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## ARTICLE

# Procrastination and health: A longitudinal test of the roles of stress and health behaviours

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## Abstract

**Objectives:** Procrastination is a common form of self-regulation failure that a growing evidence base suggests can confer risk for poor health outcomes, especially when it becomes habitual. However, the proposed linkages of chronic procrastination to health outcomes have not been tested over time or accounted for the contributions of higher-order personality factors linked to both chronic procrastination and health-related outcomes. We addressed these issues by examining the role of chronic procrastination in health outcomes over time in which the hypothesized links of procrastination to health problems operate via stress and health behaviours.

**Design:** Three-wave longitudinal study with 1-month intervals.

**Methods:** Participants ( $N = 379$ ) completed measures of trait procrastination at Time 1, and measures of health behaviours, stress and health problems at each time point, in a lab setting.

**Results:** Procrastination and the health variables were inter-related in the expected directions across the three assessments. Chronic procrastination was positively associated with stress and negatively with health behaviours at each time point. Path analysis testing a cross-lagged longitudinal mediation model found an indirect relationship operating between procrastination and health problems via stress, after accounting for the contributions of conscientiousness and neuroticism.

**Conclusions:** This research extends previous work by demonstrating that the links between chronic procrastination and poor health are accounted for mainly by higher stress, after accounting for other key traits, and that these associations are robust over time. The findings are discussed in terms

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of the importance of addressing habitual self-regulation failure for improving health.

#### KEYWORDS

health behaviours, health problems, procrastination, self-regulation, stress

### Statement of Contribution

#### *What is already known on this subject?*

- Chronic procrastination is associated with poor health outcomes including higher stress, poor health behaviours and poor health.
- The procrastination–health model posits that higher stress and poor health behaviours are two routes through which chronic procrastination can confer risk for poor health.
- Previous research testing the procrastination–health model is limited and has used cross-sectional designs.

#### *What does this study add?*

- This study used a three-wave longitudinal design to test the procrastination–health model.
- Chronic procrastination was associated with health problems over time via stress but not health behaviours.
- Chronic self-regulation failure may confer short-term risk for poor health due to higher stress.

## INTRODUCTION

Defined as the voluntary delay of taking action on important, necessary and intended tasks despite knowing there will be negative consequences for this delay (Ferrari & Tice, 2000; Sirois & Pychyl, 2013), procrastination is a ubiquitous and prevalent form of self-regulatory failure that can be a chronic tendency for many individuals. Indeed, research estimates suggest that 50 per cent of students and 15–25 per cent of adults chronically procrastinate (Ferrari et al., 2007; Steel, 2007). In addition to having negative consequences for academic study (Hen & Goroshit, 2014) and work life (Beheshtifar et al., 2011), there is growing evidence that chronic or trait procrastination can also be detrimental to health and well-being. For example, research indicates that chronic procrastination is linked to higher stress, poor health behaviours, poor sleep and a greater number of physical illnesses and symptoms (Flett et al., 2012; Johansson et al., 2023; Kelly & Walton, 2021; Li et al., 2020).

Theoretical accounts of why chronic procrastination may confer vulnerability for poor health are consistent with classic models of personality and health (e.g. Smith, 2006; Suls & Rittenhouse, 1990), and implicate heightened stress and poor practice of health-promoting behaviours as two key explanatory routes (Sirois et al., 2003). However, evidence supporting the procrastination–health model (Sirois et al., 2003), the first model to explicate the links between chronic procrastination and physical health, is mainly cross-sectional (e.g. Sirois, 2007; Sirois et al., 2003). This is problematic not only with respect to making inferences regarding the directionality of the links between trait procrastination and health outcomes but also because cross-sectional designs provide only a snapshot that cannot account for any ongoing effects of the proposed relationships. Research on the procrastination–health model has rarely accounted for the contributions of the higher-order personality factors linked to both procrastination and health. In the current study, we aimed to address these important issues and provide a temporal test of the procrastination–health model to better understand the pathways linking this chronic form of self-regulation failure to poor health.

## Chronic procrastination and health

The relationship between procrastination and health was first noted in a longitudinal study by Tice and Baumeister (1997) in which students who habitually procrastinated reported lower stress and fewer health problems at the beginning of the term relative to non-procrastinators. However, by the end of the term, they reported higher stress and more health problems. Although the reasons for the links between procrastination and health were not empirically tested, it was speculated that increased stress associated with chronic procrastination may be one possible explanation for the poor health of procrastinators. Building on this initial study, the procrastination–health model (Sirois, 2007; Sirois et al., 2003) offered a theoretically driven answer to the question of why chronic procrastination may be linked to a greater number of health issues.

