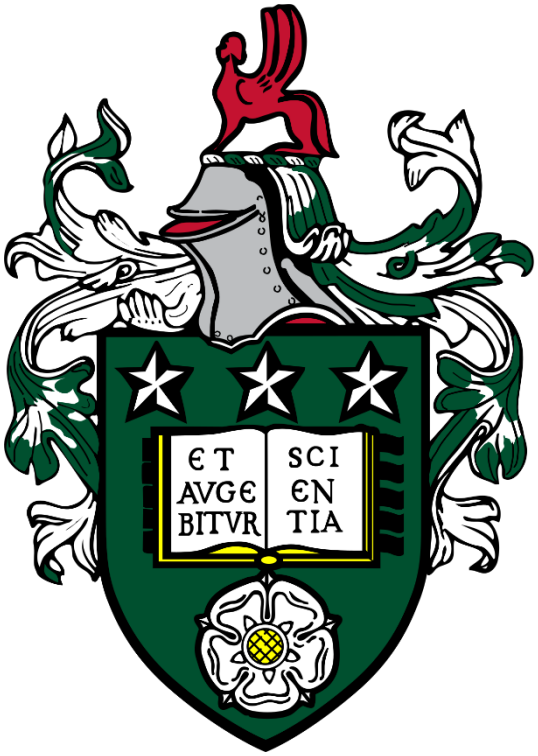
The extended perseverative cognition hypothesis: Testing the effects of worry/rumination on physical and behavioural health outcomes

Dane Jamie McCarrick

Submitted in accordance with the requirements for the degree

of Doctor of Philosophy



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This thesis is an alternative style doctoral thesis and comprises jointly authored published manuscripts; therefore, the contribution of the candidate and the other authors to this work has been explicitly indicated below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to the work of others.

Chapter 2 contains the manuscript, 'The role of Perseverative Cognition in the Job Strain-Health Outcome relationship' by McCarrick, D., Prestwich, A., & O'Connor, DB., in *Psychology and Health* (2022). This online cross-sectional study was jointly devised by Andrew Prestwich, Daryl O'Connor, and I. Data analysis was solely performed by me (with advice and supervision from my supervisors), and both myself, Andrew Prestwich and Daryl O'Connor contributed to the written publication.

Chapter 3 contains the manuscript, 'Health effects of psychological interventions for worry and rumination: *A meta-analysis*' by McCarrick, D., Prestwich, A., O'Connor, D. B., & Prudenzi, A., in *Health Psychology* (2021). This systematic review and meta-analysis was jointly conceived by Andrew Prestwich, Daryl O'Connor and I. I conducted the data analysis under the supervision of Andrew Prestwich and with input and advice from Daryl O'Connor, and each of us contributed to the written publication. Arianna Prudenzi, the fourth author on this publication, was the second reviewer and provided comments on the final manuscript.

Chapter 4 contains the study, 'Effects of worry postponement on daily worry and sleep: A randomised controlled trial', by McCarrick, D., Prestwich, A., Ferguson, E., & O'Connor, D. B, is currently under review. This (online) randomised controlled trial was jointly devised by Andrew Prestwich, Daryl O'Connor, and I. Due to the complexity of the hierarchal data within this study, primary data analysis was performed by Eamonn Ferguson. I, Andrew Prestwich, Eamonn Ferguson and Daryl O'Connor contributed to the written manuscript.

Chapter 5 contains the manuscript, 'Perseverative cognition and health behaviours: Exploring the role of intentions and perceived behavioural control' by McCarrick, D., Prestwich, A., & O'Connor, D. B. (2022) in *Psychology and Health*. This online prospective study was jointly devised by Andrew Prestwich, Daryl O'Connor, and I. Data analysis was solely performed by me (with advice and supervision from my supervisors), and both myself Andrew Prestwich and Daryl O'Connor contributed to final publication.

The candidate confirms the material submitted in this thesis complies with the University of Leeds 'submission of doctoral thesis by alternative format' guidelines and satisfies the criteria for this submission route therein. The candidate confirms that he is the first author on each of the publications included in this thesis and that each derived from original research undertaken whilst registered as a doctoral student at The University of Leeds.

The thesis is constructed in this format in view of providing the examiner(s) optimum clarity over the variety of research findings derived from the PhD. There are six chapters within this thesis, encompassing: Chapter 1) a general introduction section containing the background, rationale and aims of the thesis; Chapter 2) Study 1: The role of Perseverative Cognition in the job strain-health outcome relationship; Chapter 3) Study 2: Health effects of psychological interventions for worry and rumination: *A meta-analysis*; Chapter 4) Study 3: Effects of worry postponement on daily worry and sleep: A randomised trial; Chapter 5) Study 4: The role of intentions and perceived behavioural control in perseverative cognition and health behaviour relations; Chapter 6) a general discussion containing implications, future directions, strengths and limitations of the thesis and a conclusion.

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research contained within this thesis, as without their time and participation this PhD would not have been possible.

Abstract

Perseverative cognition (PC) is the repeated psychological activation of stress-related cognitions (including worry and rumination). The Perseverative Cognition Hypothesis (PCH) posits that, in the same way as stress, PC damages health via its ensuing physiological activation and, in recent years, PC has been shown to influence health via an indirect, behavioural pathway, termed the Extended PCH (EPCH). However, this evidence seldom considers experimental methodologies testing interventions, pathways, or how robust this is across health outcomes. The principal aim of this thesis was therefore to examine methods to reduce PC, its relationship with health behaviours/physical health outcomes, and moderating/mediating variables that may exacerbate and/or attenuate this relationship.

Both general and work-related worry and rumination predicted significantly higher scores in burnout and somatization, as well as lower scores in sleep quality while several mediation effects were found for the indirect pathway from job strain, through PC, to several health outcomes (Chapter 2; Study 1). Meta-analysis of 36 studies testing (non-pharmacological) interventions produced medium effect sizes for worry and rumination, corresponding to small, but positive, effect sizes for health behaviours (and small-medium positive effect sizes for sleep) (Chapter 3; Study 2). In a randomised controlled trial, participants in an augmented worry postponement intervention produced significantly lower worry duration (by ~15 minutes, on average, per day), relative to an active-control arm and those in the augmented worry postponement condition reported significantly shorter worry duration and lower worry frequency, relative to the standard arm. Neither of the interventions had any effect on sleep (Chapter 4; Study 3). In another study, worry and rumination (at baseline) predicted significantly poorer sleep quality (at 7 days follow-up). Worry, but not rumination, and PBC interacted to predict significantly lower physical activity frequency and consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant (Chapter 5; Study 4).

The findings of this thesis provide some support for the EPCH and varying degrees of support for the PCH. PC poses a serious, indirect, risk for disease processes via modifying health behaviours and influencing some physical health outcomes. Further work is needed to elucidate how PC interacts with other components known to predict (or influence) disease processes and to uncover new interventions that can attenuate the now axiomatic relationship between PC and ill-health.

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List of Abbreviations

Abbreviation	Explanation
PC	Perseverative Cognition
PCH	PC Hypothesis
EPCH	Extended PC Hypothesis
HPA	Hypothalamic-pituitary-adrenal-axis
ANS	Autonomic Nervous System
HR	Heart Rate
BP	Blood Pressure
SNS	Sympathetic Nervous System
PNS	Parasympathetic Nervous System
HRV	Heart Rate Variability
TPB	Theory of Planned Behavior
AWP	Augmented Worry Postponement
WP	Worry Postponement
A-C	Active Control
NA-C	Non-Active Control

Chapter 1

Introduction: Background, Rationale, and Aims of the Thesis

1.1 Chapter Summary

This chapter will outline the theory that Perseverative Cognition (PC), the cognitive representation of past stressful events (rumination) or feared future events (worry), mediates the relationship between stress and physical disease; both directly via prolonging the hypothalamic-pituitary-adrenal-axis stress response, and indirectly by influencing negative health behaviours. To demonstrate this, the theory will be discussed within the broader context of the stress literature, specifically touching on evidence linking PC to ill-health. To conclude the chapter, the aims of the thesis are summarised, and the content of the ensuing chapters are outlined.

1.2 Stress and Physical Health

There is a vast cumulative science connecting stress to reduced health status (O'Connor, Thayer & Vedhara, 2021). Stress can impede a range of key biological mechanisms and, when maintained, can lead to organic disease and ultimately premature mortality. There are decades of supporting evidence for the relationship between stress and physical health and an array of theoretical arguments have now been put forward for how and why this link exists. However, the evidence base demonstrating the stress-ill-health relationship, for the purposes of this thesis, will be broken down into three key categories: endocrine responses (Adam et al. 2017, Clow et al. 2010, Fries et al. 2009, Pruessner et al. 1997), autonomic nervous system (ANS) regulation dynamics (Beauchaine & Thayer, 2015; Benarroch, 2008; Thrasher, 2006), and most recently, human genome advances (Cole et al., 2007; Miller et al., 2014). Research on each of these distinct, yet interrelated physiological processes, provides some of the most compelling and contemporary evidence for the role of stress as a damaging precursor in the development of organic disease.

Early work by Walter Cannon termed the acute physiological changes in the autonomic and sympathetic nervous systems that occur when an animal is exposed to an external threat, which he described as the 'fight or flight response' (Cannon, 1939). When stress is perceived, in the short-term, the sympathetic-adrenomedullary axis (SNS) is activated and, if stress persists, the hypothalamic-pituitary-adrenocortical (HPA) axis is activated. Research on cortisol, the primary effector hormone of the HPA, offers some of the most robust evidence in the stress-disease relationship. Similar to other aspects of the endocrine system, the HPA axis is controlled by a negative feedback system, whereby the hypothalamus and the pituitary gland have receptors that detect changes in cortisol levels. For instance, cortisol secretion is inhibited when circulating levels rise and is stimulated when levels fall. But, if the HPA axis is repeatedly activated, greater cortisol is generated, thereby exposing bodily tissues to excessive concentrations of the hormone (Lovallo 2016; Miller et al. 2007). Over time, such repetitive activation can lead to tissue damage and prospective ill-health via placing a disproportionate degree of strain on various bodily systems, such as the HPA axis and cortisol regulation (c.f., 'allostatic load', McEwan, 1998).

