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Perseverative cognition and health behaviours: exploring the role of intentions and perceived behavioural control

Dane McCarrick, Andrew Prestwich  and Daryl B. O'Connor

School of Psychology, University of Leeds, Leeds, UK

ABSTRACT

Objective: Worry and rumination (Perseverative Cognition, PC) have been associated with health behaviours, but the underlying mechanisms are unknown. Given the role of physiological experiences on perceived behavioural control (PBC) and emotion regulation on intention-health behaviour relationships, we tested whether: PC prospectively predicts poorer health behaviours; PC moderates the relationship(s) between intentions/PBC and health behaviour, as well as whether the relationship between PC and health behaviour is mediated by intentions and PBC.

Methods and Measures: In a prospective design, 650 participants (mean age = 38.21 years; 49% female) completed baseline measures of intentions, PBC and PC (worry and rumination) and 590 (mean age = 38.68 years; 50% female) completed follow-up (Time 2) measures of health behaviours (physical activity, sleep, sedentary activity, unhealthy snacking) 1-week later.

Results: Worry and rumination (at T1) predicted poorer sleep quality. Worry, but not rumination, moderated PBC-physical activity frequency relations. Consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant.

Conclusion: PC is associated with poorer sleep quality and PBC can play a mediating role in such relationships. Future research should further consider the role that PBC plays in PC-health behaviour relations.

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KEYWORDS

Perseverative cognition; theory of planned behaviour; health behaviours

Introduction

Recent advances in stress theory have demonstrated the complex challenge stress represents for neural, endocrine, and behavioural systems (O'Connor et al., 2021). For instance, traditional models of stress have linked high levels of stress with a greater risk of a range of diseases and health problems such as cardiovascular disease, hypertension, stroke, obesity, immune function, and accelerated rates of disease progression

CONTACT Dane McCarrick  D.J.McCarrick@leeds.ac.uk  School of Psychology, University of Leeds, Leeds LS2 9JT, UK.

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(Cohen et al., 2007, 2012; O'Connor et al., 2021; Steptoe & Kivimäki, 2012; Tomiyama, 2019). While stress has been shown to influence health via this direct, biological pathway, stress can also influence health via behavioural pathways such as through changes in health behaviours (such as unhealthy eating, sedentary behaviour) (Finch et al., 2019; O'Connor et al., 2021). These two distinct but interacting pathways perform a bi-directional, yet pervasive function, with adaptations in behaviour impacting biology and changes in biology influencing behavioural changes that, in turn, may modify health status over time. This is important as, when prolonged, increased periods of stress may adversely impact on health outcomes and disease states (Appel et al., 2021; Larsen & Christenfeld, 2009; Renna et al., 2021).

A number of theoretical models now exist that have improved our understanding of how stress may lead to disease. One leading theoretical model, the Perseverative Cognition (PC) Hypothesis (PC Hypothesis, Brosschot et al., 2006), proposes that, where a physical stressor is absent, the cognitive representation alone induces the physiological stress response; such that when stress is perseverated upon, the damaging physiological activation associated with stress is also extended, increasing susceptibility to stress-related ill-health. Thus, the direct relationship between stress and disease is intensified when a stressor is subject to repetitive thought, as the duration of time that the body is exposed to the damaging physiological stress response is prolonged (for recent meta-analysis, see Ottaviani et al., 2016). Crucially, it has now been shown that worry and rumination serve as key vehicles for stress, with past stressful events (rumination) or feared future events (worry), observed as acting as key mediators through which psychosocial stress leads to ill-health (for reviews, see Ottaviani, 2018; Verkuil et al., 2010).

Moreover, the PC Hypothesis was further extended in 2016 to a model that incorporates not only the direct biological pathway to disease, but also to one including an *indirect* behavioural pathway (EPC Hypothesis). In this 2016 meta-analysis, increased levels of PC were shown to be associated with increased health risk behaviours (e.g. greater substance use, unhealthy eating and smoking, but not health promoting behaviours, Clancy et al., 2016), and similar findings were found in a later meta-analysis for sleep outcomes (Clancy et al., 2020). These findings were further supported, in a meta-analysis of 36 RCTs, where psychological interventions to reduce worry and rumination (relative to control groups) produced (on average) medium-sized effects on rumination ($g = -.58$) and small-to-medium sized effects on worry ($g = -.41$) and consequently influenced health behaviours ($g = .31$) (McCarrick et al., 2021). More evidence then soon followed, Clancy et al. (2022) demonstrated associations between worry, rumination and health behaviours, cross-sectionally and prospectively, including in sleep and unhealthy snacking. Together, these recent findings provide support for the PC Hypothesis and the health risk it poses not only directly via the neuroendocrine responses originally cited by Brosschot et al. (2006), but also indirectly via the adoption of unhealthier behaviours.