Consistent with extant models linking personality to health in general (Friedman, 2000; Sergerstrom, 2000; Suls & Rittenhouse, 1990), the procrastination–health model (Sirois et al., 2003) proposed that procrastination confers risk for poor health through both a stress-related route and a behavioural route. The *stress-related* route refers to the health risks posed by the activation of physiological systems involved in the stress response and their role in the development of physical health issues both in the short and long term. For example, activation of the stress system creates increased risk of infections and autonomic nervous system arousal, which can elevate heart rate, increase muscle tension and disrupt digestive functioning and sleep (Taylor et al., 2020). The *behavioural* route involves poor health behaviours including avoidance of health-promoting behaviours, such as healthy eating and regular exercise. Although lack of exercise and eating unhealthy foods may have minor immediate effects on health, this pattern of poor health-promoting behaviours is known to increase risk for disease, especially among those with pre-existing risk factors (World Health Organization, 2015).

The stress-related route in the procrastination–health model captures the contribution of chronic procrastination for generating unnecessary stress via behavioural and cognitive pathways. An enduring tendency to put off important and intended tasks across a variety of life domains can generate unnecessary stress as the procrastinator has to deal with the personal (Solomon & Rothblum, 1984) and social (Ferrari et al., 1999; Giguère et al., 2016) consequences of delaying tasks. Theory and research also indicate that chronic procrastination is associated with a tendency towards *stressogenic* thoughts (Sirois, 2016), and negative, harsh self-evaluations (Flett et al., 1995; McCown et al., 2012) that can maintain stress (Flett et al., 2012; Sirois, 2014).

A general tendency to procrastinate also includes unnecessarily delaying the practice of important health-promoting behaviours. For example, in one study of adults in Israel, procrastinating on health-promoting behaviours was the most common life domain in which people procrastinated, with 40 per cent reporting doing so (Hen & Goroshit, 2018). Changing health behaviours can be challenging for many individuals, as it often requires breaking unhealthy habits by drawing on more conscious and effortful self-regulation resources to resist temptations and monitor goals in order to bridge the intention–behaviour gap (Allom et al., 2013; Sheeran & Webb, 2016). However, for those with chronic self-regulation difficulties, engaging in and maintaining a practice of healthy behaviours can be especially challenging (Sirois & Giguère, 2018).

## Evidence for the procrastination–health model

Research over the past two-plus decades has found evidence for the proposed links of chronic procrastination with stress, health behaviours and physical health. Trait procrastination is associated with higher levels of stress across both student (Flett et al., 2012; Jackson et al., 2000; Johansson et al., 2023; Sirois et al., 2003; Sirois & Tosti, 2012; Stead et al., 2010; Tice & Baumeister, 1997) and community adult samples (Sirois, 2007, 2015; Sirois & Kitner, 2015). Research has also found that chronic procrastination is associated with poor health behaviours in the form of less practice and weaker intentions to engage in health-promoting behaviours, such as healthy eating, physical activity and healthy sleep behaviours (Kelly

& Walton, 2021; Kroese et al., 2014; Li et al., 2020; Sirois, 2004, 2007, 2015; Sirois et al., 2003), as well as greater engagement in unhealthy behaviours such as tobacco, cannabis and alcohol use (Johansson et al., 2023). Trait procrastination has also been linked to a greater number of physical health problems (Sirois et al., 2003) and physical symptoms (Johansson et al., 2023), poor self-rated health (Sirois, 2007) and poor cardiovascular health (Sirois, 2015).

Previous tests of the mediating pathways of the procrastination–health model have found some support for both routes. In a sample of undergraduate students, procrastination was associated with a greater number of self-reported health problems; however, only stress and medical visits, but not health behaviours, mediated the relationship between procrastination and health problems in separate path models (Sirois et al., 2003). These findings were partially replicated in a sample of community adults, with both stress and health behaviours explaining the link between procrastination and illness in nested models, but only stress in the full model (Sirois, 2007).

Although this previous work provides some support for the procrastination–health model, several methodological issues need to be considered. First, both studies were cross-sectional. This limits the extent to which causal inferences can be made regarding the temporal precedence of trait procrastination in relation to health. A longitudinal test of the procrastination–health model is, therefore, essential to provide more robust support for the suggested direction of relationships. Second, the cross-sectional designs of these studies mean it is unclear whether the findings with respect to the health behaviour pathway that was significant in the community sample but not in the student sample are an artefact of the study design or a reflection of actual differences between students and adults in their vulnerability to the detrimental short-term effects of procrastination on health behaviours. Examining these relationships prospectively over several assessments would help address this issue. Lastly, the robust, non-trivial associations of trait procrastination with conscientiousness and neuroticism (Van Eerde, 2003), two higher-order traits known to predict health outcomes (Bogg & Roberts, 2013; Hampson et al., 2016), was not accounted for in previous tests of the procrastination–health model. Assessing the contributions of conscientiousness and neuroticism when testing the procrastination–health model is, therefore, crucial for improving our understanding of the implications of chronic procrastination for health.