A large body of science has examined the relative impact of a heightened cortisol response following – and in anticipation of – stress within the context of health outcomes (for review, see Staufenbiel et al., 2013). Cardiovascular reactivity to stress was among the first systems to be empirically investigated and was heavily underpinned by the Obrist (1981) reactivity hypothesis; conceptualizing that people who exhibit the largest increases in blood pressure (BP) or heart rate (HR) in response to acute stressors are at greatest risk of future ill-health. A number of seminal papers in favour of this theory revealed that heightened cortisol reactivity to stress is associated with negative health outcomes (e.g., al'Absi & Wittmers 2003; Hamer & Steptoe 2012; Hamer et al. 2012). In a 3-year prospective study of the Whitehall II cohort, Hamerand Steptoe (2012) discovered a 59% increase in the odds of incident hypertension (per standard deviation change) in cortisol responsivity to a stressor; while a longitudinal analysis of the same participants evidenced the long-term impact of

these endocrine changes, as coronary artery calcification was also present some 3 years later (Hamer et al. 2012). al'Absi and Wittmers (2003) also found evidence that enhanced HPA activity in response to an acute stressor was (cross-sectionally) associated with risk of hypertension, while Hamer et al. (2010) showed increased reactivity to a stressor was associated with coronary artery calcification (a marker of subclinical coronary atherosclerosis).

There is also evidence to suggest that a blunted cortisol response to acute stress, that is a smaller incremental change, may also be a determinant of reduced health status (Lovallo, 2006) or pose greater health risk through adverse behaviour change (Roseboom et al., 2006). O'Connor et al. (2017), for instance, showed that individuals who had previously made a suicide attempt exhibited low levels of cortisol in response to an acute stressor (compared to control participants). A recent meta-analysis comprising 4292 individuals reported robust associations between stress (through early-life adversity) and a blunted cortisol response (Bunea et al. 2017). There is also a vast body of evidence for the role of the ANS within the stress and health relationship. The SNS, linked with energy mobilization and the fight-or-flight response, and the parasympathetic nervous system (PNS), related with the restorative functionality reflect the two major arms of the ANS. Normal functioning of these systems results in a dynamic balance but with the PNS taking the leading role; however, under conditions of stress, an imbalance takes hold placing the body in 'fight or flight' mode which can lead to excessive wear and tear of the bodies physiological systems (see Cannon, 1939). Here, it is important to note the key role the baroflex plays in maintaining BP at nearly constant levels. Arteries housing the carotid and aortic arches contain pressure sensitive receptors (which regulate blood flow). These signal to the brain to create alterations in BP (i.e., either increases or decreases depending on the environment) via the regulation of sympathetic and parasympathetic outflows in order to maintain blood flow to vital organs (i.e., the brain and heart; Benarroch 2008). Therefore, ANS activity governed by peripheral vascular resistance, myocardial contractility, HR, and works to

regulate BP via the baroreflex and, in terms of how this can be implicated in long-term health outcomes, there is promising evidence showing the baroreflex is also responsible long-term BP regulation (Thrasher 2006).

This process, under or in anticipation of stress, can be a life saver. It serves to help us evade and escape from immediate danger to maintain homeostasis (see Lerner, 1964). However, when this autonomic imbalance is maintained over time its health penalties can be vast and enduring. On a theoretical level, Sterling and Eyer (1988) termed this as 'allostasis', before McEwen (1998) argued that the adaptive nature of allostasis comes at a cost, known as 'allostatic load'. McEwen (1998) describes allostatic load as physiological wear and tear on the body which occurs when the stress systems are chronically activated. For example, one of the first studies to investigate the relationship between indices of heart rate variability (HRV) and mortality in humans, Kleiger et al. (1987) showed across 900 patients that HRV was a significant independent predictor of mortality. Thus, together, this evidence base is sufficient to conclude that autonomic imbalance, through the ANS system, may be a final common pathway to increased morbidity and mortality from a host of conditions and diseases.

Thirdly, advances in technology have allowed stress researchers to develop our understanding in how the human genome is implicated in downstream health outcomes. Emergent developments in Psychoneuroendocrinology have associated exposure to stress with DNA damage and accelerated telomere shortening, which crucially is thought to be a main predictor of human life expectancy, thus enhancing the possibility that chronic stress may impact biological aging pathways, ultimately increasing risk for age-related diseases (Cole, 2019). For example, Cole (2007) showed thar the immune cells of chronically lonely individuals were characterised by an upregulation of pro-inflammatory genes, and the downregulation of genes associated with antiviral resistance and antibody production. In other words, the genes associated with increasing the risk of or exacerbating inflammation-related conditions were more likely to be switched on, and those associated with protection

from viral illness were more likely to be switched off – should that person having been reported higher than average levels of social isolation. Remarkably, these findings reflect the first set of evidence for the molecular explanation of how stress can increase the risk of disease.

Further, the molecular fields of study concerning cellular aging and cellular senescence represent exciting modes of research through which we can learn more about the causal pathway between stress and health. For example, in a daily-diary study of parents, Rentscher et al. (2019) revealed parents with higher chronic stress exposure in the six months prior to study entry showed elevated $p16^{INK4a}$ expression; a robust indicator of cellular senescence (see, Da Silva-Álvarez et al., 2016). Moreover, parents who perceived their lives as more stressful in the week prior to study entry and reported a greater percentage of stressful days over the 8-week diary period also showed elevations in $p16^{INK4a}$ expression and, crucially, global perceptions of stress—including feeling stressed, upset, and unable to control important things in life —was most robustly associated with $p16^{INK4a}$ mRNA levels. Together, these data suggest that people who are exposed to higher levels of chronic stress or perceive their life experiences as more stressful may show signs of accelerated biological aging in mid-life that have not yet manifested into disease process but may place them on the trajectory.

Therefore, to summarise, stress can impact physical health in a variety of ways. The evidence discussed thus far serves to document how the autonomic imbalance attached to the stress response can damage health if not properly moderated by placing excessive pressure on various bodily systems, chiefly the HPA axis (i.e., allostatic load and overload; McEwen 1998). However, there is also evidence for an association between stress and a reduced health status via an indirect pathway; health behaviours, which will be considered in the next section.

1.3 Stress and Health Behaviours

In addition to directly impacting our health through neuroendocrine responses it is also now relatively well-established that stress can influence our health through a more indirect pathway, via influencing how we behave. A variety of work now exists exploring how we behave in response to, and in anticipation of, stressors and stressful events (e.g., Hill et al., O'Connor et al., 2008). The central tenet to this line of enquiry proposes that in the same way stress can damage health through continual HPA activation, it can also damage health via contributing to obesity, cardiovascular disease, and cancer risk by influencing health behaviours (O'Connor & Connor, 2011). This is because, much like the disease outcomes owing to the physiological response stress elicits when the HPA axis is continually activated, or when one's cortisol response is blunted, stress-induced eating, alcohol consumption and stress-related sleep disturbances (when excessive or prolonged) can result in the same outcome. Put simply, stress may also influence health by promoting unhealthy behaviours that if maintained overtime may have an adverse effect on health outcomes. This next section unpacks this potential pathway.

It is first important to make the distinction between harmful and healthy behaviours. Some behaviours, such as smoking, are harmful to health while others, such as exercise, may promote health. A number of health behaviours, including smoking, alcohol consumption, (non) exercise, and overeating are associated with morbidity, disability, and mortality (US Department of Health and Human Services, 2000). It is therefore unsurprising that stress is associated with higher fat diets (Laitinen, Ek, & Sovio, 2002) and greater fast food consumption (Steptoe, Lipsey, & Wardle, 1998); higher levels of smoking and reduced probability of smoking cessation (House, Strecher, Metzner, & Robins, 1986; Steptoe, Wardle, Pollard, Canaan, & Davies, 1996); increased alcohol consumption (House et al., 1986; Steptoe et al., 1998); and lower levels of physical activity (Kivela & Pahkala, 1991; Steptoe et al., 1998). Thus, stress can hinder health to the extent that it produces deleterious changes in and/or helps maintain unhealthy behaviours such as those outlined above. There

is now evidence for the maladaptive effects of stress on a variety of health behaviours, which is outlined next.

One of the first health behaviours to be empirically examined in respect to stress and health was eatingbehaviours. Folkman and Lazarus (1980) provided one of the earliest explanations for why stress may lead to unhealthier behaviours, placing focus on eating's utility as a 'coping response'. Stress appears to alter overall food intake in two ways, resulting in under- or overeating, which may be influenced by the type of stress experienced by the individual (Umberson & Reczek, 2008; O'Connor & Conner, 2011). Acute stressors, that exist in the environment for a defined period, are likely to trigger a different response to those chronic life stressors which hold a more permanent appraisal role. It is thought this is because when exposed to an acute stressor the instant 'fight or flight' response leads to the suppression of appetite (Hayward & Gorman, 2004), but that when an individual is continually exposed to more chronic stressors, they may cause longer-term changes in dietary choices which contribute to the onset of overweight and obesity (George, 2003). Sominsky and Spencer (2014) provide a biological account for the former, suggesting that, after the initial appetite suppressing effect in response to acute stress, chronic elevation of glucocorticoids increases appetite via several appetite-regulating hormones and neurotransmitters. Whereas, for sustained stress, several explanations exist pointing to the context of stress as an ego-threatening, physical and inter-personal source that can influence eating behaviour. Ego-threatening, interpersonal are known to be associated with increased snacking, whereas, physical stressors are known to be associated with decreased snacking (O'Connor et al., 2008). Specifically, stressors of an ego-threatening nature (e.g. where there is a fear of failure) have distinct effects from those that elicit physical threat (e.g. fear of an electric shock). Heatherton et al. (1992) suggests that situations involving potential negative evaluation or task failure (ego-threats) will lead to disinhibition (over-eating) in restrained eaters (i.e., those attempting to control their food intake) or current dieters whereas physically threatening situations will not. Thus, the type – and context – of the

stressor an individual is exposed to is a likely causal factor as to the immediate effect it has on the bodily systems and subsequent health behaviours.