However, despite the clear directionality of these findings in terms of the consequences that stress, through PC, holds for health, questions remain around how PC may function as part of a larger, more complex, behavioural system. For instance, an important unresolved question is how does increased PC lead to poorer health behaviours? The answer may gravitate around the role of behavioural appraisal and/

or goal attainment, both of which are constructs receiving a great deal of empirical attention within behaviour change literature (see Armitage & Conner, 2000; Hawkes et al., 2021; Sheeran et al., 2005). Crucially, some explanation of the PC-health behaviour link may be sought from theories of understanding health behaviours which emphasise the role of intentions as the proximal determinant of action (e.g. Theory of Reasoned Action (TRA), Ajzen & Fishbein, 1980, 1977; Theory of Planned Behaviour, TPB, Ajzen, 1991).

The TPB posits intention as a direct predictor of behaviour and PBC, when it reflects actual control, to be a moderator of intention-behaviour relations. As such, intentions and PBC have the most proximal roles in influencing behaviour on the basis of this model. Related models, such as the COM-B (Michie et al., 2011) also highlight the important role of capability (related to PBC) and motivation (related to intention), plus opportunity, for behaviour. Increased capability, opportunity and motivation for a behaviour increase the likelihood of a behaviour being enacted. Various studies have demonstrated significant relations between intention-health behaviours and PBC-health behaviours (see, for example, McEachan et al., 2011, for a review). According to the TPB, the relationship between a range of 'background factors' such as personality, religion and, importantly here, emotion with behaviour are subsumed or mediated by more proximal determinants of behaviour (including beliefs underlying PBC and, in turn, intentions). It is possible, therefore, that levels of PC (worry or rumination) may adversely affect intentions or PBC that in turn reduce the likelihood of behaviour being enacted (i.e. a mediated pathway). In other words, worry and/or rumination may attenuate the relationship between TPB variables such as intentions and PBC and behaviours relating to health.

Further support for a potential PC-PBC-behaviour pathway can be taken from Bandura's (1977) work on self-efficacy. Like PBC, self-efficacy reflects people's beliefs about their ability to perform a particular behaviour. An important source of self-efficacy, according to Bandura (1977), is an individual's physiological experiences and how these are interpreted. As such, worry and rumination, as negative physiological experiences, could serve to lower self-efficacy or PBC, which, in turn, negatively influences health behaviours.

The relationship between intentions and health behaviours is thought it be important for achieving behavioural goals (e.g. getting better sleep, reducing alcohol consumption, and increasing exercise frequency; Baumeister & Bargh, 2014; Kuhl & Quirin, 2011) and, significantly, for emotion regulation (Bandura, 1992, 1998). Indeed, unwanted thoughts and feelings may disrupt behavioural efforts to enact an intention or, equally, interfere with the known determinants of intentions. For example, worry about an upcoming psychotherapy appointment predicted non-attendance, despite participants' holding strong intentions to keep the appointment (Sheeran et al., 2007) and negative mood and high levels of worry led to unintended risk behaviour (Webb et al., 2010). In other words, despite one's best intentions to engage in healthier behaviours, PC, whether in the form of worry and/or rumination, may get in the way and attenuate the intention-behaviour relationship (i.e. the intention-behaviour gap, Sheeran & Webb, 2016).

For these reasons, a fresh consideration of how PC may serve as a moderator between intentions and behaviour, and how intentions and PBC may mediate this

relationship, is both timely and warranted. Therefore, here, we tested the role of intentions and PBC in the relationship between PC (i.e. worry and rumination) and a range of health behaviours (i.e. sleep, unhealthy snacking, physical activity and sedentary activity). Specifically, we tested whether PC moderates the relationship(s) between the intentions/PBC and behaviour, as well as whether the relationship between PC and health behaviours was mediated by intentions and PBC. Therefore, informed by the existing literature, the following was hypothesised:

Hypothesis 1 (H1a and H1b):

Higher levels of PC (worry (H1a) & rumination (H1b)) will significantly predict poorer health behaviours (i.e. poorer sleep outcomes, more unhealthy snacking, less physical activity, and more sedentary activity).

Moderation Hypotheses (H2a and H2b):

The relationships between intentions (H2a) and PBC (H2b) with behaviour will be moderated by PC; such that the intention-behaviour and PBC-behaviour links will be attenuated at higher levels of PC.

Mediation Hypotheses (H3a & H3b):

The relationship between PC and behaviour will be mediated by intentions (H3a) and perceived behavioural control (H3b).