## The current study

To address the limitations of previous research and obtain a temporal view of the procrastination–health model, we tested whether chronic procrastination longitudinally predicted stress, health behaviours and health problems using data collected with three repeated measures of the health-related variables approximately 1 month apart. Given that stress and health behaviours are known to covary, with higher stress linked to less practice of health-promoting behaviours (Rod et al., 2009; Steptoe et al., 1998), we hypothesized that stress and health-promoting behaviours would be negatively associated across the three time points. Consistent with previous investigations (Sirois, 2007; Sirois et al., 2003), we also hypothesized that stress would be positively related to health problems, whereas health-promoting behaviours would be negatively related to health problems at each time point.

Having tested the stability of the above relationships, we examined the total, direct and indirect effects of trait procrastination on health-related outcomes over time, with the indirect effects posited as operating via stress and health behaviours. Specifically, we hypothesized that: 1) trait procrastination would predict higher stress and less frequent health behaviours at each time point and 2) that stress and (less frequent) health behaviours would predict subsequent health problems. Given 1) and 2), we expected that stress and health behaviours would mediate the link between procrastination and health problems and that stress would be the dominant mediational pathway. We also controlled for both conscientiousness and neuroticism in the analyses with the expectation that the above hypotheses would hold after accounting for their contributions.

## METHODS

### Participants and procedure

Participants were recruited from the Carleton University Psychology Department participant pool which included undergraduate students from departments across the university. The first survey (T1) was administered via paper during the winter at the beginning of the second academic term in 2002, the second (T2) approximately 1 month later and the third (T3) during the last month of the second term. All T1 participants agreed to be contacted for the two follow-up studies. Participants received course credits for their participation along with a chance to enter a draw to win one of three small cash prizes. The study hypotheses were not pre-registered, but were based on extant theory, as the data were collected prior to pre-registration becoming routine practice. No power analysis was conducted *a priori*; however, the aim was to recruit as many students as possible initially as it was expected that there would be high attrition in the two subsequent administrations.

Of the 407 participants recruited to complete the initial survey package, 401 returned at least partially completed surveys at the second time point and 379 at the third time point. A total of 328 participants had complete responses on all study variables at all three time points.

### Measures

Measures of stress, health behaviours and health problems were included in the survey at all three time points. A measure of trait procrastination and the Big Five personality factors was included only at Time 1. Other measures included but not analysed in the current study are listed in a supplemental file available here: [https://osf.io/nsvmd/?view\\_only=eab0a1d97aef4637842a66339a8f8291](https://osf.io/nsvmd/?view_only=eab0a1d97aef4637842a66339a8f8291)

### Chronic/trait procrastination

Lay's General Procrastination scale (GPS; Lay, 1986) assessed stable global tendencies towards procrastination across a variety of tasks. This 20-item scale, consisting of 10 positively worded and 10 negatively worded items, has been used previously to assess the relation of procrastination to health-related behaviours and outcomes (Sirois, 2004, 2007). Items such as 'In preparing for some deadlines, I often waste time by doing other things' and 'I generally delay before starting work I have to do' are scored on a 5-point Likert-type response scale ranging from 1 (*false of me*) to 5 (*true of me*). Negatively worded items were reverse scored before summing all items into a single score with high values indicating a higher tendency to procrastinate. The GPS has demonstrated stability over a 10-year period (Steel, 2007), and good internal consistency in both previous investigations ( $\alpha = .082$ ; Lay, 1986) and the current study ( $\alpha = .88$ ).

### Stress

The severity of common daily stressors occurring within the past month was assessed with an abbreviated version of the Hassles Scale (Kanner et al., 1981). We used the abbreviated 70-item version by Lu (1991), which is free from items related to psychological and somatic symptoms, and further removed the six open-ended items. We also merged items with similar content (e.g. 'Not enough money for clothing and housing?'), and removed an item related to not having enough money for health care as it was not relevant to the current sample. This resulted in a 60-item abbreviated version of the scale. For each of the three assessments (T1, T2 and T3), participants indicated which of the 60 listed hassles (e.g. too many things to do) occurred within the previous month, and then rated the severity of these hassles on a scale ranging

from 1 (*Somewhat severe*) to 3 (*Extremely severe*). If a hassle was not experienced, it was recorded as '0'. Cronbach's alphas for the current study ranged from  $.905 < \alpha < .931$  for Time 1 to Time 3.

## Health behaviours

The Wellness Behaviour Inventory (WBI; Sirois, 2001) is a 12-item, theoretically derived measure of how often people engage in common health-promoting behaviours organized across four conceptual categories: healthy eating, regular exercise, sleep behaviour and stress management. Theory and research indicate that these behaviours are conceptually clustered according to their behavioural consequences (Flay et al., 2009; Lippke et al., 2012; Vickers et al., 1990), and therefore provide a reasonable index of key health-promoting behaviours. The WBI mean is based on 10 of the 12 items and excludes two filler items related to vitamin and supplement use. Items such as 'I exercise for 20 continuous minutes or more, to the point of perspiration' and 'I eat healthy, well-balanced meals' are rated on a 5-point scale with possible responses ranging from 1 (*less than once a week or never*) to 5 (*every day of the week*). After reverse keying two items, a mean of all items is calculated with higher scores indicating more frequent performance of health-promoting behaviours. The timeframe for Times 2 and 3 was 'since the previous survey administration', and for the Time 1 administration, participants reported how often they practised the given health behaviours in general over the preceding 3-month period. The WBI has demonstrated good test-retest reliability over a 2-week period ( $r_{TT2} = .89$ ), and sensitivity to change among adults intending to change their health behaviours over a 6-month period (Sirois et al., [in preparation](#)). A psychometric meta-analysis of the coefficient alpha for the WBI across diverse samples found an overall average alpha of  $.69$  ( $k = 54$ ,  $N = 14,517$ ). The internal consistency of the WBI in the current study was acceptable, ranging from  $.715 < \alpha < .757$  across Time 1 to Time 3.