A retrospective survey of United States Marines' food intake during combat provided an opportunity to examine the effect of acute stress on eating behaviour (Popper et al., 1989). Sixty-eight percent of marines reported eating less than usual during their first day of training; with the main reasons being stress-related, such as fear, feeling nervous, tense, and scared. In a separate study, 158 individuals completed daily records of stress, reporting significant fluctuations in eating behaviour; wherein individuals were more likely to eat less in accordance with the severity of the stressor they were experiencing (Stone & Brownwell, 1994). Self-reported stress has also been explored in undergraduate students around exam periods, with total energy intake being significantly greater on the examination day when compared with the stress-free day (2225 versus 2074 calories), respectively (Grunberg & Straub, 1992). While experimental evidence shows that in the presence of a mild stressor (i.e., a stress-inducing film) stressed males consumed significantly less food compared with the male control group (99 versus 242 calories), respectively (Michaud et al., 1990). Therefore, there are clear signs that exposure to acute, short-term stress can cause unhealthy deviations in eating behaviour. However, these studies only concern exposure to acute stressors either in naturalistic setting or in the laboratory and do not account for the long-term impact stress may have when an individual is exposed to a stressor over the longer term.

The evidence base showing the degree to which both chronic stressors (i.e., consistent/overwhelming over time) and daily hassles (i.e., everyday minor stressors that are minimally stressful, frustrating, or irritating) can influence health behaviours is beginning to accumulate. For example, early studies show stress to be associated with a greater preference for energy- and nutrient-dense foods, such as those that are high in sugar and fat (Greeno & Wing, 1994) and evidence from longitudinal studies suggests that chronic life stress may be causally linked to weight gain (Geiker et al., 2018; Steptoe & Wardle, 2005).

Whereas, for daily hassles, a recent meta-analysis revealed patterns between stress and unhealthy eating in both children and adolescents; notably, in older children, stress was found to be associated with a decrease in unhealthy eating behaviour (Hill, Moss, Sykes-Muskett, Conner, & O'Connor, 2018). Daily stress has also been associated to greater instances of between-meal snacking (Newman, O'Connor, & Conner, 2007) and in a diary study to the increased consumption of high fat and high sugar between-meal snack foods in addition to a reduction in vegetable consumption (O'Connor, Jones, Conner, McMillan & Ferguson, 2008). There are also neuroendocrine correlates of stress-induced eating. High cortisol reactivity assessed in the lab, for example, has been shown to be a significant predictor of greater stress related eating in the field (for review, see Hill et al., 2022).

Furthermore, a number of other studies exist showing the indirect effects of stress on other health behaviours. Pearlin and Radabaugh (1976) were among the first to study alcohol consumption as a way of coping with stress. Their cross-sectional analysis provided support for the theory that individuals use alcohol consumption as a way of coping with economic strain; particularly when individuals are high on anxiety and low on mastery and self-esteem. A decade later, House and colleagues (1986) analysed prospective data from a community survey and found that certain types of work stress were positively associated with subsequent alcohol consumption and also smoking. Since then, a multitude of studies have been published depicting the same harmful trend. Niaura, Shadel, Britt and Abrams (2002) reported some of the first evidence that subjective affective and efficacy responses during a stressful social encounter were associated with smoking urges. Meanwhile, cross-sectional from Ensel and Lin (2004) data shows us that physical fitness is both a buffer and a mediator in the relationship between stress and distress (psychological distress and physical symptoms). Ensel and Lin (2004) conclude that "we need to extend the model to include (other) health behaviours" and to "determine the extent of subgroup differences in the effects of fitness in the life stress paradigm" (p. 97).

Sleep is also a key health outcome to consider. An early study into how stress may influence sleep quality unearthed the prospect that stress is intrinsically linked to circadian rhythms, much empirical attention has been paid to this area (Van Reeth et al., 2000). Chiefly, it is thought the underpinning link between stress and sleep quality is caused by the inhibition of the HPA-axis during the early stages of sleep (see, Born & Fehm, 1998), which is echoed by the fact subsequent studies have shown sleep onset is associated with decreasing cortisol levels (Weibel, Follenius, Spiegel, Ehrhart, & Brandenberger, 1995). Thus, it would follow that stress impairs sleep and, owing to technological advances, evidence now exists associating psychosocial stressors to sleep using the gold-standard of sleep measurement, polysomnography (Kim & Dimsdale, 2007; Singh et al., 2022). Acute experimental stressors were significant predictors of increased sleep onset latency, more awakenings and decreased sleep efficiency. Moreover, a recent meta-analysis of 3,733 participants (k =8) showed the damaging roles of worry and rumination in the context of stress and sleep outcomes. A meta-structural equation modelling analysis reported a significant component effect of perceived stress on worry/rumination, which in turn was related to sleep disturbance. The direct effect of stress on sleep disturbance was significant and the indirect effect between stress and sleep disturbance via worry/rumination supported full mediation hypothesis, even controlling for sex and age, respectively (Zagaria et al., 2022). In other words, worry/rumination may be one of the mechanisms explaining how perceived stressful experiences lead to sleep disturbance.

The aforementioned evidence merely represents a snapshot of the literature associating stress, and its mediating mechanisms, to unhealthier behaviours. Though, what is immediately apparent is that there is a clear association between markers of psychological stress and a variety of behaviours that are known to influence or impact physical health. Together with the previous section, the cumulative science now directly associating stress with adverse physical health outcomes, as well as indirectly via health behaviours, is vast and continually growing. The sections to follow will build upon this view, noting how recent

advancements in the stress literature have highlighted a new vehicle through which stress can impact health, remarkably, even long after its physical or psychological presence has dissipated.

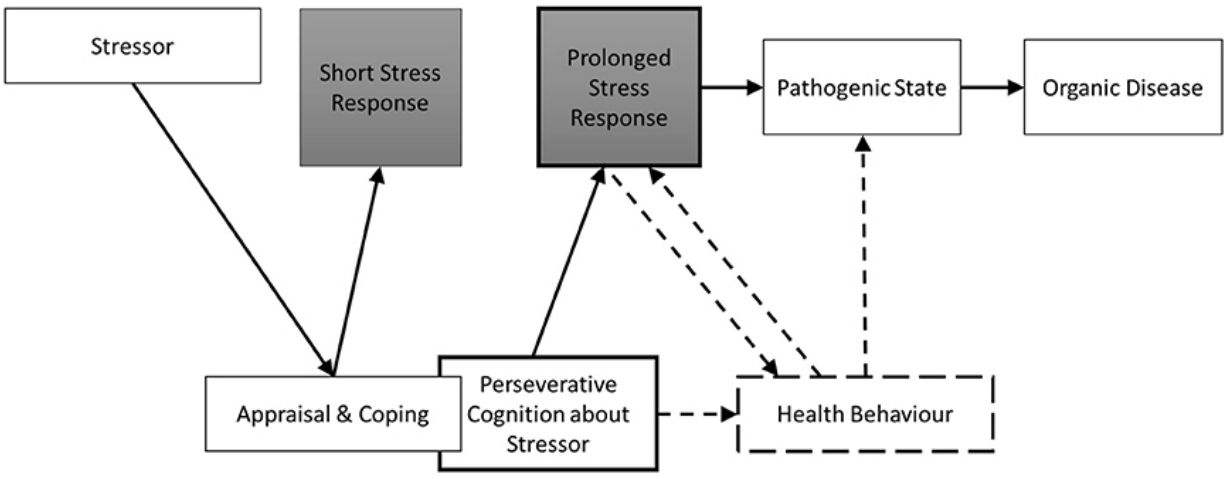
1.4 The Perseverative Cognition Hypothesis

Lazarus and Folkman (1984)'s seminal work proposes that stress represents the unique interplay between environmental events and the individual's perception of these events as threatening. In other words, a mismatch between an individual's appraisal of a stressor and their perceived ability to cope, will dictate the extent to which they experience the symptoms of stress. Using this notion, in 2004, Jos Brosschot and Julian Thayer published a book chapter in *Emotional Expression and Health*, whereby they emphasised the importance of cognitive-level representations of past and future stressors; specifically focussing on the variable of worry, implying that they play a much broader role as a mediating factor between stress and health (Brosschot & Thayer, 2004). This was significant as, until then, worry had mostly only been considered as a central component of generalized anxiety disorder, so the view that long lasting or persistent worrying may reactivate the cognitive schemata associated to stress was viewed as a compelling school of thought among psychologists.

Evidence was then presented a year later by Pieper and Brosschot (2005) showing, for the first time, that negative perseverative cognition (PC) prolonged both discrete stress as well as negative emotional episodes and that, crucially, these were related to continued periods of cardiovascular activity. Pieper and Brosschot (2005) argued that these finding highlighted the importance of psychological mediators, such as worries, that may sustain the physiological concomitants of the stress response long after its onset. Then, in 2006, a landmark paper in The *Journal of Psychosomatic Research* detailed how stress theory had omitted the role of anticipation and recovery from stress, accompanied with data showing that cognitive-level representations of past and future stressors are associated with physiological activation, including cardiovascular, endocrinological and immunological

parameters (Brosschot, Gerin & Thayer, 2006). They described PC as any type of stress-related negative, repetitive thought. Importantly, the term encompasses thoughts about feared future events (worry) and thoughts about distressing past experiences or current negative feelings (rumination). The authors described this phenomenon as the 'Perseverative Cognition Hypothesis' (PCH; see Figure 1.1), stating that worry and/or rumination activate the body's stress response in the same way as stressors in the physical environment and serve to prolong the HPA-axis stress response. It would therefore follow that when stress is perseverated upon, the damaging physiological activation associated with stress is also protracted, thus increasing susceptibility to stress-related ill-health; which, interestingly, is also consistent with the McEwen (1998) conceptualization on allostatic load. Crucially, these advancements in the stress theory suggested that PC may serve as a key mediator in the stress-disease relationship; going as far to argue that it may reflect the final pathway through which stress impacts health (Brosschot et al., 2006).

Figure 1.1. The PC Hypothesis (Brosschot et al., 2006).



