively correlated with those genetic influences shared between procrastination and goal-management failures. Specifically, if we decompose variation in procrastination into (a) those portions shared with everyday goal-management ability, and (b) those unique to procrastination, then the genetic

influences on common EF should be largely (or even exclusively) correlated with those influences shared between procrastination and goal management (a), rather than those unique to procrastination (b).

### Method

#### Subjects

The analysis was based on 751 individuals (401 females, 350 males) from 386 same-sex twin pairs (206 monozygotic [MZ] and 179 dizygotic [DZ]) and 1 twin pair with unknown zygosity).<sup>3</sup> Twins completed questionnaire and computer measures at about age 23 ( $M = 22.66$ ,  $SD = 1.12$ ) as part of the ongoing Colorado Longitudinal Twin Study. All subjects who completed the EF battery and the self-report questionnaires for procrastination, goal failures, and social desirability were included in these analyses. In some cases, individual observations were missing due to participant, experimenter, or computer errors, or if a subject did not respond to at least 80% of the items on each individual questionnaire (see Table 1 for  $ns$  for each measure). The twins in these analyses are representative of the general population (for detailed characteristics of this sample, see Rhea, Gross, Haberstick, & Corley, 2013).

#### Measures

All self-report questionnaires and lab-based EF tasks were administered on computers. The items in those questionnaires were broad in scope and applicable to various aspects of everyday lives, but also contained some domain-specific items. All the self-report questionnaires used in this study asked about subjects' stable behavioral characteristics (e.g., what they typically do, or what they have done in the past 6–12 months), rather than their transient, state-level characteristics at the time of testing.

These measures reported here were completed as part of a larger battery of tasks and questionnaires (e.g., substance use, psychopathology). They are only a small subset of the measures performed by twins at this time point, but only those measures relevant to this research motivated earlier in the introduction were analyzed in this study.

**Procrastination.** Levels of procrastination were measured with (a) the General Procrastination Scale (GPS; Lay, 1986); (b) the average of three subscales (external control, goal neglect, and effort avoidance) of the Volitional Components Inventory (VCI; Kuhl & Fuhrmann, 1998); and (c) the prospective and decision-related action orientation versus hesitation subscale of the Action Control Scale (ACS; Kuhl, 1994). None of the questionnaires included items related to academic procrastination, because not all subjects were attending college at the time of responding to the questionnaires. Example items from these questionnaires are *I am continually saying "I'll do it tomorrow,"* (GPS), *I readily put*

<sup>3</sup> Because the zygosity for this twin pair was unknown at the time of data analysis and the initial submission of this article, the data from this pair was excluded from all genetic analyses, but included in all phenotypic analyses. Since then, this pair was identified as a DZ pair. Including this pair in the genetic analyses did not change the pattern of results reported here (i.e., all factor loadings changed by .01 at most).

Table 1

*Descriptive Statistics and Twin 1–Twin 2 Correlations for the Measures Used in the Study*

Measure	N	M	SD	Range	Skewness	Kurtosis	Reliability	$r_{MZ}$	$r_{DZ}$
Procrastination									
GPS	751	2.59	.57	1.05–4.60	.04	–.30	.89 <sup>c</sup>	.51*	.09
VCI	750	3.03	.85	1.05–6.19	.35	.03	.92 <sup>c</sup>	.41*	.19*
ACS	747	7.90	2.90	.00–12.00	–.47	–.59	.76 <sup>c</sup>	.31*	.05
Goal failures									
CFQ	750	1.39	.45	.12–3.20	.30	.54	.90 <sup>c</sup>	.49*	.24*
PMQ	736	.63	.29	.00–1.70	.47	.08	.91 <sup>c</sup>	.46*	.14
Executive function tasks									
Antisaccade	748	62 %	16.10	20–96	–.13	–.67	.90 <sup>c</sup>	.61*	.40*
Stroop	737	156 ms	74.15	–73–387	.71	.71	.96 <sup>b</sup>	.52*	.08
Stop signal	735	215 ms	30.35	116–315	–.23	.25	.63 <sup>c</sup>	.27*	.22*
Number–letter	748	246 ms	157.12	–241–735	.91	.92	.91 <sup>b</sup>	.51*	.17*
Color–shape	743	221 ms	181.51	–239–792	1.05	1.19	.90 <sup>b</sup>	.32*	.23*
Category switch	747	198 ms	160.79	–81–735	1.14	1.28	.94 <sup>b</sup>	.50*	.21*
Spatial <i>n</i> -back <sup>a</sup>	749	–.01	.91	–2.7–2.7	–.31	–.03	.75 <sup>b</sup>	.55*	.16*
Letter memory	749	70%	13.27	38–100	.22	–.64	.92 <sup>c</sup>	.69*	.34*
Keep track	749	72%	9.13	44–96	–.36	.11	.66 <sup>c</sup>	.60*	.15
Social desirability									
MCSDS	745	.60	.16	.09–.97	–.26	–.15	.78 <sup>c</sup>	.44*	.30*

Note. GPS = General Procrastination Scale; VCI = Volitional Components Inventory; ACS = Action Control Scale; PMQ = Prospective Memory Questionnaire; CFQ = Cognitive Failures Questionnaire; MCSDS = Marlow-Crowne Social Desirability Scale.  $r_{MZ}$  and  $r_{DZ}$  = Twin 1–Twin 2 correlations for monozygotic and dizygotic twins, respectively.

<sup>a</sup> Accuracy scores were arcsine transformed for two- and three-back, z-scored, then averaged. <sup>b</sup> Internal reliability was calculated by adjusting odd/even or part1/part2 correlations with the Spearman-Brown prophecy formula. <sup>c</sup> Internal reliability was calculated using Cronbach's alpha.

\*  $p < .05$ .

difficult things aside (VCI), and *When I know I must finish something soon: A. I have to push myself to get started, or B. I find it easy to get it done and over with* (ACS).

**Goal failures.** Levels of goal-management failures, or the tendency to forget to activate, maintain, and retrieve task goals, were measured with (a) the total score of the Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, FitzGerald, & Parkes, 1982); and (b) the log-transformed average of items from three subscales (short-term, long-term, and internally cued) of the Prospective Memory Questionnaire (PMQ; Hannon, Adams, Harrington, Fries-Dias, & Gipson, 1995). Example items are *Do you find you forget what you came to shops to buy?* (CFQ) and *I forgot to return something I borrowed* (PMQ).

**Social desirability.** Levels of social desirability were measured using the 33-item Marlow-Crowne Social Desirability Scale (MCSDS; Crowne & Marlowe, 1960). Examples of these true/false items include *I am always courteous, even to people who are disagreeable* and *I have never deliberately said something that hurt someone's feelings*.