## Acute health problems

The number and type of acute health problems were assessed using the Brief Health History questionnaire (Sirois & Gick, 2002), which includes 13 acute physical health problems (e.g. colds, headaches and digestive problems) plus an 'other' category with a space to list any other health problems not listed. Participants report whether they experienced any of the listed health problems within a particular timeframe. For the Time 1 administration, the timeframe was the last 6 months, and for Times 2 and 3, the timeframe was the previous month. The sum of health problem scores formed the acute health problems variable at each time point.

## Control variables

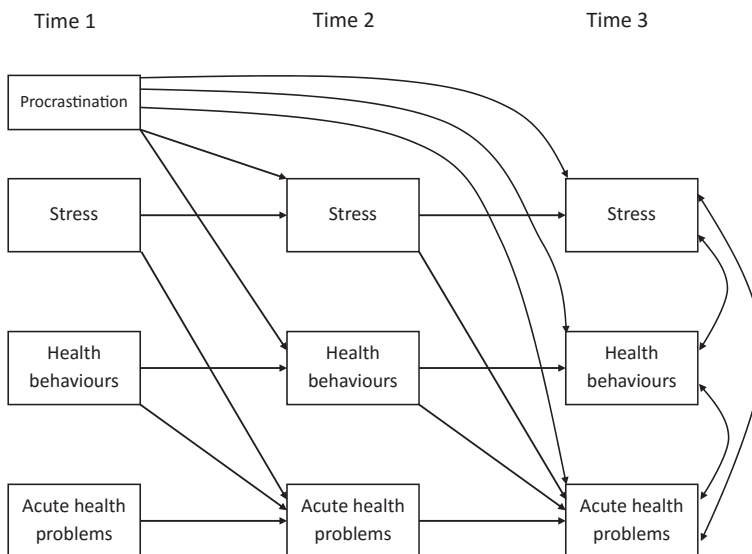
In addition to the measures described above, at Time 1, we collected data on the potentially confounding variables of sex (coded 0 = Male, 1 = Female) and age (in years), as well as the five factors of personality. Participant age and sex were included as there is some evidence that chronic procrastination scores are negatively related to age and are higher among younger males (Beutel et al., 2016; Ferrari & Díaz-Morales, 2007).

The 44-item Big Five Factor Inventory (BFFI; John & Srivastava, 1999) assessed the Big Five personality factors: openness, agreeableness, neuroticism, extroversion and conscientiousness. For the current study, we were only interested in the effects of conscientiousness and neuroticism, as these are the factors most strongly related to trait procrastination (Van Eerde, 2003). A list of characteristics is presented after the statement 'I see myself as someone who ...' and respondents rate how much they agree with each characteristic on a 5-point Likert scale ranging from 1 (*Disagree strongly*) to 5 (*Agree strongly*). Higher scores reflect greater identification with that particular personality factor. The BFFI has demonstrated

good internal consistency, with Cronbach's alpha coefficients ranging from .81 for conscientiousness to .88 for extraversion (John & Srivastava, 1999). In the current study, both the Conscientiousness and Neuroticism subscales demonstrated good internal consistency when collected at Time 1 (alpha = .803 and .820, respectively).

## Statistical analyses

To test our hypothesized model, we used a cross-lagged path analysis mediation model (see Figure 1), similar to that outlined by Cole and Maxwell (2003), but with our predictor variable (procrastination) collected only at Time 1 given its trait status. While the optimal analytical procedure would have been to have first run a confirmatory factor analysis (CFA) testing the measurement model for all our scales across all time points, and then 'extend' this to a cross-lagged structural equation model (SEM) to test our hypotheses using latent variables, the very large number of items across all scales and time points (288) and hence the very large number of parameters to be estimated in such a CFA or SEM (approximately 950, depending on fixings) were incompatible with our sample size. Hence, having first checked the internal consistency reliabilities as described above, we calculated scale means (i.e. composite) scores for each measure at each time point it was collected and tested our hypothesized cross-lagged model using these observed variables. In addition to the hypothesized paths displayed in Figure 1, our control variables were regressed upon the mediators (stress and health behaviours) and outcome (health problems) at each time point, and these were also correlated with procrastination. Starting with an unrestricted model, we applied a series of fixings to test the stability of relationships over time; specifically, first fixing within-variable autoregressive paths equal across time for stress, health behaviours and health problems, then fixing the two mediator-to-outcome paths (i.e. stress and health behaviours to health problems) equal across time. If model fit was not significantly weakened, these fixings were retained. We then calculated the indirect effects of procrastination on health problems via both mediators using bootstrapped confidence intervals to assess their statistical significance (Hayes, 2013).