1.5 Conceptualising PC: The role of worry and rumination

While the conceptualisation of PC presented in the original PCH (Brosschot et al., 2006) is an umbrella term which encompasses any type of negative or repetitive thought process, worry and rumination are by far the most empirically examined. Within the context of PC, worry is thought to encompass thoughts about feared future events, while rumination is said

to be experienced through thoughts and negative feelings about distressing past experiences (Watkins, Moulds, & Mackintosh, 2005). Thus, there is clear temporal distinction between these distinct, yet closely related, constructs that defines their function as a vehicle for stress. The following section will explore this further, defining worry and rumination, as well as considering their conceptual merits under the framework of the PCH.

It has frequently been noted that a comprehensive psychological model is obligated to account for all pathogenic pathways that cause disease (Lazarus & Cohen, 1977); which is significant as, until the late 1980's, psychological research has neglected the role worry may play as a risk-factor influencing somatic health outcomes. This is surprising given that worry – a chain of negatively uncontrollable affect-laden thoughts or images (Borkovec et al., 1983) – is an extremely common response to stressful events and a central feature in several mood disorders including generalised anxiety disorder (Wells & Carter, 2001), major depressive disorder (Arditte-Hall, Quinn, Vanderlind & Joormann, 2019) and bipolar disorder (Popolo et al., 2017). It is also a cardinal feature of the DSM-IV (American Psychological Association, 2021) and is thought it be also initiated by involuntary intrusive thought, sparking worrisome episodes cantered around 'What if…?'. Thus, when Brosschot et al. (2006) reflected upon these writings in 2006, they highlighted the role of worry among fear and anxiety processes and emphasized that worry may also serve as a problem-solving mechanism; although its efficacy is likely flawed due to efforts that are counter-productive. This is evidenced by reports associating worry to self-doubts concerning one's problem-solving skills and the tendency to be pessimistic about the outcome of a stressful event (Robichaud & Dugas, 2005). Therefore, as the total time (or intensity) of worry over stressful events increases, it would follow that so does the total duration of PC and its associated physiological activation.

Rumination was considered before worry within the context of PC and stress reactivity research. It is a common response to negative mood (Rippere, 1977) and is a salient feature cognitive component of dysphoria (American Psychological Association, 2021). Like worry,

rumination may initially be activated as a response to an intrusive thought, but its function is generally more perpetuated by both imaginative and verbal questions of 'Why?', rather than the 'What if?' associated to worry (Wells & Matthews, 1994). On the back of seminal work by Nolen-Hoeksema (1991) concerning rumination's role in depression, and Martin and Tessers' (1996) model of ruminative thought, numerous experimental and correlational studies have shown that rumination is a deeply pervasive cognitive-process. Indeed, it predicts greater depressive symptoms, the onset of major depressive episodes (e.g., Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Wood, Saltzberg, Neale, Stone, & Rachmiel, 1990). These early studies sparked an array of psychological enquiry into rumination, leading to several working definitions of the construct but, by far, the most accepted and used in the literature is that rumination is a method of coping with negative mood that involves self-focused attention on past events (Lyubomirsky & Nolen-Hoeksema, 1993). According to Response Styles Theory (Nolen-Hoeksema, 1987), it is characterized by self-reflection (Morrow & Nolen-Hoeksema, 1990) as well as a repetitive and passive focus on one's negative emotions (Nolen-Hoeksema, 1991, 2000; Nolen-Hoeksema, Parker, & Larson, 1994). Further, more recent developments also place rumination as an emotion regulation strategy, helping with problem solving and goal attainment, or aiding individuals in the processing of traumatic events (Smith & Alloy, 2009; Watkins & Roberts, 2020).

Importantly, factor analysis of rumination has identified sub-factors within the construct. Brooding is a passive and judgemental form of rumination, where reflection is more contemplative with a focus on problem-solving. Treynor et al. (2003) provided evidence that brooding is the more maladaptive component of rumination, given it has been shown to predict symptoms of depression longitudinally; whereas, although reflection has been shown to precede current depression, it predicted relatively lower levels of depression over time. Equally, later studies have shown that brooding is a stronger predictor of other markers of depression, such as suicide ideation, with individuals reporting significantly higher scores in

brooding at follow-up (relative to reflection). However, in the same study, both brooding and reflection did predict whether an individual thought about suicide at 1-year follow-up, even after adjusting for baseline suicidal ideation (Miranda & Nolen-Hoeksema, 2007), and given that these variables also correlate highly with one another, this also suggests they share a common purpose underpinning the construct of rumination (Segerstrom, Stanton, Alden, & Shortridge, 2003). Together, alongside worry, they represent a key dimension of the PCH, as when individuals engage in ruminative thought, studies have shown it also prolongs the physiological activation owing to the stress response, as well as influencing key health behaviours (Brosschot et al., 2007).

Despite the unity in how these sub-components of PC are theorized there is, however, several different methods through which psychologists measure both worry and rumination, which the section to follow will serve to unveil. Importantly, the relative contribution of worry and rumination to the PCH will be central to this thesis, in that while conceptually similar, worry and rumination may play distinctly different mediatory roles as transdiagnostic risk factors to health.

1.6 Measuring PC

Efforts to distinguish worry from the other related cognitive mechanisms that make up intrusive thought has led to the development of a number of assessment tools. The Penn State Worry Questionnaire (PSWQ) (Meyer, Miller, Metzger, & Borkovec, 1990), for example, is a reliable and valid measure of worry (van Rijsoort, Emmelkamp, & Vervaeke, 1999), which has been extensively used to capture levels of trait worry (see, Verkuil, Brosschot, Gebhardt, & Thayer, 2010). According to Molina and Borkovec (1994), who were pioneers in the early assessment of worry, the PSWQ's key strength is its ability to evaluate the excessiveness and intensity of worry both in pathological individuals and also in a more general sense. During psychometric development, the measure was completed by 337 college students and subject to oblique rotation, resulting in one main factor (accounting for

22.6% of the variance) relating to the excessiveness/uncontrollability of worry. Despite the PSWQ's universal application within worry research other measures do exist. Another popular measure is The Worry Domains Questionnaire (WDQ; Tallis, Davey & Bond, 1992) which focusses on considering clusters of worry content types and was based on the theoretical grounds that worry-related material is likely stored in memory (Eysenck, 1984). It concerns six key areas of worry: relationships; lack of confidence; aimless future; work incompetence; financial; and socio-political. The scale is also highly intercorrelated with the PSWQ (r = .68), presumably because the measures are tapping into highly related though conceptually distinct components of the same construct. The PSWQ has been found to be more predictive of daily worry duration than the WDQ (Verkuil et al., 2007) suggesting that this questionnaire is more useful in measuring the underlying construct of worry. There are also a variety of other questionnaires available to test worry; namely, The Anxious Thoughts Inventory (Wells, 1994), The Why Worry Questionnaire (Freeston et al., 1994) and the Consequences of Worrying Scale (Davey, Tallis & Capuzzo, 1996), albeit these are less used and lack the robust empirical validation that the PSWQ and WDQ obtain.

The most frequently used measure of rumination is related to ruminative response styles: the Ruminative Response Scale (RSS; Nolen-Hoeksema & Morrow, 1991). The RRS instructs participants to indicate how frequently they engage in a number of thoughts, feelings, and behaviours people commonly engage in when feeling down, sad, or depressed. In a landmark prospective study, Nolen-Hoeksema and Morrow (1991) examined the effect of response styles on depression after exposure to a major stressor in the form of an earthquake. They found that participants who had comparatively higher scores in ruminative response style prior to the earthquake, and who had more quake-related stress, had elevated depression scores 10 days following the event. Since then, several studies have examined the factor structure of the RRS and extracted factors concerned with critical self-focus and introspection (Bagby & Parker, 2001; Cox, Enns, & Taylor, 2001). However, given the RRS has been criticized on the grounds of its alleged association with depression, later,

Treynor, Gonzalez, and Nolen-Hoeksema (2003) developed an instrument of rumination independent from its indexing with depression. Their efforts resulted in two factors being extracted: brooding and reflection. Importantly, brooding emerged as the more maladaptive part of rumination, showing a stronger cross-sectional and predictive association with depressive and anxious symptoms, relative to reflection (Armey et al., 2009; Burwell & Shirk, 2007; Treynor et al., 2003).

The overlapping nature of rumination, reflection, and worry has led to some arguing they culminate collectively in a shared variable termed 'repetitive thought'; which is defined as prolonged cognitive focus which can be directed on the domains of self, emotions or past or future life events (Segerstrom et al., 2003). Around the same time, Segerstrom et al. (2002) developed a four-factor solution for repetitive thought, consisting of two worry and two rumination factors that also share equal strength with anxiety and depression (Fresco et al., 2002). Worry engagement and dwelling on negative factors correlated highly, so in this sense, repetitive thought serves a similar function to what is hypothesized in the PCH, although it also holds a place for adaptive thoughts. Notably, Segerstrom et al. (2003) consider worry, rumination and depressive rumination as maladaptive categories of repetitive thought. Using multidimensional scaling, the authors suggested that repetitive thought varies along at least two dimensions: content valence (the degree to which the thought is positive or negative) and the purpose of the thought (searching for perspective or understanding versus preparation and problem-solving), which are likely to be important determinants of wellbeing.

There has also been a great deal of investigation into repetitive negative thinking, which more closely resembles the definition of PC proposed by Brosschot et al. (2006) than the broader definition of repetitive thoughts (i.e., excluding positive ones) offered by Segerstrom et al. (2003). Ehring et al. (2011), for example, suggests that common measures of worry and rumination are too specific and that a wider construct is required. The authors suggested that a looser definition of repetitive negative thinking is required and that this

exploration should not be subject to a temporal focus or incorporate any consideration of the thought content, but rather focus on ones difficulty in disengaging from them. Based on this, the Perseverative Thinking Questionnaire (PTQ) was developed, consisting of three high-order factors: repetitiveness, intrusiveness and difficulty to disengage. Interestingly, scores on this measure correlate highly with the PSWQ (r = .70) and the brooding subscale of the RSQ (r = .63), suggesting these efforts to classify the common components of repetitive thinking align most closely with the factors set out in the PCH: worry, brooding and reflection (Devynck et al., 2017).