Method

Design

The study employed a prospective survey design. Participants completed measures of intentions, perceived behavioural control, and PC (worry and rumination) at Time 1 (T1; baseline); and measures of self-reported health behaviour (sleep, unhealthy snacking, physical activity and sedentary activity) at Time 2 (T2; follow-up) one week later. Subjective norms and attitudes were also measured at T1 but were not part of any of the preregistered hypotheses and thus are not reported here. This study was preregistered on AsPredicted (see, here).

Participants

A power calculation (in G*Power version 3.1; Faul et al., 2009) revealed 588 participants were required to detect an effect size of $f = .02$ based on a power ($1 - \beta$) of 0.80 in a two-tailed test with alpha set at .01. However, to account for potential attrition, we planned to recruit 650 participants. The study was powered a-priori to detect a small moderator effect; a conservative approach when the statistical parameters relating to the study variables are unknown and where no pilot data are available (Aguinis et al., 2005). Consequently, 650 participants (49% female; 84.75% from United Kingdom & Ireland; 86.49% of White ethnicity; 18.98% educated to degree level; mean age = 38.2 years (SD = 11.59); mean BMI = 23.81 (SD = 6.45)) were recruited via Prolific and completed the baseline (T1) survey. Of these, 590 completed the follow-up (T2) survey (50% female; 84.75% UK & Ireland; 87.46% of White ethnicity; 19.32%

educated to degree level; mean age = 38.68 (SD = 11.64); mean BMI = 23.58 (SD = 6.82)), representing a 9.23% attrition rate. Participants were not eligible for the study if they were under 18 or if they were not fluent in English. Participants received a £5 credit voucher (£2.50 for each timepoint) after completing both surveys.

Measures

Intentions and PBC

Intentions to enact a specific behaviour was measured via the following three items for each behaviour (e.g. *I intend to avoid unhealthy snacks over the next 7 days; I want to avoid unhealthy snacks over the next 7 days; I plan to avoid unhealthy snacks over the next 7 days*; Definitely don't (1) – Definitely do (7)). PBC was tapped via two items for each health behaviour (e.g. *How confident are you that you will do sedentary activity over the next week?*; Not at all confident (1) – Very confident (7) and *How much control do you have over whether or not you will do sedentary activity over the next week?*; No control (1) – Complete control (7)). All constructs showed acceptable internal consistency ($\alpha = .79-.90$) and were coded so that high values indicated high levels on the variable of interest.

Perseverative cognition

At T1, worry and rumination were assessed via brief versions (Topper et al., 2014) of the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) and the Ruminative Responses Scale (RRS; Nolen-Hoeksema, 1991). In this study, the brief versions have acceptable to high internal consistency (brief PSWQ: $\alpha = .89$; brief RRS: $\alpha = .79$) and have been shown to correlate highly with the full questionnaires (brief PSWQ: $r = .91-.94$; brief RRS: $r = .88-.91$; Topper et al., 2014). For the brief-RRS, participants are instructed to indicate what they generally do when they feel down or depressed (for 4 items) on a five-point scale varying from 1 ('almost never') to 5 ('always'). Example items include *Think about all of your shortcomings, failings and faults* and *Think about your feelings of fatigue and achiness*. A total score is calculated by summing the items and scores range from 4–20, with higher scores representing a greater degree of rumination. For the brief-PSWQ, participants are instructed to indicate how typical statements are of them (for 5 items) on a five-point scale varying from 1 ('not at all typical of me') to 5 ('very typical of me'). Example items include *Many situations make me worry* and *When I am under pressure, I worry a lot*. A total score is calculated by summing the items and scores range from 5–25, with higher scores representing a greater degree of worry.

Health behaviours

Physical Activity. At T2, levels of self-reported physical activity were measured via the strenuous and moderate activity items from the Godin-Shephard Leisure-Time Physical Activity Questionnaire (2011). The scale yields a frequency and duration score for each type of intensity, which is then averaged to create a gross score for physical activity duration and frequency across the past 7 days, respectively. Note, the scale also includes mild physical activity items; however, we did not include these as they reflected routine daily behaviours (e.g. walking/lifting shopping) and not the type of

conscious physical activity behaviours associated to the moderate and vigorous activity items (e.g. playing sports/long distance running, respectively). In this study, the constructs showed acceptable internal consistency ($\alpha = .77-.92$). This measure has been validated (see, Godin, 2011) and widely employed in previous prospective TPB/health behaviour change studies (e.g. Lesser & Nienhuis, 2020; Marker et al., 2018)

Sleep. Sleep Onset Latency (SOL; i.e. *Over the past week, how long did it take you (on average each night) to get to sleep in minutes?*), Total Sleep Time (TST; i.e. *Over the past week, how long (on average each night) did you sleep for in hours?*) and subjective Sleep Quality (SQ; i.e. *Overall, rate the quality of your sleep over the past 7 nights on a scale of 1 (very poor) to 7 (very good)*) were taken from the Consensus Sleep Diary (Carney et al., 2012) to assess markers of sleep at T2. These measures have been extensively used in a variety of studies aiming to capture sleep quality/quantity and these items, from the Consensus Sleep Diary, showed acceptable internal consistency in the present study ($\alpha = .91-.96$).