**EF tasks.** The EF tasks are based on the nine tasks presented in Friedman et al. (2008), adapted for adults in their twenties. They were scored in the same way reported by Friedman et al. (2015). These EF tasks are briefly described here, but more information on these specific tasks is provided by Friedman et al. (2008, 2015).

**Inhibition.** Prepotent response inhibition is the ability to deliberately control or inhibit automatic, prepotent, or dominant responses (Miyake et al., 2000). Tasks that assess this ability typically compare performance on trials that do not require overriding a prepotent response (e.g., color naming strings of asterisks) with performance on trials that require overriding a prepotent response (e.g., naming the color of the word *RED* printed in green). The three tasks used to measure prepotent response inhi-

bition were (a) the antisaccade task (adapted from Roberts, Hager, & Heron, 1994); (b) the Stroop task (Stroop, 1935); and (c) the stop signal task (Logan, 1994).

In the antisaccade task, participants saw cues (black squares) quickly flash on the left or right of the screen, and had to avoid the reflexive tendency to saccade to these cues. Instead, they had to immediately look to the opposite side of the screen in order to identify a digit that appeared very briefly before being masked. The dependent measure was accuracy of digit identification on 108 trials (three blocks of 36 trials with increasing difficulty). In the Stroop task, subjects named the color of strings of either asterisks (neutral blocked trials) or color words printed in different colors (e.g., *RED* printed in green; incongruent blocked trials). Thus, the incongruent trials required individuals to avoid their prepotent tendency to read words. The dependent measure was the difference in mean reaction time (RT) for correct trials for incongruent blocks (two blocks of 42 trials) and the neutral block (42 trials). In the stop signal task, participants saw green arrows on the screen and quickly responded (left or right) with the direction of the arrow. However, sometimes arrows became red shortly after they were displayed. In these trials participants were instructed to stop their responses. The dependent measure was the stop-signal RT, which is the estimated time at which the stopping process finishes (Logan, 1994).

**Shifting.** Set shifting is the ability to flexibly switch back and forth between multiple task sets or operations (Miyake et al., 2000). In these tasks, stimuli could be categorized on two dimensions (e.g., shapes on a colored background can be classified according to the shape or color), and participants completed blocks of trials in which they categorized each stimulus according to one of these dimensions, depending on a cue that appeared 350 ms before each stimulus and remained on the screen along with the

stimulus until the response. In all tasks, the dependent measure was the “switch cost:” the difference in response time between switch trials (e.g., judge color after having judged shape in the prior trial) and repeat trials (e.g., judge color after having just judged color on the previous trial). Trial types were mixed in two blocks of either 56 (color-shape) or 64 trials (number-letter, category switch), where half of the trials required a switch between subtasks. The three tasks used to measure set-shifting switching ability were (a) number-letter (adapted from Rogers & Monsell, 1995); (b) color-shape (adapted from Miyake, Emerson, Padilla, & Ahn, 2004); and (c) category-switch (adapted from Mayr & Kliegl, 2000).

In the number-letter task, participants saw a number-letter pair (e.g., E7) presented in one quadrant of a box and identified whether the number-letter pair included an odd or even number or a vowel or consonant, depending on whether the pair appeared in the top 2 quadrants (odd-even) or bottom 2 quadrants (vowel-consonant). In the color-shape task, participants identified whether a colored shape was either green or red or was a circle or triangle, depending on a cue letter (C or S). In the category-switch task, participants identified whether a word (e.g., lizard) described something living or nonliving or bigger or smaller than a soccer ball, depending on a cue symbol (heart or crossed arrows) that appeared above the word.

**Updating.** Working memory updating is the ability to monitor incoming information and, when relevant, add newly relevant items into, and remove no-longer relevant items from, working memory (Miyake et al., 2000). The three tasks used to measure updating ability were (a) the keep track task (adapted from Yntema, 1963); (b) the letter memory task (adapted from Morris & Jones, 1990); and (c) the spatial *n*-back task (Friedman et al., 2008).

In the keep track task, participants saw a list of 15–25 words drawn from multiple categories (e.g., animals, metals, or countries) and were instructed to remember only the most recent word from each of two to five prespecified categories. The dependent measure was the proportion of total words recalled across 16 trials. In the letter memory task, participants saw a series of letters (of unpredictable length: nine, 11, or 13 letters long) displayed for 3 s each. As each letter appeared, they rehearsed aloud the four most recent letters they had seen (including the current letter). The dependent measure was the proportion of sets correctly rehearsed (points given for rehearsing the correct letters, and only those letters, in the correct order) across all the letters presented in 12 trials (132 total sets rehearsed). In the spatial *n*-back task, participants saw an array of empty squares that were filled briefly one at a time. After each square flashed on the screen, participants respond yes or no with whether that specific spatial position was the same that was flashed two or three trials ago (i.e., two-back or three-back). The two- and three-back versions were presented in separate tasks separated by multiple other tasks to reduce interference. The dependent measure was the average of the *z*-scores of arcsined accuracy (yes and no responses) for the two-back and three-back tasks (six blocks of 24 trials each).

## Data Analysis

All phenotypic and genetic analyses were performed with Mplus (Version 7.2; Muthén & Muthén, 2010). Model fit was evaluated

with chi-square tests ( $\chi^2$ ), the root-mean-square error of approximation (RMSEA), and the Comparative Fit Index (CFI). With larger sample sizes, one can obtain a significant chi-square for overall model fit despite relatively trivial differences between predicted and observed covariances. Thus, we considered  $\chi^2$  values less than two times the degree of freedom, RMSEA values  $<.06$ , and CFI values  $>.95$ , as indicators of good fit (Byrne, 1989; Hu & Bentler, 1998). Parameters were determined significant using chi-squared difference tests ( $\chi^2_{diff}$ ). In all analyses, a full-information maximum likelihood approach was used to account for missing data. For phenotypic models, standard errors and chi-squares were adjusted for clustering (i.e., within families) by using the “type = complex” command, and chi-square difference tests were appropriately scaled (Satorra & Bentler, 2001) using scaling factors provided in the output.

Behavioral genetic analyses are based on the following assumptions: Additive genetic (A) influences on a trait are assumed to correlate at 1.0 in MZ twin pairs, because they share 100% of their segregating alleles identical by descent, whereas this correlation is fixed to 0.5 in DZ twins, who share an average of 50% of their segregating alleles identical by descent. Because both types of twins are reared together, their shared environmental influences (C; those environmental effects that lead twins to be correlated) are assumed to correlate at 1.0; nonshared environmental influences (E; environmental influences that lead twins to be uncorrelated) are set to not correlate, by definition. Within each pair, twins were assigned to Twin 1 versus Twin 2 according to the same random assignments used by Friedman et al. (2008).