**FIGURE 1** Cross-lagged mediation path analysis model testing the relationships of chronic procrastination to health through stress and health behaviours over time. Time 1 variables were correlated with control variables (age, sex, conscientiousness and neuroticism). Time 2 and Time 3 mediator and outcome variables were regressed upon control variables, but these paths are omitted from the diagram above for presentational clarity.



As a supplementary analysis to probe the causal direction of the stress and health behaviours relationship with health problems, we then added reverse causal paths from health problems to both stress and health behaviours at the subsequent time point (i.e. from T1 health problems to T2 stress and health behaviours, and from T2 health problems to T3 stress and health behaviours). We tested the difference between the health problems to stress and the stress to health problems paths, and likewise for health problems to health behaviours versus health behaviours to health problems.

Analyses were run using Mplus software v7.4 using full-information maximum-likelihood estimation (FIML) to fit each model. This gave an analysis sample of 379 cases who had responded to all the exogenous variables and at least one outcome – however, as a robustness check, we also repeated the analysis using maximum-likelihood estimation on a listwise deleted sample (328 cases who had completed all study variables at all time points). Chi-square difference tests were used for model comparison; bootstrapped confidence intervals were applied to assess indirect effects, with 10,000 bootstrap replications being used (Hayes, 2013). Exact *p* values are reported below, along with confidence intervals and effect sizes. Two-tailed tests were applied throughout. Hypotheses were not pre-registered as data collection occurred prior to when pre-registration of research hypotheses was possible. Data files and all data analysis scripts are available via this link: [https://osf.io/nsvmd/?view\\_only=eab0a1d97aef4637842a66339a8f8291](https://osf.io/nsvmd/?view_only=eab0a1d97aef4637842a66339a8f8291)

## RESULTS

### Preliminary analyses

Of our analysis sample of 379 participants, the majority were female (67%) and in their first year of study (81%). They ranged in age from 17 to 56 (median age = 19, mean age = 20.5; SD = 4.2), and the majority reported their ethnicity as Caucasian (77%). Table 1 presents the descriptive statistics and inter-correlations among trait procrastination and the study variables for these participants across the three time points. When checking assumptions with respect to fitting our path analysis model, outcomes and mediators had approximately symmetrical unimodal distributions at each time point, and there was no evidence of non-linear relationships among predictors, mediator and outcomes. There was, likewise, no evidence of multicollinearity among our predictor and mediator variables, with none of the correlations between these distinct measures at the same time point exceeding .5 (see Table 1). The mean GPS score ( $M = 2.76$ ,  $S.D. = .65$ ) was comparable to that reported in other research with undergraduate students (e.g.  $M = 2.81$ ,  $S.D. = .62$ ; Blunt & Pychyl, 2000).

### Hypothesis testing

The hypothesized cross-lagged mediation model (Figure 1) with all parameters free to vary overtime gave a satisfactory fit to the data (model chi-square = 53.802 on 18df, RMSEA = .072, CFI = .982 and SRMR = .039). This was not significantly weakened by first fixing autoregressive paths for each of stress, health behaviours and health problems to be equal across time (i.e. for each of these variables the T1 to T2 path was equal to the T2 to T3 path; model chi-square = 59.132 on 21 df, change in chi-square compared to the free model = 5.330 on 3 df,  $p = .149$ , RMSEA = .069, CFI = .981 and SRMR = .040).

Furthermore, fixing the paths from each mediator to the outcome to be equal across time (i.e. the T1 stress to T2 health problems path was equal to the T2 stress to T3 health problems path; and likewise for the respective health behaviours to health problems paths) did not significantly weaken model fit (model chi-square = 62.259 on 23 df, change in chi-square compared to the model with autoregressive paths only fixed = 3.223 on 3 df,  $p = .200$ , RMSEA = .067, CFI = .980 and SRMR = .041), providing evidence for the stationarity of these relationships. The path coefficients from this model are given in Table 2 and were used with respect to testing our hypotheses.

**TABLE 1** Descriptive statistics and intercorrelations among trait procrastination and the study variables across the three time points (343 < N < 379).