Sedentary Activity (Gardiner et al., 2011). At T2, the Self-Report Sedentary Behaviour Questionnaire assessed time spent engaging in specific sedentary activities common among older adults: watching television (TV), computer use, reading, socialising, transport and hobbies. Responses were summed to reflect time spent engaging in sedentary activity during the past 7 days. In this study, total sedentary time showed acceptable internal consistency ($\alpha = .79$) and, in previous studies, has been shown to have acceptable test-retest reliability ($r = .70$) and validity ($ICC = .52$), to be sensitive to change ($r_s = .47$), and responsive to change as accelerometer-derived sedentary time ($r_s = .39$) (for review, see Gorman et al., 2014).

Unhealthy Snacking (Gardner et al., 2015). At T2, unhealthy snacking was measured using a pre-defined food frequency questionnaire for 21 snack foods. Participants reported the frequency of consuming each snack food over the past 7 days, from 'not at all' (1) to 'twice a day' (5). Fourteen of the 21 snack foods were classified as unhealthy and from this, an unhealthy snack intake variable was generated, with higher scores reflecting greater instances of unhealthy snacking across the 7-day study period (see Gardner et al., 2015, Table 2). This measure has been used in a variety of studies assessing intention-(snacking) behaviour relations (see, Hagger, 2019; Inauen et al., 2016). In this study, acceptable internal consistency was found across each of the snacking outcomes ($\alpha = .74-.91$).

Procedure

Participants accessed a link to the online survey via their Prolific account. In the first survey, participants read study information, consented and then provided their demographic details (e.g. age, sex, height, weight and education). The following measures were then completed in the following order: intentions then PBC in relation to physical activity, sleep (sleep onset latency, total sleep time, sleep quality), sedentary activity, unhealthy snacking; worry, followed by rumination. At T2, participants were contacted by email (within Prolific) with a link to the second survey to complete measures of physical activity, sleep, sedentary behaviour and unhealthy snacking. Participants were then debriefed. The surveys were completed in February 2022. The average time taken to complete the survey was 7.12 minutes ($SD = 4.06$ minutes; T1) and 6.56 minutes

($SD=5.11$ minutes; T2). Ethical approval was granted by a university ethics committee (Ethics number: PSY-320, date of approval 21.11.21).

Analysis strategy

All analyses were conducted using R-Studio (3.6.2) software. The data can be accessed here, via the Open Science Framework. Prior to conducting the main analyses, a comprehensive check of the associated statistical assumptions for normality, linearity, statistical independence and homoscedasticity/homogeneity of variance were conducted. In addition to visual checks (scatter plots, Cullen & Frey graphs, QQ-plots, PP-plots) formal tests (Durbin-Watson, Goldfield-Quandt, Variance Inflation Factor) were also computed to ensure the data were appropriate for regression/mediation analysis. In short, no major concerns were raised by these checks and the data were considered suitable for regression-based analyses.

Correlational analyses assessed the interrelationships between the measured variables. Multiple regression models assessed whether higher levels of worry or rumination (at T1) predicted poorer health behaviours (at T2) [Hypotheses 1a, 1b] and whether worry or rumination moderated intention- or PBC-behaviour relationships [Hypotheses 2a, 2b]. For Hypotheses 2a and 2b, health behaviours (at T2) were regressed on the predictor variables (step 1: intention or PBC), the moderator variables (step 2: worry or rumination) and their respective interaction terms (step 3). Simple slopes analyses were used to decompose significant interactions (see, Preacher et al., 2006). Ordinary-least squares path analyses (Hayes, 2017) tested whether the relationships between intention/PBC and health behaviours were mediated by worry or rumination [Hypotheses 3a, 3b].