The phenotypic and genetic models for the individual self-report questionnaires on procrastination and goal failures (Gustavson et al., 2014) and for the performance-based EF tasks (Friedman et al., 2015) are described in earlier work. Thus, the univariate models for these traits are not discussed further.<sup>4</sup> Additionally, because there was no evidence for significant shared environmental influences in either the self-report measures of procrastination and goal failures or the performance-based EF data (Friedman et al., 2015; Gustavson et al., 2014), we focus on additive genetic and non-shared environmental (AE) models here. ACE models (including shared environmental influences) were also tested, but all estimates of shared environmental influences were not statistically significant,  $\chi^2_{diff}(1) < 2.03$ ,  $p > .154$ . Consistent with previous research, these influences were almost uniformly estimated at 0, and, if not, explained less than 4% of the variance in any latent variable. Moreover, leaving out shared environmental influences from these models increases power to detect genetic correlations and aids in model convergence. Thus, only the AE models will be presented in the Results section.

There was no evidence for sex differences in models of either the self-report questionnaire data or the performance-based EF data (Friedman et al., 2015; Gustavson et al., 2014). Moreover, there was no interaction between sex and EF to predict procrastination at the phenotypic level,  $\chi^2_{diff}(1) < 2.66$ ,  $p > .102$ . Thus, we

<sup>4</sup> Approximately 100 additional subjects completed the measures of procrastination and goal failures compared with the models presented in Gustavson et al. (2014), but these additional subjects did not change the overall factor structure or pattern of genetic and environmental correlations.



did not test for sex differences at the genetic level or in any other analyses.

**Accounting for social desirability.** An important aspect of this study was to ensure that the estimates of the relationships between procrastination, goal failures, and EF were not affected by potential biases due to the nature of these self-report questionnaires. Consistent with some earlier research (Tangney et al., 2004), preliminary analysis revealed that social desirability scores (MCSDS) were moderately correlated with all of the self-report measures of procrastination and goal failures,  $r_s = .40-.49$ ,  $p < .001$ , explaining between 16% and 24% of the variation in any given questionnaire. Importantly (and as expected), social desirability was largely unrelated to performance-based measures of EF. Only three of nine tasks were significantly correlated with MCSDS,  $r = -.09-.08$ ,  $p > .012$ , but even these significant correlations explained less than 1% of the variation in any given EF task.

Because it is possible that some of the heritability estimates of procrastination, as well as the estimates of the shared variation between procrastination and goal failures, are due to social desirability,<sup>5</sup> we used residualized measures of all of the questionnaires of procrastination and goal failures in the subsequent analyses such that all variation shared with the MCSDS scale was removed from each self-report measure. With a few exceptions, these results did not differ substantially from the models where original (nonresidualized) measures of procrastination and goal failures were used. However, some important differences will be discussed below where relevant, and equivalent models for the main figures presented in the results (without residualized measures of social desirability) are provided in [Appendix A](#).

## Results

Descriptive statistics and twin correlations are summarized in [Table 1](#) (for phenotypic zero-order correlations between the procrastination, goal failure, and EF measures, see [Appendix B](#)). Additionally, phenotypic correlations between all of the latent variables used in these analyses are displayed in [Table 2](#) below the diagonal (the latent variable correlations that did not remove any effects of social desirability are presented above the diagonal). This basic measurement model fit the data well,  $\chi^2(63) = 94.87$ ,  $p = .006$ , RMSEA = .026, CFI = .985.

Table 2  
Latent Variable Correlations Among Procrastination, Goal Failures, and EF Components

	Proc	GF	cEF	Shift	Up
Procrastination	1	.76*	-.13*	.06	.04
Goal failures	.67*	1	-.27*	.04	.06
Common EF	-.15*	-.32*	1	—	—
Shifting-specific	.01	.00	—	1	—
Updating-specific	-.03	-.01	—	—	1

*Note.* Correlations between all latent variables are presented below the diagonal. These same correlations without removing social desirability from the measures of procrastination and goal failures are presented above the diagonal, in italics. EF = executive function; Proc = procrastination; GF = goal failures; cEF = common EF; Shift = shifting-specific; Up = updating-specific.

\*  $p < .05$ .

## Hypothesis 1: Procrastination and EF

The first hypothesis concerned the relationship between self-reported procrastination tendencies and the three performance-based EF abilities. Specifically, we tested the prediction that high procrastination is related to worse common EF ability. As shown in [Table 2](#), a phenotypic model with procrastination and the three EF components confirmed this prediction: There was a significant negative correlation between procrastination and common EF ability,  $r = -.15$ ,  $\chi^2_{diff}(1) = 6.31$ ,  $p = .012$ , suggesting that individuals who report more procrastination have lower common EF ability. Procrastination was not significantly correlated, at the phenotypic level, with either the shifting-specific,  $r = .01$ ,  $\chi^2_{diff}(1) = .04$ ,  $p = .847$ , or updating-specific,  $r = -.03$ ,  $\chi^2_{diff}(1) = .24$ ,  $p = .625$ , components of EF. This model also included goal failures, but there were no substantial differences in the estimates of these correlations in a model with only procrastination and the EF components.

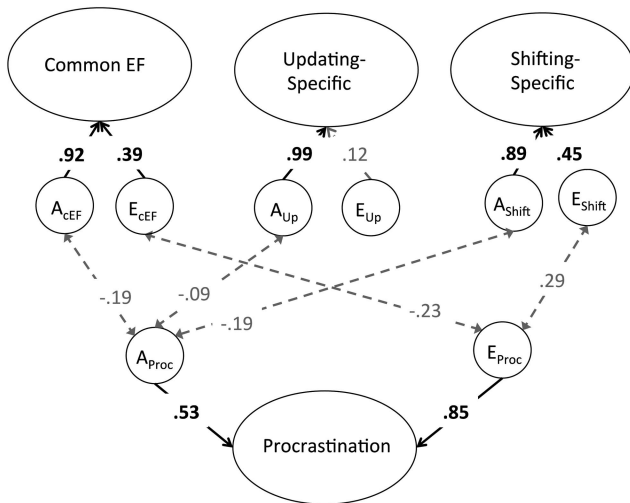
To understand the nature of this phenotypic correlation between procrastination and common EF, we fit a multivariate AE model for procrastination, common EF, shifting-specific, and updating-specific latent variables. In this model, variation in procrastination was broken down into genetic and nonshared environmental influences (i.e.,  $A_{Proc}$ ,  $E_{Proc}$ ), and these influences were allowed to correlate with the nonzero genetic and environmental components of the EF model (i.e.,  $A_{cEF}$ ,  $E_{cEF}$ ,  $A_{shift}$ ,  $E_{shift}$ ,  $A_{up}$ ). This model, displayed in [Figure 2](#), fit the data well,  $\chi^2(584) = 694.08$ ,  $p = .001$ , RMSEA = .031, CFI = .955.