	<i>N</i>	<i>Mean</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10	11	12	13
1 Gender (1 = Female, 0 = Male)	379	.673	.469	---												
2 Age (years)	379	20.549	4.241	-.011	---											
3 Conscientiousness	379	3.555	.650	.148	.109	---										
4 Neuroticism	379	2.958	.792	.227	-.011	-.094	---									
5 Trait procrastination	379	2.763	.647	-.099	-.078	-.572	.120	---								
6 Stress – time 1	379	.575	.336	.020	.006	-.148	.404	.274	---							
7 Health behaviours – time 1	379	3.164	.688	-.114	.082	.334	-.170	-.341	-.227	---						
8 Acute health problems – time 1	379	3.604	1.756	.118	.008	-.214	.157	.203	.277	-.256	---					
9 Stress – time 2	371	.666	.351	.095	-.011	-.189	.437	.313	.671	-.297	.377	---				
10 Health behaviours – time 2	378	3.151	.669	-.073	.049	.345	-.229	-.300	-.241	.754	-.240	-.306	---			
11 Acute health problems – time 2	373	2.552	1.697	.115	.003	-.293	.177	.225	.305	-.342	.565	.426	-.317	---		
12 Stress – time 3	356	.619	.361	.141	-.012	-.180	.434	.277	.693	-.288	.360	.838	-.300	.404	---	
13 Health behaviours – time 3	354	3.163	.701	-.039	.016	.289	-.165	-.328	-.211	.759	-.216	-.303	.815	-.308	-.329	---
14 Acute health problems – time 3	343	2.300	1.493	.182	.009	-.154	.200	.233	.352	-.345	.510	.445	-.279	.681	.453	-.307

TABLE 2 Path coefficients from the cross-lagged mediation model linking trait procrastination to health.

Outcome	Health behaviours – time 2								Health behaviours – time 3				Acute health problems – time 3					
	Stress – time 2		Acute health problems – time 2				Stress – time 3				Direct effect		Indirect effect via stress, t2		Indirect effect via health behaviours, t2		Total effect	
	<i>B</i>	95%CI	<i>B</i>	95%CI	<i>B</i>	95%CI	<i>B</i>	95%CI	<i>B</i>	95%CI	<i>B</i>	95%CI	ind' eff <sup>a</sup>	95%CI†	ind' eff <sup>a</sup>	95%CI <sup>a</sup>	<i>B</i>	95%CI
Gender (1 = Female, 0 = Male)	.046	-.011, .102	.016	-.081, .113	.254	-.046, .555	.058*	.014, .101	.054	-.032, .141	.267*	.020, .513	--	--	--	--	--	--
Age (years)	.000	-.006, .006	-.003	-.013, .007	.009	-.022, .041	.000	-.005, .004	-.006	-.015, .003	.002	-.023, .027	--	--	--	--	--	--
Conscientiousness	-.013	-.061, .034	.120*	.037, .203	-.415**	-.634, -.195	-.019	-.056, .018	-.074	-.149, .000	.291*	.080, .501	--	--	--	--	--	--
Neuroticism	.072**	.037, .106	-.091*	-.148, -.034	.023	-.159, .205	.020	-.008, .049	.019	-.032, .070	-.030	-.182, .122	--	--	--	--	--	--
Trait procrastination	.066*	.017, .116	.008	-.076, .092	--	--	.000	-.039, .039	-.092*	-.168, -.016	.231*	.012, .450	.041*	.004, .078	-.002	-.019, .015	.270*	.050, .290
Stress – time 1	.631**	.572, .690	--	--	.615**	.308, .922	.283**	.210, .356	--	--	--	--	--	--	--	--	--	--
Health behaviours – time 1	--	--	.654**	.598, .709	-.203*	-.343, -.063	--	--	.294**	.220, .367	--	--	--	--	--	--	--	--
Acute health problems – time 1	--	--	--	--	.453**	.393, .513	--	--	--	--	.118*	.046, .190	--	--	--	--	--	--
Stress – time 2	--	--	--	--	--	--	.631**	.572, .690	--	--	.615**	.308, .922	--	--	--	--	--	--
Health behaviours – time 2	--	--	--	--	--	--	--	--	.654**	.598, .709	-.203*	-.343, -.063	--	--	--	--	--	--
Acute health problems – time 2	--	--	--	--	--	--	--	--	--	--	.453**	.393, .513	--	--	--	--	--	--
Total variance explained in outcome by all antecedents	52.3%		57.0%		38.7%		73.7%		71.7%		50.7%							

*N* = 379, ind' eff' = indirect effect.

<sup>a</sup>Bias-corrected bootstrapped 95% CI.

\* *p* < .05 and \*\* *p* < .0005.

In support of our hypotheses regarding the linkages among the health variables, statistically significant effects were found between trait procrastination and stress ( $B = .066$ , 95% CI = .017, .116,  $p = .009$ , with trait procrastination (which was collected at T1) explaining 1.4% unique variance in T2 stress), between stress and health problems ( $B = .615$ , 95% CI = .308, .922,  $p < .0005$ , with T2 stress explaining 2.6% unique variance in T3 health problems) and between health behaviours and health problems ( $B = -.203$ , 95% CI =  $-.348$ ,  $-.063$ ,  $p = .004$ , with T2 health behaviours explaining 1.0% unique variance in T3 health problems).