Due to the large number of analyses, a Bonferroni correction was applied to reduce the type 1 error rate. This consisted of dividing the alpha level by the number of comparisons (Haynes, 2013). Outcomes which would typically be considered significant ($p < .05$) were not interpreted as such here unless they met the corrected alpha level. Alphas were corrected per block of analyses (i.e. each set of analyses had 4 types of behavioural outcomes, so $0.05/4 = .0125$). Therefore, .0125 was the a-priori, corrected alpha level and outcomes were considered significant if $p < .0125$.

Results

MANOVA, for continuous T1 variables (worry, rumination, intentions, PBC, and chi-square analyses, for categorical T1 variables (sex, nationality, ethnicity, employment status & education), revealed no significant differences between completers ($n=590$) and drop-outs ($n=60$). The percentage of missing data across T1 and T2 was 9.23%. Therefore, given 590 participants satisfied the sample size power requirements, and in view of maximising temporal validity, we proceeded with complete-case-analysis ($n=590$) using listwise deletion to remove the 60 missing responses from T1 (see, Kang, 2013). We did, however, perform a sensitivity analysis to ensure that the missing data obtained at T2 was missing at random. An expectation maximisation chi-square test (Little, 1988; performed in SPSS) was non-significant at T1 (all participants; $p = .371$) and at T2 (completers only, $p = .472$) indicating data was missing completely at random.

The correlational analyses found that both worry, $r(588) = -.257, p < .001$, and rumination, $r(588) = -.215, p < .001$, (at T1) were related with sleep quality (at T2). Significant (cross-sectional) relationships were present between worry, $r(588) = -.133, p < .01$, and rumination, $r(588) = -.114, p < .01$, (respectively) and PBC over sleep quality, while rumination was also significantly correlated with intentions about unhealthy snacking (cross-sectionally), $r(588) = .110, p = .007$. There were no other significant correlations between worry or rumination and any of the other health outcomes; therefore, the non-significant relationships were not tested in the regression analyses related to hypothesis 1(a&b) (see, Hawkes et al., 2012).

Intentions and PBC (at T1) were significantly correlated with their respective health behaviours (at T2). Intentions were significantly correlated with sleep quality, $r(588) = .260, p < .001$, and with total sleep time, $r(588) = .381, p < .001$. PBC was also significantly correlated with sleep quality, $r(588) = .501, p < .001$, and with total sleep time, $r(588) = .532, p < .001$.

The analyses also revealed that worry and rumination were modestly correlated with each other, $r(588) = 0.55, p < .001$, suggesting that they are distinct constructs and that testing them as individual predictors was justified. Descriptive statistics and Pearson's correlations between worry and rumination, intentions and PBC, and the rest of study variables are reported in Table 1.

Hypothesis 1: Higher levels of PC (worry (H1a) & rumination (H1b)) will significantly predict poorer health behaviours

In partial support of Hypothesis 1, in separate regressions, worry, $\beta = -.257, p < .001, R^2 = .066$ (H1a), and rumination, $\beta = -.215, p < .001, R^2 = .046$ (H1b) (at T1), significantly predicted poorer sleep quality (at T2) such that higher levels of worry and rumination were associated with poorer sleep quality. These associations remained significant after controlling for age, gender, ethnicity and nationality. However, worry and rumination did not significantly predict any of the other health behaviour outcomes.

Hypothesis 2: the relationships between intentions (H2a) and PBC (H2b) with behaviour will be moderated by PC; such that the intention-behaviour and PBC-behaviour links will be attenuated at higher levels of PC

There was no support for H2a. The relationship between intentions and behaviour was not moderated by worry (physical activity frequency, $p = .121$, or duration, $p = .291$; sedentary activity, $p = .766$; unhealthy snacking, $p > .05$; sleep time, $p = .455$, quality, $p = .239$, latency, $p = .221$) or rumination (physical activity frequency, $p = .930$, or duration, $p = .563$; sedentary activity, $p = .757$; unhealthy snacking, $p > .05$; sleep time, $p = .772$, quality, $p = .301$, latency, $p = .169$).

There was limited support for H2b. For all but one outcome, the relationship between PBC and behaviour was not moderated by worry (physical activity duration, $p > .05$; sedentary activity, $p = .236$; unhealthy snacking, $p = .672$; sleep time, $p = .805$, quality, $p = .290$, latency, $p = .248$) or rumination (physical activity frequency, $p = .701$, or

Table 1. Relationships between worry and rumination and study variables.