In this model with procrastination and EF alone, we were not able to distinguish whether the phenotypic correlation between procrastination and common EF observed earlier was due to shared genetic or environmental influences. Both the genetic and environmental correlations between procrastination and common EF were nonsignificant when tested separately,  $\chi^2_{diff}(1) < 3.28$ ,  $ps > .070$ . However, these relationships were in the expected direction (more procrastination was related to worse common EF at both genetic and environmental levels), and both of these correlations could not be simultaneously removed from the model without a significant reduction in fit,  $\chi^2_{diff}(2) = 10.73$ ,  $p = .005$ . These results suggest that although procrastination is related to worse common EF ability, it was not possible to attribute definitively to common genetic or environmental influences (or both).

One other important result in this first model is that the heritability of procrastination was smaller than the original estimates reported in our earlier study (Gustavson et al., 2014). In the model shown in [Figure 2](#), procrastination had a heritability of 28%, meaning that 28% of the variation in procrastination is explained by genetic influences (percent of variation explained can be computed by squaring factor loadings on the latent genetic factor,  $A_{Proc}$ ; i.e.,  $.53^2 = .28$  [28%]). In the Gustavson et al. (2014) study and the analyses without residualized measures ([Figure A1](#) of [Appendix A](#)), this heritability estimate was about 46%. These results suggest that variation in social desirability contributed

<sup>5</sup> A univariate analysis of the MCSDS revealed that the heritability of this scale was 25%, whereas 18% of the variation was accounted for by shared environmental influences. The remaining variation (57%) in this scale, which includes measurement error, was accounted for by nonshared environmental influences.





**Figure 2.** Genetic model of the relationship between procrastination and the executive function (EF) factors (after removing social desirability from all individual measures of procrastination and goal failures). Significant genetic ( $A_{cEF}$ ,  $A_{Up}$ ,  $A_{Shift}$ ,  $A_{Proc}$ ) and nonshared environmental ( $E_{cEF}$ ,  $E_{Up}$ ,  $E_{Shift}$ ,  $E_{Proc}$ ) factor loadings, and the correlations between latent factors are indicated in bold and with solid black arrows when significant ( $p < .05$ ). Individual factor loadings on the latent variables for each trait are not shown, but were all statistically significant ( $p < .05$ ) and almost identical to those presented in Figure 1 (for EFs) and Figure 3 (for procrastination).

substantially to the prior heritability estimate of procrastination, but that this trait still has considerable genetic and nonshared environmental influences.

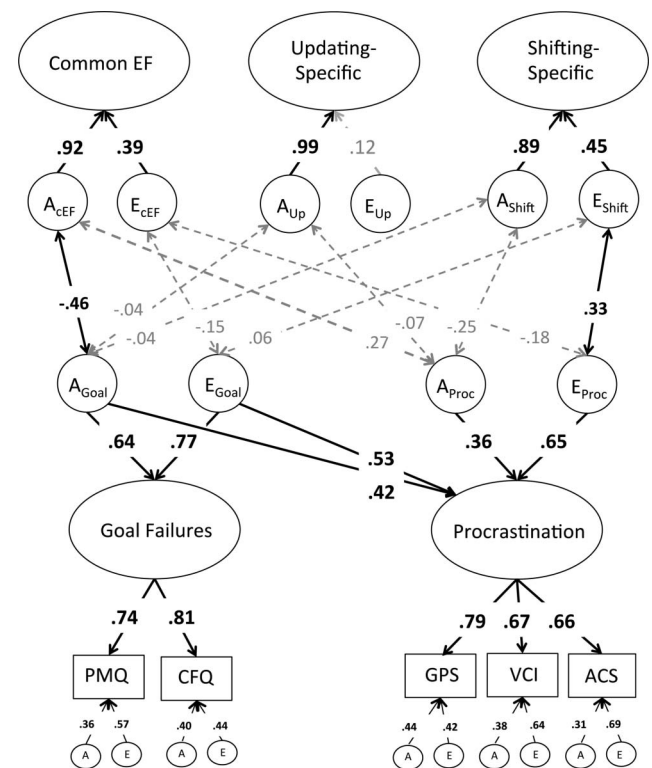
## Hypothesis 2: Procrastination, Goal Management, and EFs

The second main goal of this study was to test the hypothesis that the relationship between procrastination and common EF ability was due primarily to the overlap between procrastination and goal failures (rather than what is unique to procrastination). The latent variable correlations from the full phenotypic model, shown in Table 2, revealed that goal failures had a similar pattern of correlations with the EF components as procrastination, providing some support for this hypothesis. However, to more directly test the hypothesis that procrastination is linked to EF through aspects shared with goal failures, we fit a multivariate AE model of procrastination, goal failures, and EF abilities.

In this genetically informative Cholesky model, displayed in Figure 3, we first decomposed the variation in procrastination into (a) genetic and environmental components shared with goal failures ( $A_{Goal}$ ,  $E_{Goal}$ ); and (b) genetic and environmental components unique to procrastination ( $A_{Proc}$ ,  $E_{Proc}$ ). Next, we combined this model of procrastination and goal failures with the EF model used in the first analyses. As with the model of procrastination and EF alone, we only allowed the nonzero genetic and environmental components of the EF model (i.e.,  $A_{cEF}$ ,  $A_{Up}$ ,  $A_{Shift}$ ,  $E_{cEF}$ ,  $E_{Shift}$ ) to correlate with each of these Cholesky factors that decompose variation in procrastination ( $A_{Goal}$ ,  $E_{Goal}$ ,  $A_{Proc}$ ,  $E_{Proc}$ ). This model fit the data well,  $\chi^2(788) = 930.59$ ,  $p < .001$ , RMSEA = .031, CFI = .953.

**Procrastination and goal failures alone.** The relationship between procrastination and goal failures was similar to that reported by Gustavson et al. (2014). Although the total heritability estimates were smaller after removing social desirability, much of the heritability of procrastination ( $A_{Goal} + A_{Proc}$ ) was explained by the genetic influences common with goal failures ( $A_{Goal}$ ). This model revealed that 17% of the total variation in procrastination was explained by genetic influences also shared with goal failures ( $A_{Goal}$ ), accounting for over half of the overall heritability estimate. There were also nonshared environmental influences common to both traits that explained 28% of the total variation in procrastination ( $E_{Goal}$ ). Finally, there were substantial genetic influences unique to procrastination ( $A_{Proc}$ ) that explained 13% of the total variation, as well as substantial environmental influences also unique to procrastination ( $E_{Proc}$ ) explaining the remaining variation (42%). Therefore, even after removing social desirability from the self-report measures, both procrastination and goal failures still share considerable overlap at the genetic and environmental levels.