The psychometric tools discussed thus far aimed at capturing both worry and rumination, in both a non-clinical and pathological sense, are concerned with trait-level measures but there have also been efforts to measure these constructs at a state level. For context, Ottaviani et al. (2016) assessed whether outcomes differed depending upon whether PC was measured at a state or trait level (i.e., the difference between engaging in worry/rumination at a point in time versus the overall tendency to engage in it). They found that this covariate moderated the association between PC and HR and HRV and significant associations between PC and HRV was also found in studies assessing state, as opposed to trait worry and rumination. Therefore, there is clear merit in measures entirely devoted to assessing daily fluctuations in both worry and rumination. Takano and Tanno (2011), for example, designed a measure of state rumination which has been shown to correlate with levels of trait rumination. In addition, Cropley, Michalianou, Pravettoni, and Millward (2012) initially developed a trait measure of rumination specifically related to post-work ruminative thinking; however, it was later adapted and successfully employed at a state level (Cropley, Rydstedt, Devereux, & Middleton, 2015). That said, this is an ever-evolving area which is reflected by the fact a scarcity of measures exist to test state levels of worry. One exception to this is the 4-item Stress Arousal Scale (Smith & Everly, 2012), while others have leant on the superiority of the PSWQ and amended it for weekly assessments of daily worry levels during intervention (e.g., Stober & Bittencourt, 1998). Tools for assessing both trait and state levels of worry and

rumination clearly do exist and are highly cited within the associated literature; however, it does highlight that there is less research on state measures of PC in general suggesting further empirical testing would be beneficial in this area.

In sum, worry and rumination are constructs that have attracted much research attention in recent years, leading to an array of assessment tools that measure PC holistically as well as considering its individual facets (e.g., reflection, brooding, worry). It does, however, demonstrate that PC is a complex term and this is reflected by the numerous different measures and definitions within the literature. Importantly, despite this, the balance of evidence now shows that PC mediates the association between stress and disease. The next section will outline the evidence underpinning this in relation to physical health outcomes, before moving onto the pervasive indirect effect PC can have on health behaviours in the section that follows.

1.7 PC and Physical Health

An abundance of evidence has now amassed showing the utility of both worry and rumination as mediators in the stress-disease relationship, which the following two sections will serve to document. Evidence will be presented to show that worry and rumination have physiological sequelae that can lead to long-term health consequences, before considering the etiological role they play in the build up to somatic disease, via indirectly influencing health behaviours.

In the original PCH, Brosschot et al. (2006) suggested that stressful thoughts activate the body's stress response in the same way as stressors in the physical environment and serve to prolong the hypothalamic-pituitary-adrenal-axis stress response. This has now been supported by several important reviews encompassing a range of study designs and health outcomes. The first was a systematic review by Verkuil, Brosschot, Gebhardt and Thayer (2010). Across 64 studies the authors showed engagement in PC leads to: increased cardiovascular activity; reduced secretion of antibody productions; a blunted cortisol

response; and, increased somatization. Ottaviani et al. (2016), in a more recent meta-analysis, found large-sized associations between PC and a range of physiological health markers, including higher systolic and diastolic BP, increased heart and lower HRV and higher cortisol levels. Ottaviani (2018), then, provided morphometric evidence that PC about stress alters primitive functions via diminishing prefrontal-amygdala functional connectivity; these effects were maintained when controlling for different populations (i.e., healthy, generalized anxiety disorder), locations (i.e., United States, Europe) and age groups (i.e., adults, children).

Aside from these seminal reviews, there is also other evidence that ought to be more closely considered about the physical health consequences of PC. For instance, state worry duration has been found to prospectively predict somatic health complaints and has been shown to be sensitive to worry reduction interventions (Brosschot & van der Doef, 2006). Glynn, Christenfeld and Gerin (2002) discovered, in two experimental studies, that ruminative tasks were associated with delayed BP recovery and elevations during later rumination, and that, in a separate study, participants presented with a distraction during ruminative periods showed quicker BP recovery. Further, studies have evidenced a link between state (experimentally induced) and trait worry and chronic HR elevation (Palatini & Julius, 1997) and reduced HRV (Tsuji et al., 1994), while Brosschot et al. (2006) revealed that trait worry predicted a second myocardial infarction; which is significant given these are established risk-factors for mortality (Kubzansky et al., 1997). Rumination has also been associated to slower HR recovery after stress (Roger & Jamieson, 1988) and it has also been found that individuals high in angry rumination have higher levels of resting systolic BP (Chambers & Davidson, 2000).

There is also new evidence from neuroimaging studies that episodes of PC elucidate a series of neurobiological substrates across individuals with and without psychopathological conditions. Makovac et al. (2020) meta-analysed 43 neuroimaging studies showing that engagement in PC triggers cranial activity in the medial frontal gyrus, cingulate gyrus, insula,

and posterior cingulate cortex, which are associated with self-referential and affective processing. Thus, this objective neurophysiological evidence reveals key brain areas are activated when an individual engages in PC, stimulating (aberrant) cognitive control and embodied (autonomic) arousal that is typical to the activation of the HPA-axis (c.f., Pratt & Davison, 2009). Of equal weight, there is also evidence associating PC to endocrinological parameters. For example, associations have been found between higher trait rumination and higher morning cortisol levels (Schlotz, Hellhammer, Schulz, & Stone, 2004), as well as higher leukocyte count (independent of negative affect, Thomsen, Mehlsen, Hokland, et al., 2004). These findings, together, reflect the increased immunity in response to acute stress noted in section 1.2.

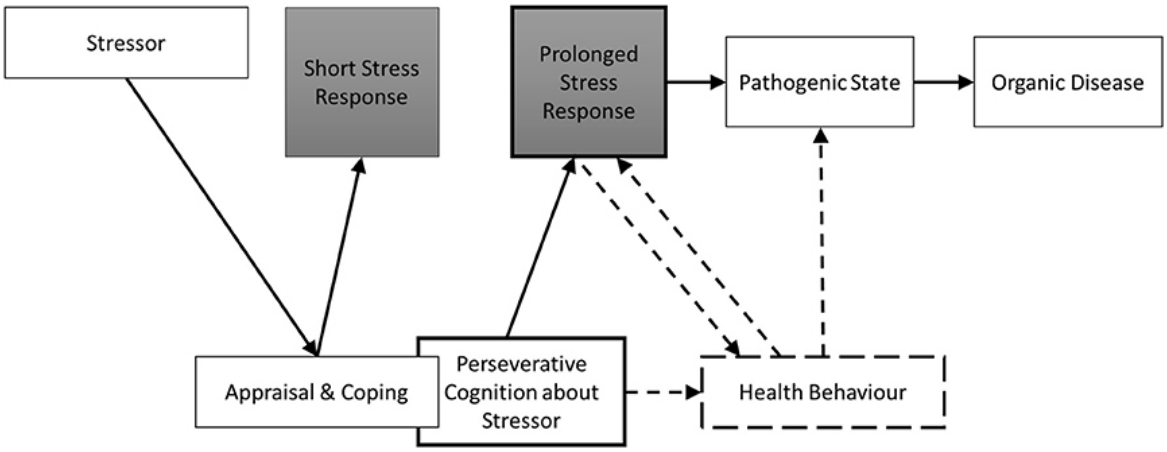
Thus, when considering this empirical landscape, several under researched areas remain within the stress and PC literature. A synthesis of interventional studies that aim to reduce PC and also impact physical health does not yet exist, for example, and further work is required to unpick the mediating/moderating role of worry and/or rumination play in influencing other physical health outcomes (i.e., burnout/somatization). However, this aside, the evidence presented thus far provides a clear message: that PC affects cardiovascular, autonomic, and endocrine nervous system activity, and that it serves as a mediatory pathway to long-term disease between stress and health vulnerability. The section to follow will uncover this relationship further and consider the weight of evidence underlying the damaging link between PC and ill-health through a range of maladaptive health behaviours.

1.8 PC and Health Behaviours

Within the broader stress literature there is support for the proposal that stress can affect health indirectly, through the modification of health behaviours (O'Connor, Thayer & Vedhara, 2021). It is proposed here that, in the same way as stress, there may be an additional indirect pathway between PC and health outcomes via health behaviours. The central tenet of this argument is that given the abundance of evidence associating PC to the

physiological parameters of the stress response, it is also likely that, in the same way the stressor is prolonged by worry or ruminative processes, so too may be its harmful impact on health behaviours. For example, PC might be more strongly associated with behaviours such as alcohol consumption and smoking, as well as limiting behaviours such a physical exercise and other wellness activities in an attempt to evade ruminative and worrisome episodes. Importantly, evidence is now growing to suggest this proposal is underpinned by several studies and datasets and has been recently termed the 'Extended PC Hypothesis' (EPCH; see Figure 1.2; Clancy et al., 2016). The following section will outline this new evidence and show that people who engage in greater bouts of PC are more prone to engage in significantly poorer health behaviours, and that when these are maintained over time, they may pose a critical health-risk.

Figure 1.2. The 'Extended PC Hypothesis' (EPCH; Clancy et al., 2016).