The relationship between procrastination and health behaviours at T2 was not statistically significant ( $B = .008$ ,  $p = .847$ , with trait procrastination explaining just .1% unique variance in T2 health behaviours) – hence, unsurprisingly given this, there was no evidence that the indirect effect of procrastination on health problems via health behaviours was non-zero (indirect effect =  $-.002$ , bootstrapped 95% CI =  $-.019$ ,  $.015$ ). However, the indirect effect of procrastination on T3 health problems via T2 stress was supported (indirect effect =  $.041$ , bootstrapped 95% CI =  $.004$ ,  $.078$ ), and comprised 15% of the total effect of procrastination on health problems ( $B = .270$ , bootstrapped 95% CI =  $.050$ ,  $.490$ ).

Adding reverse causal paths from health problems to stress and to health behaviours significantly improved model fit (model chi-square = 43.793 on 21 df, change in chi-square = 18.466 on 2 df,  $p < .0005$ , CFI = .988, RMSEA = .054, SRMR = .027). However, of these paths, the health problems to health behaviours path were non-significant ( $B = -.013$ , 95%CI =  $-.032$ ,  $.005$ ,  $p = .152$ , with T2 health problems explaining just .1% unique variance in T3 health behaviours). The health problems to stress path ( $B = .022$ , 95%CI =  $.010$ ,  $.033$ ,  $p < .0005$ , with T2 health problems explaining 1.1% unique variance in T3 stress), although significant, were significantly weaker than the stress to health problems path (test of difference: difference =  $.584$ , 95%CI =  $.260$ ,  $.912$ ,  $p < .0005$ ). This supports not only the existence of effects between stress and health problems in both directions but also that the dominant effect occurs in the direction hypothesized, that is, from stress to health problems.

When rerunning these analyses on the sample of 328 respondents who had completed all model variables at all three time points, the results and conclusions mirrored those described above. The corresponding Table S1 and Table S2 for this sample can be accessed in the online Supplementary Materials.

## DISCUSSION

The current study aimed to address the limitations of previous research by providing a temporal test of the procrastination–health model (Sirois et al., 2003). Consistent with our hypotheses, trait procrastination was associated with higher perceived stress and less frequent practice of health-promoting behaviours. However, only the stress-mediated pathway linked trait procrastination to health problems over time; the indirect effect through health behaviours was not significant. Importantly, these findings held after accounting for the contributions of conscientiousness and neuroticism, suggesting that trait procrastination has incremental value in predicting health problems via higher stress in relation to these higher-order personality factors.

Our findings are generally consistent with previous cross-sectional investigations of the procrastination–health model and provide the first longitudinal test of the ways in which chronic procrastination is linked to short-term health outcomes. The two previous tests of the pathways linking trait procrastination to health similarly found that stress was the key pathway linking procrastination to poor health (Sirois, 2007; Sirois et al., 2003). In a sample of students, health behaviours did not mediate the procrastination–health relationship (Sirois et al., 2003), and in replication with community adults, the health behaviour pathway was only significant when the stress pathway was not included in the model (Sirois, 2007). Our findings are also consistent with a 9-month longitudinal investigation of the effects of chronic procrastination on health outcomes in a large sample of Swedish university students (Johansson et al., 2023). Procrastination predicted higher stress, physical symptoms, poor sleep quality and physical inactivity, after accounting for a number of potential confounds. However, key distinctions between the current investigation and the Swedish study are that we tested the links between procrastination and a set of physical health problems

rather than a single physical symptom (i.e. disabling pain), as well as the potential pathways that might explain the link between procrastination and health in accordance with the procrastination–health model. Despite these distinctions, overall, our findings generally align with this previous research and further demonstrate that these associations and the linking pathways hold over time, even after accounting for the contributions of related personality traits.

There are several factors that may explain the non-significant behavioural pathway from procrastination to health problems in the current study. Given the known reciprocal links between stress and health behaviours (Rod et al., 2009), it may be that both routes are important in the long-term, but only stress is key when assessing short-term effects of chronic procrastination. Accordingly, this finding may be attributable to the short-term time scale of the current study and what is needed for the proposed links between procrastination and health behaviours to accumulate and manifest as health problems. As Andreou (2007) has noted, the intransitive preference structures that characterize procrastination are based on small incremental differences in the potential negative outcome of failing to adhere to a health behaviour; these accumulate over time and do not have an immediate health effect. Arguably, this is at the heart of the problem in considering the behavioural effects of procrastination on health. For example, in the case of someone who is overweight or obese, procrastinating on behaviours to reach a healthy weight today will neither actually kill the individual nor make him or her noticeably sicker. However, the cumulative effects of this delay are well established (World Health Organization, 2013). Understanding the impact of chronic procrastination on health via the behavioural pathway will therefore likely require investigation over much longer periods of time.

It is also possible that the health behaviour pathway was non-significant in the current study because of the nature of the health problems that were assessed. Participants reported acute health problems such as headaches, digestive issues, muscle pain and flus/colds. Health problems of this nature are more likely to be affected in the short term by the experience of stress than by poor eating habits or lack of regular exercise (e.g. Cohen et al., 2012). As noted above, a more complete understanding of the contribution of chronic procrastination to health requires examining a wider range of health issues, over longer periods of time, and thus is a key agenda for future research on the health implications of chronic self-regulation failure. That said, examining the linkages between procrastination and health on a micro-level scale, such as with daily diary methods, might also provide important insights into the acute effects of chronic procrastination on stress and any associated and more immediate health effects.