**Procrastination, goal failures, and EF.** The results of this final model provide strong support for the second hypothesis that



**Figure 3.** The relationship between procrastination, goal failures, and executive function (EF) factors (after removing social desirability from all individual measures of procrastination and goal failures). Significant ( $p < .05$ ) genetic (e.g.,  $A_{Goal}$ ,  $A_{Proc}$ ) and nonshared environmental (e.g.,  $E_{Goal}$ ,  $E_{Proc}$ ) factor loadings and correlations are indicated in bold and with solid black arrows. Individual factor loadings on the latent variables and residual genetic (A) and nonshared environmental (E) influences for procrastination and goal failures are also displayed here; those for EF factors are statistically significant ( $p < .05$ ) and are nearly identical to those presented in Figure 1.

the relationship between procrastination and EF ability was due to the component of procrastination shared with goal failures. Specifically, as shown in Figure 3, there was a significant correlation between the genetic factor of procrastination shared with goal failures ( $A_{\text{Goal}}$ ) and the genetic factor for common EF ability ( $A_{\text{cEF}}$ ),  $r_G = -.46$ ,  $\chi^2_{\text{diff}}(1) = 17.61$ ,  $p < .001$ , suggesting that many of the same genetic influences that support common EF ability are also related to procrastination and everyday goal-management failures. Furthermore, the genetic correlation reported here ( $r_G = -.46$ ) was considerably larger than in the model without removing social desirability from the self-report measures ( $r_G = -.26$ ; see Figure A2 of Appendix A), suggesting that removing the genetic variation due to social desirability was useful in revealing that procrastination, goal failures, and EF ability are more closely related than would have been observed without accounting for social desirability in self-report measures.

We were not able to observe this important genetic relationship in the initial analyses shown in Figure 2 because there was an opposing positive correlation between genetic influences unique to procrastination ( $A_{\text{Proc}}$ ) and those for common EF ( $A_{\text{cEF}}$ ),  $r_G = .27$ ,  $\chi^2_{\text{diff}}(1) = 2.08$ ,  $p = .149$ . This correlation was not significant, but was strong enough to suppress the significant negative relationship described between  $A_{\text{Goal}}$  and  $A_{\text{cEF}}$  observed here (the overall  $r_G = -.19$ ). The nonshared environmental correlations between the procrastination components and common EF in this model were not significant, both  $\chi^2_{\text{diff}}(1) < 1.58$ ,  $p > .210$ .

Finally, the model shown in Figure 3 revealed one other interesting relationship between procrastination and EF ability that was not predicted. Specifically, we observed a positive correlation between the nonshared environmental component of shifting-specific ability ( $E_{\text{Shift}}$ ) and the nonshared environmental component unique to procrastination ( $E_{\text{Proc}}$ ),  $r_E = .33$ ,  $\chi^2_{\text{diff}}(1) = 3.89$ ,  $p = .049$ . This finding suggests that there are environmental factors that contribute to both better shifting-specific ability and more procrastination, but that those environmental factors have little to do with everyday goal-management abilities,  $r_E = .06$ ,  $\chi^2_{\text{diff}}(1) = .16$ ,  $p = .689$ . It may be surprising that greater procrastination would be related to *better* shifting-specific ability, but this positive relationship is consistent with other data suggesting that shifting-specific ability correlates in the opposite direction as the common EF component for a range of behavioral measures (Herd et al., 2014; Miyake & Friedman, 2012), a point we will discuss in more detail in the Discussion section.

## Discussion

The current study tested two hypotheses about how self-reported measures of procrastination are related to performance-based measures of EF: (a) that the tendency to procrastinate is associated with poor common EF ability, and (b) that this association is largely due to the genetic aspect of procrastination that is shared with the inability to manage goals effectively. The first set of analyses revealed that at the latent variable level, procrastination was indeed negatively correlated with common EF ability ( $r = -.15$ ). Further analyses supported the second hypothesis that procrastination was related to common EF through its component shared with goal failures ( $A_{\text{Goal}}$ ). Moreover, this correlation was substantially due to genetic influences ( $r_G = -.46$ ) rather than nonshared environmental influences ( $r_E = -.15$ ). Taken together,

these results provide new evidence for the proposal that procrastination and EF ability are systematically linked, and that this relationship is driven primarily by genetic influences shared between procrastination, EFs, and everyday goal-management abilities.

These results are important for a number of reasons. First, this is the first study to quantify the relationship between procrastination and EF using performance-based measures of the latter. Second, we demonstrated these links between procrastination and EF at the level of latent variables and in a large sample. Third, by removing statistical variance associated with social desirability from self-report measures, these findings revealed that the relationships between procrastination, everyday goal-management failures, and EF abilities were not due primarily to self-report biases, such as social desirability.

Despite these strengths, however, we acknowledge that the study also has some limitations. First, the twin sample used in the current study was large enough to quantify the additive-genetic relationship between procrastination and EF, but was not large enough to detect whether other types of genetic influences (i.e., nonadditive genetic influences) also play a role in this relationship (Eaves, Heath, Neale, Hewitt, & Martin, 1998). Second, this research has focused on the goal-management abilities that underlie domain-general aspects of procrastination, but it is possible that domain-specific types of procrastination (e.g., academic procrastination) are related differentially to these general and specific EF abilities at the phenotypic and genetic levels. Third, because we did not administer intelligence or personality measures at the same time as the procrastination and EF measures analyzed in the current study, we cannot assess whether (and to what extent) the genetic and environmental relationships observed between procrastination and EF in the current study are mediated by other related constructs (e.g., general intelligence, conscientiousness).<sup>6</sup> Finally, the reported data are correlational and thus cannot speak to the causal direction of the relationships described here. It is reasonable to hypothesize that procrastination can be a result of poor common EF ability (rather than poor common EF ability resulting from procrastination), but these results cannot directly test such directional claims and are thus subject to alternative interpretations (e.g., involving other mediating variables). Despite these limitations, however, we believe that the current study makes novel and substantial contributions to our understanding of the goal-related cognitive abilities underlying individual differences in procrastination.

<sup>6</sup> Our longitudinal sample of twins completed the Wechsler Adult Intelligence Scale-III test approximately a year before the first wave of EF testing, which was conducted approximately 6 years earlier than the wave reported in this study. As reported by Friedman, Miyake, Robinson, and Hewitt (2011), age 16 full-scale IQ correlated .51, .49, and -.24 with the age 17 common EF, updating-specific, and shifting-specific latent variables, respectively. Thus, although it is possible that some of the relationship between common EF and procrastination is mediated by general intelligence (or vice versa), IQ explained only 25% of the variance in the age 17 common EF factor, so it is unlikely that our current EF measures (which are highly stable with this earlier assessment) are essentially the same as general intelligence measures.