Firstly, it is imperative to differentiate between health promoting and health risk behaviours. Health-promoting behaviours are health-enhancing behaviours which individuals are encouraged to perform more to protect their health; whereas health risk behaviours are health-damaging behaviours which individuals are urged to perform less (Clancy et al., 2016). In the first study to assess the relative impact of PC on health behaviours, Clancy et al. (2016) meta-analysed 19 cross-sectional studies, hypothesizing – in line with the EPCH– that that higher levels of PC would be associated with more health risk behaviours (defined

as those behaviours which, if performed, would hinder health) as well as less health promoting behaviours (defined as those behaviours which, if performed, would benefit health). The random-effects meta-analysis revealed moderate, negative, associations between increased worry and rumination and health risk behaviours, but not for health promoting behaviours. Further analyses indicated that increases in rumination (r = 0.12), but not reflection (r = −0.08), or worry (r = 0.05), were associated with health risk behaviours. This new evidence thus provided partial support for the hypothesis; however, the associations were statistically weak, highlighting the need to identify new moderators in future studies. One route may be to explore how PC interacts with, or otherwise relates to, other cognitive processes to influence (health) behaviour. Equally, there is a need to consider the role of personality traits (i.e., neuroticism) within the context of perseverative cognition and health behaviours and how they may serve as protective/exacerbating factors

Given that, under the framework of the EPHC, it is theorised that rumination about past stressful events or worry about feared future events will mediate the effects of stressors on health behaviours, there is a great deal of other primary empirical evidence to consider spanning multiple health behaviours. This is key given the cross-sectional nature of the studies reviewed by Clancy et al. (2016). One of the key outcomes to be identified is sleep quality. Longitudinal evidence from Van Laetham et al. (2016) revealed that, in a sample of 44 Dutch PhD students who were followed-up during a two-month period, from one month before their public thesis defence (i.e., a stressful life event) and until one month thereafter, that levels of PC acted as a mediator between daily stress levels and a variety of sleep parameters. Higher levels of PC were significantly related to sleep efficiency, marginally to number of awakenings, and wake after sleep onset; thus highlighting the pervasive mediatory nature of PC in the stress-sleep relationship. Other studies also report the same trend: association between worry and rumination and difficulty falling asleep (McGowan, Behar, & Luhmann, 2016; Zoccola, Dickerson, & Lam, 2009), poorer quality sleep (Barclay & Gregory, 2010; Cropley et al., 2015) and shorter total sleep duration (Cropley, Dijk, &

Stanley, 2006; Nota & Coles, 2015) have all recently emerged. And although the precise causal mechanisms for these associations are still largely unknown, it is thought the physiological arousal known to predict sleep disturbance (Hall et al., 2007) combined with the physiological activation associated to episodes of PC, might be a sleep depriving factor in itself.

Furthermore, a later review and meta-analysis by Clancy et al. (2020) revealed some of the most compelling evidence yet between PC and adverse sleep outcomes. Random-effects meta-analysis of 55 cross-sectional and prospective studies found small-to medium-sized associations between higher PC and poorer sleep quality (r = −0.28), shorter total sleep time (r = −0.15) and longer sleep onset latency (r = −0.16). Interestingly, associations between sleep quality and rumination (r = −.33) were stronger than they were with worry (r = −.23). There is also evidence from other health behaviours worthy of note. PC has also been shown to amplify, prolong and reactivate the same physiological and psychological processes that account for the negative effects of stress on eating behaviour. Cropley et al. (2012) draws an association between rumination and the consumption of unhealthy foods such as cakes, crisps and confectionary, and some emerging work by Eschle and McCarrick (2021; 2022) has shown that state level worry and rumination may also prompt unhealthier snack choice. Another study has also found a link between worry and lower engagement in physical activity (Ferrer, Portnoy, & Klein, 2013), while others investigating alcohol consumption and smoking in response to PC have revealed that rumination is associated with more alcohol consumption on workdays (Frone, 2015); an association between emotional rumination and greater alcohol consumption (Aldridge-Gerry et al., 2011); and that high worriers were more likely to be current smokers than never before smokers (Rutten, Blake, Hesse, Augustson & Evans 2011).

Taken together, this evidence underpins the relationship between PC and health behaviours; such that those with high levels of worry and/or rumination may be more prone to engage in (more) health damaging and (less) health promoting behaviours. The evidence for the EPCH

is now growing, and while further study is required to ascertain the experimental evidence underlying PC and health behaviours, as well as the urgent need to target them via intervention, the weight of evidence accumulated thus far supports the view that health behaviours provide additional routes to pathogenic disease. The shortcomings of the aforementioned literature, specifically in reference to the lack of experimental evidence for the PC hypothesis and its sensitivity to interventions, the respective roles of worry and rumination play in determining ill-health, and their sensitivity to specific contexts, will serve as the central challenges to be considered in this thesis. The following section will outline these research themes in greater detail.

1.9 Thesis Aims

The evidence presented in this chapter has demonstrated the need for us to better understand the relationship between PC and physical/behavioural health outcomes. Evidence is accumulating to show that PC serves as a mediating mechanism between stress and disease, however several gaps remain, some of which this thesis aims to address within its four studies. First, little is yet known about the sensitivity of PC to environments marked by particularly high levels of stress (e.g., stressful work environments) and its harmful utility as a potential mediator in the stress-health relationship within these contexts. Thus, it is possible that PC, either in work or more generally, may mediate or moderate the relationship between job strain and physical disease. Consequently, the first aim of this thesis was to observe, cross-sectionally, the interrelationships between job strain, various measures of general and work-related PC, and a variety of physical and behavioural health outcomes (Study 1; Chapter 2).

Second, a key omission from the PC literature is that the experimental evidence testing methods to influence PC, and the subsequent relationship with health outcomes, has not yet been synthesized. Evidence for the PC-health outcome relationship has tended to be based on correlational evidence and little attention has been paid to moderating factors (i.e.,

intervention length, delivery methods etc.) that may influence this relationship. Therefore, the second aim of this thesis was to explore these possibilities further, by quantifying the extent to which changes in PC lead to changes in health behaviours/physical health outcomes in randomized intervention studies via meta-analysis and, in doing so, provide the first synthesis of experimental studies including PC and measures of health (Study 2; Chapter 3).

Third, methods to reduce PC are also not well known either within the literature or to health care professionals aiming to provide treatment. The most effective (and popular) intervention type highlighted by the meta-analysis was worry postponement (i.e., PC action plans). However, upon considering the methodological characteristics of these interventions, questions remaining regarding the strength of the evidence supporting the various effects reported. For example, the method has not been tested over more than 7 days, is restricted to pre-post designs (i.e., not daily diary to reflect changes in state worry/rumination), and it does not consider health behaviours, which is significant given they form a large part of the PCH. Thus, the third aim of this thesis was to test the relative efficacy of 'augmented' and 'standard' worry postponement interventions (in addition to active and non-active controls) at reducing (state-level) worry, and improving sleep, over a 2-week period in an (online) randomized controlled trial (Study 3; Chapter 4).

Fourth, given how much focus has solely been paid to worry/rumination in the literature, there is also merit in exploring how they interact with variables that capture the fluidity of stress in life. We also require greater consideration of how worry and rumination interact with, or otherwise relate to, other cognitive processes known to influence health behaviour. The Theory of Planned Behaviour (TPB), for example, contains variables that are direct predictors of behaviour; it may be possible that worry and/or rumination thus attenuate the relationship between TPB variables (such as intentions and perceived behavioural control) and behaviours connected to health outcomes or that relationships between PC and behaviour are mediated by TPB constructs as would be predicted by Ajzen (1991). Thus, the fourth aim of this thesis was to provide a fresh consideration of how PC may serve as a

moderator between the intention-behaviour gap (see, Sheeran & Webb, 2016), and to uncover how TPB variables may mediate the relationship between PC and a range of health behaviours (see Study 4, Chapter 5).

Therefore, in sum, the aims of this thesis were:

I: To test the potential role of both general, and work-related PC as a mediating, or potentially moderating, mechanism between job strain and a variety of physical health outcomes and health behaviours (Chapter 2).

II. To synthesise current experimental evidence aiming to reduce PC and test whether, and how, PC can be changed, along with the subsequent impact on physical health outcomes and health behaviours (Chapter 3).

III. To test the relative efficacy of an 'augmented' (i.e., including implementation intentions) and 'standard' worry postponement intervention (alongside active and non-active controls) at reducing (state-level) worry and improving sleep over a 2-week period in an (online) 4-armed randomized controlled trial (Chapter 4)

IV. To test the role of TPB variables (i.e., intentions & perceived behavioural control) in the relationship between PC and a range of health behaviours. Specifically, to test whether PC moderates the relationship(s) between the TPB constructs and behaviour, as well as whether the relationship between PC and health behaviours is mediated by intentions and perceived behavioural control (Chapter 5).

1.10 Thesis Outline

1.10.1 Chapter 2

This chapter presents the manuscript 'The role of the Perseverative Cognition in the job strain-health outcome relationship' published in *Psychology & Health*. This study examines the potential role of both general, and work-related PC as a mediating, or potentially moderating, mechanism between job strain and ill-health outcomes. 650 full-time employees

completed measures of job strain, general and work-related PC (rumination & worry) and health outcomes (burnout, somatization, health behaviours & sleep quality). General and work-related worry and rumination significantly mediated, often independently, the relationship between job strain and burnout, somatization, and sleep quality; however, no significant mediation effects were observed for health behaviours and no type of PC (general or work-related) moderated job strain- health outcome relations. These findings suggest both general and work-related worry and rumination are likely to play important, and partly independent, roles in understanding the adverse relationships between job strain and various health outcomes.

1.10.2 Chapter 3

In this chapter, the manuscript 'Health effects of psychological interventions for worry and rumination: a meta-analysis' published in *Health Psychology* is summarized. This meta-analysis included studies randomly assigning participants to treatment and control groups, measuring PC and a physical and/or behavioural health outcome after exposure to a non-pharmacological intervention. Random-effects meta-analyses revealed the interventions, relative to comparison groups, on average produced medium-sized effects on rumination (g = -.58), small-to-medium sized effects on worry (g = -.41) and health behaviours (g = .31), and small-sized effects on physical health outcomes (g = .23). Effect sizes for PC were positively associated with effect sizes for health behaviours (following outlier removal). Effect sizes for PC were significantly larger when interventions were delivered by healthcare professionals than when delivered via all other methods. No specific intervention type (when directly compared against other types) was associated with larger effect sizes for PC.