	M	SD	1	2	3	4	5	6	7	8	9	10
1. Worry (T1)	18.09	6.42	–									
2. Rumination (T1)	10.33	2.87	0.55***	–								
3. PA Intention (T1)	5.62	1.77	–0.06	–0.02	–							
4. Sleep Intention (T1)	5.22	1.55	–0.03	–0.04	0.07	–						
5. SA Intention (T1)	5.51	1.56	0.05	0.03	0.01	–0.04	–					
6. US Intention (T1)	4.72	1.77	0.01	0.11**	0.23***	0.12**	–0.10	–				
7. PA PBC (T1)	5.59	1.41	–0.07	–0.06	0.804***	0.13***	0.03	0.17***	–			
8. Sleep PBC (T1)	4.01	1.77	–0.13**	–0.11**	0.01	0.62***	–0.06	0.09	0.12**	–		
9. SA PBC (T1)	5.58	1.22	–0.04	–0.06	0.02	0.04	0.60***	–0.07	0.11**	0.10*	–	
10. US PBC (T1)	4.77	1.22	–0.10	–0.01	0.16***	0.14***	–0.07	0.55***	0.20***	0.23***	0.07	–
11. TST (T2)	6.65	1.16	0.06	0.08	0.08	0.38***	0.02	0.03	0.13***	0.53***	0.10*	0.11**
12. SOL (T2)	37.41	65.35	–0.05	–0.10	–0.04	–0.06	0.06	0.04	–0.07	–0.12**	0.05	0.05
13. Sleep Qual (T2)	4.43	1.48	–0.26***	–0.22***	0.13**	0.26***	–0.01	0.08	0.20***	0.50***	0.12**	0.20***
14. PA (Freq) (T2)	2.92	1.56	–0.02	0.05	0.37***	0.031	–0.10*	0.12**	0.34***	0.04	–0.05	0.12***
15. PA (Dur) (T2)	35.65	35.89	–0.03	–0.01	0.24***	0.05	0.04	0.01	0.18***	0.01	0.02	0.02
16. Sedentary A (T2)	41.45	31.61	–0.05	–0.01	–0.08	0.03	0.11**	–0.01	–0.09	0.05	0.05	–0.04
17. Unhealthy S (T2)	32.38	10.59	0.04	0.03	–0.01	0.02	0.01	–0.07	–0.01	–0.02	0.01	–0.16***

Note: * $p < .0125$, ** $p < .01$, *** $p < .001$; T1 = Timepoint 1, T2 = Timepoint 2; PA = Physical Activity, SA = Sedentary Activity, US = Unhealthy Snacking, PBC = Perceived Behavioural Control, TST = Total Sleep Time, SOL = Sleep Onset Latency, Sleep Qual = Sleep Quality; Measurements: Total Sleep Time = hours; Sleep Onset Latency = minutes; Physical Activity Duration = minutes.

duration, $p = .864$; sedentary activity, $p = .965$; unhealthy snacking, $p = .907$; sleep time, $p = .809$, quality, $p = .155$, latency, $p = .316$). However, worry (at T1) and PBC (at T2) interacted to significantly predict physical activity frequency, $\beta = -.449$, $p = .011$, $R^2 = .123$. A simple slopes analysis revealed that, as worry increased, the relationship between PBC and physical activity frequency remained significant, but weakened. Specifically, while PBC was positively associated with physical activity frequency at low levels of worry, $\beta = .483$, $SE=0.06$, $p < .001$, the relationship was weaker at moderate, $\beta = .382$, $SE=0.04$, $p < .001$, and weaker again at high levels of worry, $\beta = .280$, $SE=0.06$, $p < .001$ (see OSM, Figure S1). The results from these analyses are displayed in full in Supplementary Tables S1–S3 (see Online Supplementary Material, OSM).

Hypothesis 3: the relationship between PC and behaviour will be mediated by intentions (H3a) and perceived behavioural control (H3b)

In partial support of hypothesis 3, all indirect paths from worry and rumination, through PBC, to both sleep quality and total sleep time were significant. Additional mediation models revealed no significant indirect paths from either worry or rumination, through either intentions or PBC (at T1), to the other behavioural outcomes (at T2) (see Tables 2 and 3).

Discussion

The aim of the present study was to test the relative roles of PC (i.e. worry and rumination) as well as intentions and PBC for health behaviours. There was some support that both worry (H1a) and rumination (H1b) predicted significantly poorer sleep quality, when measured one week later; however, both types of PC were statistically unrelated to the other health behaviours. The relationships between intentions and health behaviours were not moderated by worry or rumination (not supporting H2a). The relationship

Table 2. Mediation analysis for worry, intentions & PBC, and sleep behaviours.