## Theoretical Implications for the Current Research: Linking Procrastination and EF Theories

A primary goal of this study was to bring together two research domains that have rarely been studied together in the past, even though both procrastination and EFs have long been linked to self-regulation or self-control abilities. Although prior studies from our group have explored the latent structure of procrastination (Gustavson et al., 2014) and of EFs (Friedman et al., 2008, 2015; Miyake et al., 2000), the results of the current study go substantially beyond those earlier studies by rigorously specifying the nature of the relationships between procrastination and EFs. In this section, we will flesh out our theoretical interpretations of the procrastination/EF relationship as revealed in the current study, with respect to both the unity (common EF) and diversity (primarily shifting-specific ability) components of EFs.

**Common EF.** The current research supports the hypothesis that procrastination is related to the ability to perform well on all types of laboratory-based EF tasks (i.e., common EF ability). Individuals who reported higher levels of procrastination had worse common EF ability, and this relationship was largely due to a correlation between the genetic influences on common EF and the genetic component of procrastination shared with goal failures ( $A_{Goal}$ ). This genetic correlation was fairly small without removing variation due to social desirability ( $r_G = -.28$ ). However, creating residualized measures of procrastination that partialled out the effect of social desirability revealed that this genetic correlation was much stronger ( $r_G = -.46$ ), suggesting that there are substantial genetic influences that play a role in everyday procrastination and goal failures as well as during performance on short-term goal-directed EF tasks.

In contrast, we were not able to identify any environmental influences that were correlated between procrastination and common EF. Although there was some evidence for a significant nonshared environmental correlation between procrastination and common EF in the analyses without removing social desirability ( $r_E = -.33$ ; Figure A2 of Appendix A), these effects were not observed in our primary analyses (see Figure 3). Thus, there was little evidence that environment influences play a direct role in this relationship after controlling for social desirability.

Our previous work (Gustavson et al., 2014) has shown that individual differences in goal-management abilities account for much of the shared variation between procrastination and impulsivity, and that this shared variation is predominantly genetic. The current study extends these conclusions by revealing that these genetic influences also play a substantial role in the goal-related processes that underlie individual differences in the performance of laboratory-based EF tasks. Although it is still unclear how other proposed factors such as goal setting (Steel & Konig, 2006), goal focus (Krause & Freund, 2014), and effective discrimination between goals (Kuhl, 1994) fit in this picture, our finding that everyday goal-management and laboratory-based EF ability share a significant genetic overlap suggest that other goal-related abilities may also share some of the same genetic influences.

Based on what we now know about other cognitive and behavioral phenotypes, these shared genetic influences on procrastination, goal management, and EF abilities likely reflect the contribution of many hundreds or thousands of genetic polymorphisms, each accounting for only a fraction of a percent of the total

variance (e.g., Manolio et al., 2009). Thus, to detect specific genetic variants that might account for this relationship, much larger samples are needed. Despite such major challenges in identifying specific genes, knowing that the three key constructs examined in the current study—procrastination, goal management, and EF abilities—share the same genetic influences may be helpful in guiding future research on not only individual differences in procrastination but, more generally, individual differences in other aspects of self-regulation or self-control abilities, given that the process of self-regulation/self-control is inherently goal-driven.

Although our discussion of the relationship between procrastination, goal management, and EFs has primarily focused on genetic influences ( $A$  estimates), one important possibility is to consider is that the genetic influences described here may reflect the contribution of not only genetic factors, but also the interplay between genes and the environment (e.g., gene–environment correlations). For example, individuals with a genetic predisposition for procrastination may be more likely to seek out jobs or classes that allow them to procrastinate more. Similarly, individuals with genetic predispositions toward high EF may be more likely to seek out peers who have less distracting influences, thereby reducing procrastination. Although other types of gene–environment correlations may affect the interpretation, such “active” gene–environment correlations would, if true, be included in the estimates of the genetic influences (i.e.,  $A$  estimates) in multivariate models of the kind presented here (e.g., Figures 2 and 3), because, in examples like these, genetic influences (i.e., genetic predispositions) are the ultimate “cause” of these environmental influences (Plomin, DeFries, & Loehlin, 1977). Although it is challenging, future research that formally examines the extent to which the genetic influences on the relationship between procrastination and EF are attributable to gene–environment correlations would be helpful in better understanding the etiology of procrastination.

**Other EF components.** The main hypotheses of this study concerned the relationship between procrastination and common EF ability, but the results also shed some new light on how procrastination is related to other important EF abilities. First, there was no evidence that procrastination was related to updating-specific ability in any of the analyses. The nature of updating-specific ability is still not entirely understood, but this ability has been hypothesized to reflect individual differences in working memory gating and/or long-term memory retrieval (Miyake & Friedman, 2012) and is substantially related to intelligence (Friedman et al., 2008).

In contrast, there was a significant relationship between procrastination and shifting-specific ability. Nonshared environmental influences on shifting-specific ability correlated positively with nonshared environmental influences on procrastination (i.e., better shifting-specific ability is associated with more procrastination). Importantly, this relationship was due to the environmental influences specific to procrastination, rather than those environmental influences on procrastination that are shared with goal-management failures. This relationship was in the opposite direction as that between procrastination and common EF, but, interestingly, similar patterns of findings have been observed in other studies. For example, better shifting-specific ability is associated with lower IQ scores, more attention problems during childhood, and more externalizing behavior problems, although these prob-



lems are negatively correlated with the common EF factor (see Herd et al., 2014; Miyake & Friedman, 2012).

These results add to the growing understanding of the nature of shifting ability. Set shifting ability as a whole has been described as a tradeoff between stability and flexibility (Altamirano, Miyake, & Whitmer, 2010; Goschke, 2000; Herd et al., 2014; Miyake & Friedman, 2012). The ability to maintain the current task goal and use it to bias processing (common EF) allows one to focus on that particular task and thus enhances performance stability. Too much focus on that goal, however, makes it difficult for one to flexibly move away from that goal and then switch to a new goal (specific-specific), thus reducing performance flexibility. From this perspective, the results of the current study suggest that procrastinators seem to have difficulty with the stability aspect (i.e., worse common EF), but in some cases may be more capable of flexibly shifting between tasks than nonprocrastinators (i.e., shifting-specific) as a result of specific nonshared environmental factors.