1.10.3 Chapter 4

In this chapter, the study, 'Effects of worry postponement on daily worry and sleep: A randomised controlled trial', currently under review, is outlined. A four-armed (online) randomized controlled trial (RCT) was conducted with interval-contingent daily self-report measures of worry each night (duration & frequency for that day) and sleep (onset latency;

disturbance, quality) each morning (for the previous night) as the main outcome variables across 14 days. The study investigated the effects of a worry postponement intervention, alongside a worry postponement + planning intervention (augmented arm) against active and non-active control arm on daily worry and sleep outcomes. Participants in the augmented arm reported significantly lower worry duration (by ~15 minutes, on average, per day). However, the intervention arms did not produce significant improvements in any of the sleep outcomes. Thus, creating specific 'if-then' plans for when and how to engage in a worry postponement can produce favourable outcomes for worry reduction; yet future work is required to understand how to dually tackle both worry and sleep via psychological interventions.

1.10.4 Chapter 5

In this chapter, the manuscript 'Perseverative cognition and health behaviours: Exploring the role of intentions and perceived behavioural control', published in *Psychology and Health*, is outlined. In a prospective design, 650 participants completed baseline measures of TPB constructs and PC (worry and rumination) and 590 completed follow-up (Time 2) measures of health behaviours (physical activity, sleep, sedentary activity, unhealthy snacking) 1-week later. Multiple regression models revealed that worry and rumination (at T1) predicted significantly poorer sleep quality; however, both types of PC were statistically unrelated to all other health behaviours. Worry, but not rumination, and PBC interacted to predict significantly lower physical activity frequency, yet no other moderation effects were observed. Significant mediation effects were present but only for total sleep time and sleep quality. Consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant. Together, these set of findings provide fresh longitudinal support for the harmful relationships between PC and sleep, while also revealing new relationships between the components of PC, PBC and health behaviours.

1.10.5 Chapter 6

This chapter comprises a discussion of the findings from the meta-analysis and empirical studies from this thesis within the context of the PCH and EPCH, along with the associated literature. The strengths and limitations of the thesis are considered and areas for future research are identified.

Chapter 2

The role of the Perseverative Cognition in the job strain-health outcome relationship

2.1 Introduction

The United Kingdom Health and Safety Executive estimates 12.8 million working days are lost each year as a consequence of work-related stress, costing the taxpayer an annual bill of £5.2 billion (HSE, 2021). Research on work-related stress was relatively sparse until the late 1970's (Beehr, 1998), but a plethora of studies now exist showing that work-related stress is not only a risk factor for absenteeism (e.g., Kinnunen & Nätti, 1994; Palmer, 2018), but also for a range of negative mental and physical health outcomes (e.g., Levenstein, Smith, & Kaplan, 2001;O'Connor, Thayer, Vedhara, 2021). For example, individuals experiencing heightened stress at work report lower self-rated health (De Witte, Pienaar, & De Cuyper, 2016; Ferrie, Shipley, Newman, Stansfeld, & Marmot, 2005), high levels of mental distress (e.g., O'Connor et al., 2000; 2021) increased instances of coronary heart disease and hypertension (Levenstein, Smith, & Kaplan, 2001; Lee, Colditz, Berkman, & Kawachi, 2004) and also are more likely to suffer from obesity (Ferrie et al., 2005; Muenster, Rueger, Ochsmann, Letzel, & Toschke, 2011). For these reasons, many now consider work-related stress a modern-day pandemic and call for urgent preventative action (Mental Health Foundation, 2018).

One of the most significant and long-standing models of occupational stress is the job demands-control model (Karasek, 1979). Two fundamental mechanisms underpin the model: psychological strain and active learning mechanisms. The former is characterised by the experience of high job demands with simultaneous low levels of control over decision-making, leading to greater psychological strain. The latter is categorized based on the experience of high job demands and low levels of control and is said to promote the

development of new adaptive behaviours. Importantly, the model is underpinned by several existing theories within the stress literature (cf., Cohen, Gianaros & Manuck, 2016; Brosschot et al. 2016, 2017, 2018) informed by the concept of 'allostatic load' (McEwen, 1998); attesting that the wear and tear that the body experiences is due to repeated and long-term exposure to stress. It would therefore follow that actions to meet work demands yield short-term psychological (e.g., mental fatigue) and physiological (e.g., increased heart rate, adrenaline secretion) reactions which, initially, are adaptive and reversible. However, when one fails to recover, the adaptive nature of the response turns into negative load effects, such as exhaustion, chronic tension and persistent sleep difficulties (Geurts & Sonnentag, 2006; Kuiper, Van der Beek & Meijman, 1998; O'Connor, O'Connor, & Marshall, 2007).

Empirical data dating back to the mid-nineties supports the adverse relationship between job strain and ill-health. The Whitehall II study (North et al., 1996) was a relatively early example of a study that demonstrated that low job control, whether through self-report or independent assessment, predicted significantly more incidences of coronary heart disease and higher rates of short term and long-term sickness absence. In fact, adjusting for low decision control reduced the odds-ratio to develop any form of heart disease in the lowest grade (compared with the highest grade) from 1.5 to 1.2. Furthermore, a range of endocrinological and physiological evidence also now exists supporting this link. Landesbergis et al. (2013), in a meta-analysis of 22 cross-sectional studies, showed that a single exposure to job strain was associated with higher ambulatory systolic blood pressure and diastolic blood pressure. Jarczok et al. (2013) systematically reviewed the association between job strain and heart-rate variability (HRV). Thirty-six studies representing over 27,000 employees from 10 countries showed that job strain and adverse work conditions again were associated with decreased HRV. This is especially significant given a recent large-scale study reported that low levels of vagally mediated HRV are associated with elevated risk of a range of

cardiometabolic and inflammatory diseases (Jarczok, Jarczok & Thayer, 2020; Mauss et al., 2014).

However, recent advances in stress theory have highlighted a new mechanism through which stress at work may affect a range of psychobiological processes, even long after the stressor is present in the environment. The perseverative cognition hypothesis (PC Hypothesis; Brosschot et al., 2005, 2006, 2013) suggests that worry and/or rumination (via repetitive thinking) may lead to disease by prolonging the stress-related physiological activity associated with facing a stressor, by both amplifying the short-term bodily response to stress, and by delaying the recovery and/or reactivating the stress response following a stressor. Rumination and worry are similar constructs but differ in terms of content. Worry is associated to future-oriented threat prediction, often in an (unsuccessful) attempt to reduce negative outcomes or solve a problem (Borkovec, 1994). Rumination is characteristically related to perseveration about one's own symptoms, the consequences of those symptoms, and past experiences habitually, in an (unsuccessful) exertion to understand oneself (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Critically, these processes represent stressors that, when prolonged, activate harmful physiological and psychological outcomes (for review, see Ottaviani, 2018) and trigger unhealthy behaviours (for review, see Clancy et al., 2019). Given that job stress is not strictly bound to the work environment and is likely, if not inevitably (see, Lourel et al., 2009), to spill over into non-work domains, the maladaptive response to stress experienced through worry or rumination may be particularly sensitive to work-related stressors.

Existing empirical studies accounting for the role of perseverative cognition (PC) at work primarily focus on rumination and one's recovery or respite from work (e.g., Cropley & Zijlstra, 2011), along with its consequences for psychological and emotional wellbeing (e.g., Hamesch, Cropley & Lang, 2014), burnout (Marinelli & Piazza, 2002), and work reappraisal (Ray, Wilhelm & Gross, 2008). Of the studies drawing associations between perseverative cognition and health consequences, many are dated (e.g., Roger & Hudson, 1995; Roger &

Najarian, 1998; Cropley & Millward-Purvis, 2003) and few of them attempt to capture a broad range of health outcomes within the same sample of participants (e.g., sleep, Van Laethem et al., 2016; burnout, May et al., 2020; eating habits, Eschle & McCarrick, 2021). Existing studies have also tended to focus solely on physical health outcomes and despite the findings being crucial to understanding the role of perseverative cognition at work – such as those showing men who cannot relax after work have a threefold increased risk of heart disease (Cropley et al., 2014) – the role of health behaviours should not be overlooked. Some evidence does exist for the relationships between work-related rumination and biomarkers of sleep; for example, whereby high ruminators have been associated to a heightened cortisol awakening response (see Cropley et al., 2017). However, less work focuses on overall sleep quality, which is concerning given non-work time largely consists of sleeping and recuperation (Åkerstedt, & Nilsson, 2003).

The respective role(s) of *work-related* worry and rumination, and how these relate to their more general counterparts, within the context of job strain and health outcomes is unclear. Because of their unique potential to prolong the impact of work-related stressors (e.g., Cropley et al., 2006), work-related worry and rumination may represent specific, and particularly damaging, manifestations of employees' inability to 'switch-off' following work. There is some evidence to suggest work-related rumination is associated to physiological markers of ill-health (such as lower parasympathetic activity, see Cropley et al., 2017); however, a scarcity of studies exist which assess the role of worry at (or about) work (exceptions are Aasa et al., 2005 & Flaxman et al., 2012; Van Laethem et al., 2015). This is significant as, in line with the PC Hypothesis, if people are likely to start thinking and anticipating work before they arrive then its more than probable that these feelings will decompartmentalize in some form of worry. Therefore, work-related (as well as general) worry and rumination may augment the adverse health impacts of stress at work, potentially serving as a moderating mechanism; such that more job strain and worry interact to produce poorer health outcomes. It may also be the case that work-related worry and rumination, in

congruence with the PC Hypothesis, mediate this relationship; such that the path from increased job strain to poor health may be fully explained by higher scores in worry and rumination.