Predictor (T1)	Mediator (T1)	Outcome(s) (T2)	Effect	b (95% CI)	S. E	R^2
Worry	Intentions	Sleep Quality	Total	-.059***	.009	.001
			Direct	-.058***	.009	
			Indirect	-.002 (-.006 – .003)	1.70	
Worry	Intentions	Total Sleep Time	Total	-.009	.007	.001
			Direct	-.007	.007	
			Indirect	-.002 (-.007 – .004)	.0003	
Worry	Intentions	Sleep Onset Latency	Total	.559	.419	.006
			Direct	.543	.418	
			Indirect	.016 (-.037 – .070)	.027	
Worry	PBC	Sleep Quality	Total	-.059***	.009	.285
			Direct	-.045***	.008	
			Indirect	-.014 (-.023 – -.005)**	.005	
Worry	PBC	Total Sleep Time	Total	.007	.007	.280
			Direct	.003	.006	
			Indirect	-.013 (-.021 – -.005)***	.004	
Worry	PBC	Sleep Onset Latency	Total	.559	.419	.018
			Direct	.411	.420	
			Indirect	.148 (.007 – .290) [†]	.072	

Note: * $p < .0125$, ** $p < .01$ *** $p < .001$; [†] $p = .05$; CI's are at 95% level; b = regression coefficient; R^2 = coefficient of determination; S.E=Standard Error, T1=Timepoint 1, T2=Timepoint 2; SQ=Sleep Quality, TST=Total Sleep Time.

Table 3. Mediation analysis for rumination, intentions & PBC, and sleep behaviours.

Predictor (T1)	Mediator (T1)	Outcome(s) (T2)	Effect	<i>b</i> (95% CI)	S. E	<i>R</i> ²
Rumination	Intentions	Sleep Quality	Total	-.111***	.021	
			Direct	-.105***	.020	
			Indirect	-.006 (-.016 – .005)	.005	.002
Rumination	Intentions	Total Sleep Time	Total	-.042*	.017	
			Direct	-.035 [†]	.015	
			Indirect	-.007 (-.019 – .006)	.006	.002
Rumination	Intentions	Sleep Onset Latency	Total	1.79 [†]	.935	
			Direct	1.72	.934	
			Indirect	.056 (-.075 – .188)	.067	.009
Rumination	PBC	Sleep Quality	Total	-.111***	.021	
			Direct	-.083***	.018	
			Indirect	-.028 (-.048 – -.008)**	.010	.273
Rumination	PBC	Total Sleep Time	Total	-.042*	.017	
			Direct	-.017	.014	
			Indirect	-.024 (-.041 – .007)**	.009	.281
Rumination	PBC	Sleep Onset Latency	Total	1.794 [†]	.935	
			Direct	1.515	.935	
			Indirect	.279 (-.008 – .565) [†]	.146	.018

Note: * $p < .0125$, ** $p < .01$ *** $p < .001$; [†] $p = .05$; CI's are at 95% level; *b* = regression coefficient; *R*² = coefficient of determination; S.E=Standard Error, T1=Timepoint 1, T2=Timepoint 2; SQ=Sleep Quality, TST=Total Sleep Time.

between PBC and health behaviours were not moderated by rumination and in most cases not by worry either. Providing limited support for H2b, the relationship between PBC and physical activity weakened as worry increased. Intentions did not mediate the relationship between PC and health behaviours (failing to support H3a). Support for relationships between PC and health behaviours via PBC (H3b) were restricted to sleep behaviours. Together, these set of findings show that PC is associated with poorer sleep quality, while revealing new relationships between the components of PC and PBC.

This study also provides partial, longitudinal support, for the extended PC Hypothesis, in which PC functions as an indirect pathway to adverse health outcomes via health behaviours (see Clancy et al., 2016). As such, consistent with the findings of this study, it would follow that worry and rumination disrupt sleep quality when measured 1-week later. These findings are broadly consistent with the McCarrick et al. (2021) and Clancy et al. (2020) meta-analyses which reported improvements in sleep following (intervention induced) changes in PC and significant small- to medium-sized associations between both worry and rumination and poorer quality sleep, respectively. They are also aligned with other studies reporting an association between thought processes such as worry and rumination and sleep quality (e.g. Barclay & Gregory, 2010; Cropley et al., 2015) and, importantly, extend the temporal validity of the (cross-sectional) correlations outlined in Clancy et al. (2022) between PC and sleep quality. It is notable that here, as in Clancy et al. (2022), total sleep time was not associated with either type of PC (in our prediction models i.e. H1) but that, unlike in the Clancy paper, sleep onset latency was not statistically related to PC. A potential reason for this may be that participant recall is poorer for total sleep time as it requires a numeric estimation of how long they slept, including the time when they were asleep (and fell asleep for SOL), which is conceivably more difficult than asking about timeframes in which participants have full consciousness. However, despite this potential measurement issue, the findings of the present study concur with recent evidence pointing to the disruptive nature of PC for sleep quality and outline the need for prospective interventions to incorporate measures of sleep in their design.