It is currently not clear what specific environmental influences account for this relationship between procrastination and shifting-specific, but it is likely that they are a combination of multiple environmental factors (Turkheimer & Waldron, 2000), including those experienced throughout the lifetime (e.g., peer groups, experience with different teaching styles) as well as others that may be specific to the day or month of testing (e.g., hours of sleep, recent drug or alcohol use, current use of planners or schedules). However, because these relationships were observed at the level of latent variables, it is unlikely that these estimates of nonshared environmental influences reflect measurement error.<sup>7</sup>

**EF versus conscientiousness.** Before concluding our discussion of theoretical implications of the current study, we briefly consider to what extent the current results regarding the relationship between procrastination and EFs may reflect the effect of a personality trait, conscientiousness. Although it colloquially means being careful and meticulous about what one does, it is often defined in the scientific literature in a manner similar to self-control or EF. For example, Bogg and Roberts (2004) defined conscientiousness as “individual differences in the propensity to follow socially prescribed norms for impulse control, to be task- and goal-directed, to be planful, to delay gratification, and to follow norms and rules” (p. 887). In light of such a definition, it is not surprising that prior meta-analyses have shown that conscientiousness is a personality trait substantially related to procrastination (e.g., Steel, 2007; van Eerde, 2003). For example, according to Steel’s (2007) meta-analysis, the mean correlation between the two constructs is estimated to be  $-.62$  (95% confidence interval of  $[-.60, -.65]$ ). Moreover, the focus of the conception of conscientiousness on behavioral control and goal-directedness conceptually overlaps with EF (e.g., Bogg & Roberts, 2013; Duckworth & Kern, 2011). Thus, it is possible that conscientiousness can substantially account for the genetic and/or environmental relationships between EF and procrastination reported in this study.

Although we do not have data to directly address this issue, the evidence for the hypothesized connection between conscientiousness and EF is weak at best, at least when performance-based measures of EFs are used (Fleming, Heintzelman, & Bartholow, 2015; Hall & Fong, 2013). In fact, using virtually the same set of performance-based EF measures used in the current study, Fleming, Heintzelman, and Bartholow (2015) observed a weak positive association (.18) between conscientiousness and shifting-specific

ability at the level of latent variables, but no relation between conscientiousness and common EF. Thus, although we cannot rule out the possibility that the nonshared environmental correlation observed between shifting-specific ability ( $E_{\text{Shift}}$ ) and the portion of variation in procrastination not shared with goal failures ( $E_{\text{Proc}}$ ) may be accounted for in part by conscientiousness, it is unlikely that conscientiousness can provide a full explanation for the patterns of correlations reported in the current study (especially the genetic correlations involving common EF). If any, the effect of conscientiousness on procrastination would likely be independent of the effect of performance-based EFs, although there is some evidence that conscientiousness correlates more substantially with self-report measures of EFs (Rabin et al., 2011).

## Methodological and Practical Implications of the Current Research

**Methodological implications.** The current research also has a number of methodological implications for future research on procrastination and/or EF ability. First, because some previous reviews have noted that the correlations between performance-based and self-report measures of self-control abilities (including some EF measures) may not be high (Duckworth & Kern, 2011; Toplak, West, & Stanovich, 2013), it is important to perform these analyses using structural equation modeling at the level of latent variables. The phenotypic relationship between procrastination and common EF described here was small, even at the latent level ( $r = -.15$ ). As displayed in Appendix B, many of the correlations between individual measures of procrastination and the EF tasks were not significant, despite being significant at the latent variable level. Future research should focus on examining the relationship between procrastination and EFs at the level of latent variables to obtain good estimates of these relationships and avoid type II error.

Second, the findings of the current study also suggest that it may be important to consider how response bias factors, such as social desirability, may be involved in estimates of self-reported measures of procrastination and other related constructs. By and large, removing variation shared with social desirability from our self-report measures did not change the qualitative relationship between procrastination, everyday goal management, and EF ability. As noted earlier, however, the heritability of procrastination, for example, was substantially reduced (28% down from 46%), once the effect of social desirability had been removed from self-report measures. These potential biases may influence the relationship between procrastination and other traits depending on how each is measured. Moreover, in this sample, the estimates of the genetic relationship between procrastination and everyday goal management were overestimated without accounting for social desirability, but the genetic correlation between these traits and EF was underestimated without accounting for social desirability. Given these patterns, it may be beneficial to control for these effects in future studies on procrastination, especially when investigating

<sup>7</sup> Nonshared environmental variance and covariance could also reflect gene-environment interactions with nonshared environmental factors. That is, if the effects of environmental influences that are unique to a particular twin depend on that twin’s genetic makeup, then variance due to that interaction would be included in the E estimates. In contrast, interactions with shared environmental influences would be included in the A estimates (Purcell, 2002).

other traits, such as impulsivity and self-control (Tangney et al., 2004), that may also be perceived as socially undesirable.

More important, the multifaceted nature of the procrastination/EF relationships revealed in the current research suggest that it may be important to disentangle the relationships between procrastination and EFs at multiple levels. The unity/diversity model of EF described here distinguishes between multiple components of EF at work during performance on EF tasks (Miyake & Friedman, 2012), and our results suggest that procrastination is related to two of these latent EF abilities in opposing ways (i.e., worse common EF but better shifting-specific). In addition, some relationships observed at the genetic/environmental level of analysis (e.g., the positive environmental correlation between procrastination and shifting-specific ability) were not apparent by simply examining the data at the phenotypic level. Finally, it is also important to consider the multifaceted nature of procrastination, such as the aspect of procrastination that systematically overlaps with common EF and goal-management abilities and the aspect that is unique to procrastination. As these considerations illustrate, understanding how complex cognitive abilities like EFs are linked to individual differences in complex traits like procrastination requires well developed models and analyses that help disentangle the various underlying processes tapped by different facets of EF and procrastination.

More generally, the current study provides a useful basis for facilitating current and future attempts to specify nomological networks for procrastination. Although some systematic (often meta-analysis-based) attempts to derive such networks have been made before (e.g., Steel, 2007; van Eerde, 2003), the constructs included in such analyses have been limited mostly to personality and motivational variables (e.g., perfectionism, impulsivity, conscientiousness, self-efficacy). Because EFs and goal-management abilities are linked to various other behavioral and cognitive constructs (e.g., self-regulation/control, intelligence), examining procrastination from the perspective of these EF and goal-related variables in future research would likely help better situate procrastination in broader nomological networks of related constructs.

**Practical implications.** The conclusion that various goal-management abilities, such as those utilized by common EF and in everyday settings, underlie substantial variation in procrastination also paves the way for potential interventions for procrastination. For example, training subjects on how to set good goals may improve their ability to manage these goals and avoid procrastination, as also suggested by temporal motivation theory (Steel & Konig, 2006). Moreover, helping subjects retrieve their important long-term goals and use those goals to avoid getting sidetracked by short-term temptations (e.g., developing implementation intentions) might also be effective at reducing procrastination (Owens, Bowman, & Dill, 2008). Such goal-based interventions could not only have the potential of benefitting some individuals who suffer from procrastination habits but also contribute to further theoretical development in the field by enabling researchers to better establish the causal nature of the relationship between procrastination and goal-management ability.