## Limitations and future directions

The current findings should be considered in light of several limitations. Although we collected data over three time points, this was a time-lagged cross-sectional study, making it difficult to confirm the directionality of the relationships among the variables tested. Nonetheless, the order of the variables tested was informed by theory, and the test of the reverse causality paths (i.e. from health problems to stress and health behaviours) indicated they were weaker than those of the hypothesized pathways, suggesting that the model may be a good approximation of the relationships between procrastination and health. We only administered the measure of procrastination at Time 1, as it was expected that there would be little change in this measure of a generalized tendency to procrastinate over a short period of time. Indeed, previous research confirms that trait procrastination as measured by the General Procrastination Scale has excellent stability over a 10-year period (Steel, 2010). However, future research could verify this by including this measure at all time points. As with any observational study, there is always the possibility that other unknown factors linked to the predictor and outcome variables play a causal role in the relationships observed. Despite this, the procrastination–health model is useful for providing a glimpse of the possible mechanisms linking chronic procrastination to health and which cannot be easily examined with a more controlled design.

In addition to the limitations noted previously regarding the short timeframe of the study and the types of health problems assessed, the undergraduate sample was relatively young and healthy, and this may also explain the lack of significant indirect effects of trait procrastination on health problems over time via health behaviours. This sample was chosen given the high rates of chronic procrastination among university students and thus the relevance of understanding the implications of chronic procrastination for this population. Given that the surveys were administered across the academic term with the final assessment just before the exam period, it is possible that stress levels, especially those due to procrastination, were heightened. Replicating the current findings longitudinally with a more representative adult sample is needed to confirm the generalisability of these results and the extent to which the behavioural route of the procrastination–health model contributes to the health outcomes among people who chronically procrastinate. In addition, it would also be useful to test the model with other measures of health behaviours, and ideally those measured with objective means such as actimeters. Although the WBI provides an overall estimate of the frequency of a general set of four health-promoting behaviours that are conceptually grouped (Lippke et al., 2012), it does not provide a more granular assessment of individual behaviours. Future research on procrastination and health would benefit from taking other approaches to assessing health behaviours and estimating the frequencies of specific behaviours.

Nonetheless, the current study has addressed the limitations of previous research on the procrastination–health model (Sirois, 2007; Sirois et al., 2003), and in doing so has highlighted new insights and areas for future inquiry. The consistency of the links between procrastination and stress, health behaviours and health problems found over the 3-month study period is a new and important finding that underscores the contribution of chronic procrastination to poor health even among a sample consisting mainly of young adults. Raising awareness about the health consequences of chronic procrastination and other forms of chronic self-regulation failure among clinicians, academic counsellors and other stakeholders could also lead to the implementation of targeted interventions to help address this problem. Indeed, a meta-analysis of psychological interventions targeting procrastination behaviour suggests that cognitive approaches can have small-to-moderate effects (Rozenal et al., 2018). However, interventions that address the dysfunctional beliefs and automatic thoughts that contribute to further stress and procrastination (Stainton et al., 2000) may be an effective approach for reducing both procrastination and any associated stress (Pychyl & Flett, 2012). In addition, finding ways to deal with the health-related by-products of self-regulation failure, such as high stress and poor health behaviours, is also an important consideration. To this end, strategies that improve coping may be beneficial given that trait procrastination is associated with less use of adaptive coping strategies and greater use of maladaptive coping strategies (Sirois & Kitner, 2015).

Evidence suggests that procrastination as a chronic problem is becoming increasingly prevalent in North America, Europe and worldwide (Ferrari et al., 2005, 2009; Hen & Goroshit, 2018; Steel, 2007). From a public health perspective, our findings suggest that the health risks from chronic procrastination are an issue that may need to be addressed sooner, rather than later.

## AUTHOR CONTRIBUTIONS

**Christopher B. Stride:** Data curation; formal analysis; visualization; writing – review and editing. **Timothy A. Pychyl:** Conceptualization; methodology; resources; writing – review and editing.

## CONFLICT OF INTEREST STATEMENT

All authors declare no conflict of interest.

## OPEN RESEARCH BADGES

This article has earned an Open Data badge for making publicly available the digitally-shareable data necessary to reproduce the reported results. The data is available at [https://osf.io/nsvmd/?view\\_only=eab0a1d97aef4637842a66339a8f8291](https://osf.io/nsvmd/?view_only=eab0a1d97aef4637842a66339a8f8291).

## DATA AVAILABILITY STATEMENT

The data, analyses and a list of materials used for this research are available on the Open Science Framework website: [https://osf.io/nsvmd/?view\\_only=eab0a1d97acf4637842a66339a8f8291](https://osf.io/nsvmd/?view_only=eab0a1d97acf4637842a66339a8f8291).

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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