The current study is one of the first to consider PC within the context of predictors of behaviour (intentions and PBC). A limitation of the current PC literature is that it contains few empirical efforts to understand how worry and/or rumination may interact with, or otherwise relate to, other cognitive processes to influence behaviour. Therefore, the findings of the present study, showing that worry and PBC interact to significantly predict a health behaviour (i.e. limits in physical activity) and that the relationship between PC and sleep outcomes are mediated by PC, are novel and interesting findings for not only for the stress literature but for understanding determinants of health behaviours more broadly. To our knowledge, there are no empirical studies to compare these results with. However, these findings add weight to the argument that the relationship between the determinants of behavioural intentions, such as PBC, and health behaviours are sensitive to worry (Baumeister & Bargh, 2014; Kuhl & Quirin, 2011) and supports the predictive utility of the TPB (Ajzen, 1991) in influencing behaviour. While the causality for these relationships are not clear, Bandura (1992, 1998) has argued that adverse physiological experiences can undermine or weaken perceptions of control or self-efficacy, which may be why the PBC-physical activity link is attenuated by higher levels of PC in the current study and explain the process through which PC influences sleep outcomes. In addition, targeting PC, or indeed PBC, may result in downstream changes in physical activity engagement and better sleep outcomes. Therefore, given the associated health-risks of PC via health behaviours such as (lack of) physical activity (see, Taylor, 2003; Vogel et al., 2022) and sleep (Radstaak et al., 2014; Van Laethem et al., 2015), the additive predictive utility of both PC and PBC should be carefully considered when designing new interventions to improve mental and physical health. This new evidence also has practical implications for future studies, such as those wishing to use ecological momentary assessment methods; indeed, examining how PC, TPB variables and health behaviours interact under a more precise temporal lens, may lead to greater understanding of the processes that influence behaviour in real-world contexts.

It must be highlighted that despite the aforementioned significant relationships between study variables, no significant relationships were observed for any of the other behavioural outcomes. Indeed, the hypotheses relating to physical activity (with the expectation of H2a), sedentary activity, and unhealthy snacking were not supported. Neither types of PC interacted with intentions or PBC to influence these outcomes and the mediation models containing intentions and PBC were statistically unrelated to the links between both types of PC and these behavioural outcomes. These null effects were surprising, however, not entirely inconsistent with previous studies. For example, Clancy et al. (2016) found that PC was associated with health-risk behaviours but not health-promoting, behaviours. Bélair et al. (2018) also reported that in a large-scale cross-sectional study ($n=9702$) both physical and sedentary activity were not consistently associated with symptoms of worry (such as anxiety), while later studies have found varied relationships between PC and other health-related behaviours, such as unhealthy snacks (see, Eschle et al., 2022; Eschle & McCarrick, 2021).

Limitations

The null findings within the current study may also have been associated with methodological and design factors. For example, we asked participants to report their

health behaviours looking back over the past 7 days. The results may have been different if we had utilised daily assessments of their behaviours over a longer period of time or if we measured worry and rumination more closely to each of the outcomes (cf., O'Connor et al., 2022). Equally, it is also possible that the sample employed in this study contributed to selection bias, such that people with Prolific accounts may be more likely to actively participate in research and may differ on other characteristics too. Therefore, future studies ought to consider employing more precise daily diary type approaches, and consider their sampling strategy, before the current results can be confirmed or otherwise (e.g. O'Connor et al., 2020). Equally, re-examination of the significant cross-sectional relationships observed in this study would be beneficial. For example, albeit small, ($r = .110$, $p = .007$), the relationship between rumination and unhealthy snacking intentions was significant. It is possible that unhealthy snacking represents a coping strategy/response; however we were not able to disentangle these links in the present study, indicating further work is needed to establish potential pathways between types of PC and health behaviours such as unhealthy snacking.

In conclusion, this study provides partial support for the extended PC Hypothesis (Clancy et al., 2016) and reveals novel findings for the role of PBC as a mediator between PC and sleep-related outcomes. Both worry and rumination were found to predict poorer sleep quality, when measured one week later. Worry and PBC interacted to predict significantly lower physical activity. In addition, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant. Worry and rumination had been consistently linked with health behaviours, however the mechanisms underlying these relationships have been, until now, largely unknown. Therefore, these findings provide new longitudinal support that PC is associated with poorer sleep quality, while also revealing new relationships between the components of PC, PBC and health behaviours.

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ORCID

Andrew Prestwich  <http://orcid.org/0000-0002-7489-6415>

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