## Conclusion

In this study, we used behavior–genetics methods to explore a theorized relationship between procrastination and EF and shed

new light on the cognitive mechanisms behind the tendency to irrationally delay action. This was the first study to show that procrastination is related to worse performance-based measures of common EF ability at the phenotypic and genetic levels, and that this genetic relationship was due to the overlap between procrastination and everyday goal-management failures. Moreover, though not part of the a priori hypotheses, the study also uncovered a significant negative environmental correlation between procrastination and shifting-specific EF ability. Furthermore, although all the self-report measures used in the current study were moderately correlated with a known self-report bias like social desirability, the relationships between procrastination and EF we observed were independent of the effects of social desirability. By rigorously specifying the procrastination/EF relationship at both phenotypic and genetic levels, the current study does not only shed new light on the etiology of individual differences in procrastination but also helps bring social/motivational theories of procrastination closer to cognitive theories of EFs.

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(Appendices follow)

Appendix A

The Relationship Between Procrastination and EF Without Accounting for Social Desirability

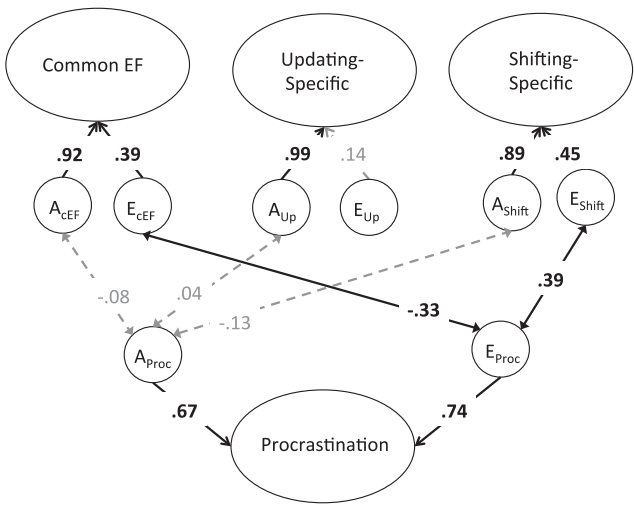


Figure A1. Genetic model of the relationship between procrastination and the executive function (EF) factors without removing social desirability from the self-report measures (cf. Figure 2). Significant ( $p < .05$ ) genetic ( $A_{\text{CEF}}$ ,  $A_{\text{Up}}$ ,  $A_{\text{Shift}}$ ,  $A_{\text{Proc}}$ ) and nonshared environmental ( $E_{\text{CEF}}$ ,  $E_{\text{Up}}$ ,  $E_{\text{Shift}}$ ,  $E_{\text{Proc}}$ ) factor loadings, and the correlations between these genetic and environmental factors are indicated in bold and with solid black arrows. Individual factor loadings on the latent variables for each trait are not shown, but were almost identical to those presented in Figure 1 (for EFs) and Figure 3 (for procrastination).

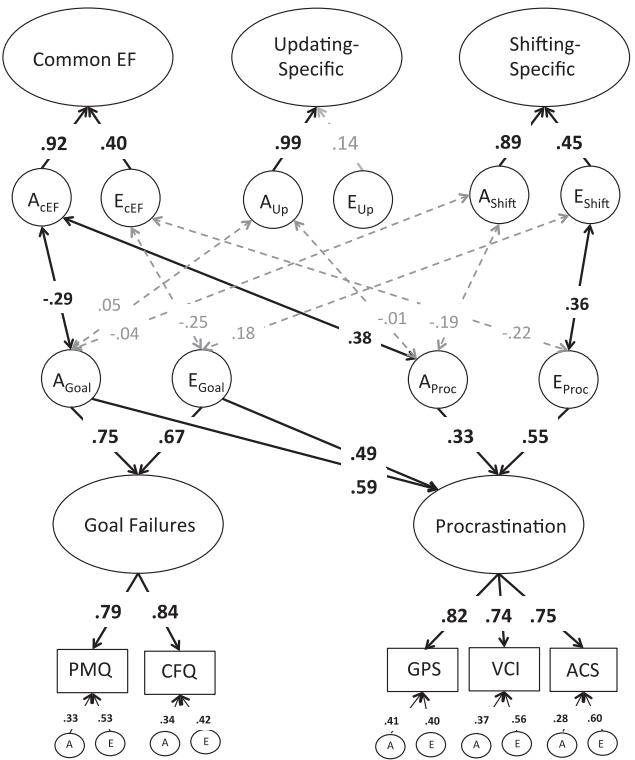


Figure A2. Genetic and environmental decompositions of procrastination, goal failures, and executive function (EF) factors without removing social desirability from the self-report measures (cf. Figure 3). Significant genetic (e.g.,  $A_{\text{Goal}}$ ,  $A_{\text{Proc}}$ ) and nonshared environmental (e.g.,  $E_{\text{Goal}}$ ,  $E_{\text{Proc}}$ ) factor loadings and correlations are indicated in bold and with solid black arrows when significant ( $p < .05$ ). Individual factor loadings on the latent variables and residual genetic (A) and nonshared environmental (E) influences for procrastination and goal failures are also displayed here, and those for EF abilities are nearly identical to those presented in Figure 1.

(Appendices continue)

## Appendix B

### Full Phenotypic Correlation Matrix

	GPS	VCI	ACS	PMQ	CFQ	Anti	Stroop	Stop	Num	Col	Cat	<i>n</i> -back	LM	KT
GPS	1													
VCI	.53*	1												
ACS	.51*	.46*	1											
PMQ	.43*	.31*	.29*	1										
CFQ	.44*	.35*	.34*	.59*	1									
Antisaccade	-.07	-.05	-.12*	-.15*	-.22*	1								
Stroop	-.03	-.02	-.07	-.07	-.09*	.24*	1							
Stop signal	-.10*	-.07	-.07	-.13*	-.11*	.32*	.13*	1						
Number-letter	-.06	-.07	-.04	-.10*	-.15*	.25*	.12*	.23*	1					
Color-shape	-.04	-.09*	-.09*	-.08*	-.12*	.39*	.11*	.29*	.51*	1				
Category switch	-.01	-.03	-.07	-.10*	-.12*	.37*	.05	.18*	.32*	.40*	1			
Spatial <i>n</i> -back	-.07	-.04	-.06	-.10*	-.14*	.26*	.09*	.18*	.11*	.09*	.04	1		
Letter memory	-.04	.01	-.06	-.05	-.01	.16*	.01	.17*	.14*	.11*	.09*	.43*	1	
Keep track	-.03	.03	.00	-.11*	-.10*	.36*	.15*	.29*	.22*	.20	.15*	.50*	.41*	1

*Note.* Phenotypic correlations between individual measures of procrastination, goal failures, and executive function. GPS = General Procrastination Scale; VCI = Volitional Components Inventory; ACS = Action Control Scale; PMQ = Prospective Memory Questionnaire; CFQ = Cognitive Failures Questionnaire; Anti = antisaccade; Stop = stop-signal; Num = number-letter; Col = color-shape; Cat = category-switch; *n*-back = spatial *n*-back; LM = letter memory; KT = keep track.

\*  $p < .05$ .

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