2.1.1 Aims

Consequently, the present study aimed to consider the relative impact of both general *and* work-related worry and rumination as a mediating/moderating mechanism between the job strain and health outcome relationship. In doing so, we aimed to provide a renewed consideration of the psychosocial work environment as a predictive factor in public health outcomes. Furthermore, due to concerns that the defence/vigilance response associated to PC is in fact derived from neuroticism (cf., Watson & Pennebaker, 1989) and in light of recent findings suggesting negative affectivity may serve as an additional emotional risk factor to health during the ongoing coronavirus pandemic (see, Kroencke et al., 2020), sensitivity analyses were conducted to check whether the key findings hold after controlling for neuroticism. Importantly, this approach enables us to determine if the relationship between different types of PC and health outcomes, as well as the associations between job strain (through PC) on health, still stand when controlling for neuroticism. To test these aims, a sample of adults in full-time employment were recruited and completed a series of measures of job demands and control (from which a measure of job strain was derived), general and work-related PC, and a range of health-related outcomes (burnout, somatization, health behaviours and sleep). It was predicted that:

Higher levels of general PC (worry & rumination), as well as work-related PC, will be significantly associated with poorer health outcomes (*Hypothesis 1*);
General PC (*Hypothesis 2A*), as well as work-related PC *(Hypothesis 2B)*, will moderate the negative relationship between job strain and health outcomes, such that this relationship will be stronger in individuals with higher levels of general and work-related PC compared to lower levels;

General PC (*Hypothesis 3A*), as well as work-related PC (*Hypothesis 3B*), will mediate the job strain and health outcome relationship. In relation to this final hypothesis, multiple mediation models will be conducted to test whether the different types of PC (general worry, general rumination, work-related worry, work-related rumination) additively contribute to the pathway between job demands and health outcomes such that they play a significantly unique and independent role, whether they play a similar role (and thus render one another non-significant), or whether some forms of PC are significant while others are not.

2.2 Method

2.2.1 Design & Participants

The present study employed an online cross-sectional design to capture self-reported feelings towards stress at work and was preregistered on AsPredicted (see, here). Recruitment was purposefully sampled across adults who reported experiencing 'stress at work' between 1st January 2020 and 30th January 2021. Social media adverts (e.g., Facebook, Twitter) and Prolific were used as the primary recruitment methods; advertising was also shared externally by the Mental Health Foundation and MIND. Participants recruited through Prolific were paid in line with Prolific's participants reimbursement policy (equivalent to £5 per hour). To be eligible, participants had to be employed on a full-time basis and be aged 18 years or older. The study received institutional ethical approval from a university-based ethics committee (REF: PSY-763).

An a-priori power calculation (in G*Power version 3.1; Faul et al., 2009) indicated a minimum of 616 participants would be required to detect an effect size of g = .28 (equivalent to r = .14) based on a power (1- β) of 0.80 in a one-tailed test with alpha set at .05. This was based on a recent meta-analysis which identified the average association between PC and health outcomes (McCarrick, Prestwich, Prudenzi & O'Connor, 2021).

Eight hundred and three participants initially provided responses to the online questionnaire. Of these, 73 did not provide any information beyond consent, 45 only reported their

demographical data, 35 progressed beyond the demographics section but did not complete all of the study variables specified in the hypotheses and 650 participants completed all measures. Consistent with our preregistration, the final sample comprised only of the 650 full-time employed adults (M^{age} = 28.9 years, *SD* = 10.9 years) completing all relevant measures. The data can be accessed via the Open Science Framework (OSF, here).

Nationality and ethnicity were classified in accordance with the categories outlined by the Office for National Statistics (ONS, 2021) and, due to the diverse range of occupations reported by participants, the International Certification of Jobs (ISCO-08) was used as a framework to organise job titles into a clearly defined set of groups based on the authority, responsibilities, tasks and duties associated to the respective job roles.

2.2.2 Measures

2.2.2.1 Predictors

Job Strain

The 35-item Health & Safety Executive Management Standards Indicator Tool (HSE; Cousins et al., 2004) is an extensively validated measure (e.g., Marcatto et al., 2014) used by organizations to monitor working conditions that can lead to increased stress. The scale comprises 6 sub-scales relating to stress in the workplace. Consistent with previously validated methodology (see, Landsbergis et al., 1994; O'Connor et al., 2000) we computed a measure of job strain by dividing *job demands* (α = .87, e.g., "It is clear what is expected of me at work") by *job control* (α = .82, e.g., "I know how to go about getting my job done").

2.2.2.2 Mediators/Moderators: Perseverative Cognition

General Worry

The 16-item Penn-State Worry Questionnaire (PSWQ; Meyer et al., 1990) is often noted as the 'gold standard' measure of state worry. It has routinely demonstrated high internal consistency in non-clinical criterion groups (α =.95, Molina & Borkovec, 1994), has high test-

retest reliability amongst adult populations (*r* = 0.74-0.92) as well as substantial inter-rater reliability (*r* = .55; Stober, 1998). Participants are instructed to indicate how typical statements are of them on a five-point scale varying from 1 (“not at all typical of me”) to 5 (“very typical of me”). Example items include “My worries often overwhelm me” and “I am always worrying about something”. A total score is calculated by summing the items and scores range from 16–80, with higher scores representing a greater degree of pathological worry.

Work-related Worry

Given there is not currently a widely accepted and implemented measure for worry in the workplace, the 4-item Stress Arousal Scale (SAS; Smith & Everly, 2012) was used and adapted for brevity. Participants responded to questions relating to the cognitive-affective precursors of physiological stress on a 1 (“never”) to 5 (“always”) point-scale, with higher scores indicative of greater worry. The items were adapted to include a work focus such as “I am concerned or worried about things, at work” and “I anticipate upsetting things, about work”. The measure has been used before to assess work-related worry (e.g., Borghini et al., 2020), has demonstrated high internal consistency within a sample of full-time employed adults (α =.88; Smith et al., 2014), and shows good convergent validity with the PSWQ (*r* = .60).

General Rumination

A shorter 10-item (Treynor, 2003) version of the Rumination Response Scale (RRS; Nolen-Hoeksema, 1991) was used to measure the frequency of depressive rumination through brooding and reflection. The scale correlates strongly with the full 21-item scale (*r* = .72 - .82) and yields an ‘overall’ score, as well as two subscales, Brooding and Reflection. Example items include: *Brooding* “Why do I always think this way?” and “What I am I doing to deserve this?”*; Reflection*: “I write things down and analyse them” and “I go someplace alone to think about my feelings”; with higher scores reflecting greater instances of

rumination. Given Brooding is seen as more damaging than reflection in terms of health (cf., Schoofs, Hermans & Raes, 2010), we repeated the analyses substituting the overall general rumination scores with the brooding scores. The results for the analyses in which the analyses are run with Brooding-specific items (rather than the 'overall' rumination measure) are reported in Appendix 1.1.4. In brief, this change did not significantly influence any of the findings relating to the present study's hypotheses

Work-related Rumination

The 15-item Work-Related Rumination Questionnaire (WRRQ) assesses the content domains of affective rumination, problem-solving pondering, and detachment from the three-factor model of perseverative thinking about work (Cropley, Michalianou, Pravettoni, & Millward, 2012). Given the detachment subscale has been shown to be strongly and negatively correlated with both the affective rumination and problem-solving pondering subscales, only the affective rumination and problem-solving pondering sub-scales were used in this study. Example items include "Are you troubled by work-related issues when not at work?", and "After work I tend to think about how I can improve my performance". Items are responded to along a 5-point Likert scale ranging from 1 ("never") to 5 ("always") and each factor yields a total score which ranges from 0 to 25 (which was summed to make a composite score).The scale has been used and validated in previous studies in working adults, with good to excellent internal consistency (α = 81 - .90; Querstret & Cropley, 2012) and Cronbach's alphas for the present study were also very good: α = 86, (affective rumination), α = 89 (problem-solving pondering).

2.2.2.3 Outcomes

Burnout

The 18-item Copenhagen Burnout Inventory (CBI; Kristensen et al., 2005) consists of 3 subscales measuring personal, work-placed, and client-related burnout. Given the scope of the present study, the *'work-placed'* subscale was used to capture participants' current

susceptibility to burnout in their workplace; which has been shown to predict future sickness absence, sleep problems and the use of pain-killers (Skakon et al., 2010). The measure employs a 1 (never) – 5 (always) scale, with higher scores indicating greater signs of burnout. Example items include “I feel worn out at the end of the working day” and “My work is emotionally exhausting”. The subscale demonstrates high levels of internal consistency (α = .87) and has promising convergent validity in both the mental (*r* = .67) and physical (*r* = .49) health subscales of the health-related quality of life inventory (SF-36 Health Survey; Ware, 1999).

Somatization

The Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982) is a multidimensional test to measure current levels of psychological and physiological symptoms. The present study used the ‘*Somatization’* subscale of the measure to capture participants’ experience of psychological distress in the form of somatic symptoms. Participants are asked to rate the degree to which they experience individual somatic symptoms on a scale of 1 (“not at all”) to 4 (“very much so”). Example items include ‘I get pains in heart or chest’, ‘I have trouble getting my breath’ and ‘I feel weak in parts of my body’. The somatization subscale has demonstrated acceptable test–retest reliability (*r* = .71) and good internal consistency (α = .85).

Sleep Quality

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is often regarded as the ‘gold-standard’ self-report measure of sleep quality (for review, see Mollayeva et al., 2016). The PSQI consists of 19 items that produce a global sleep quality score and the following 7 component scores: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction. PSQI items use varying response categories that include recording usual bed time, usual wake time, number of actual hours slept, and number of minutes to fall asleep, as well as forced-choice Likert-

type responses, resulting in an overall sleep quality score, with higher scores depicting poorer sleep. The scale has demonstrated good test–retest reliability (*r* = .79 - .83) and internal consistency (α = .83).

Health Behaviours

The 16-item Good Health Practices scale (Hampson, Edmonds & Goldberg, 2017) provides a broad coverage of health promoting behaviours. To be consistent with the other study measures, and for ease of communication, all items were reverse scored such that higher scores reflected poorer health behaviours. A total score is provided by summing all items together. Participants responded to a 5-point Likert scale (1 = not at all like me - 5 = very much like me), with higher scores indicating unhealthier behaviours. Example items include "I exercise to stay healthy" and "I eat a balanced diet". In the original study, the scale was internally consistent (α = .92) and predicted physiological dysregulation (e.g., abnormal blood glucose levels; higher body-mass-index).

2.2.2.4 Covariates

In addition to age and gender, neuroticism was also assessed as a covariate. The 10-item Neuroticism subscale of the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 2006) was used to gauge participants' self-perceptions of negativity and emotionally instability. The NEO PI-R is widely accepted as the 'gold-standard' questionnaire measure of the Five Factor Personality Model. Participants respond to a 5-point Likert scale ranging from 1 ("never") to 5 ("always"), with higher scores being indicative of higher Neuroticism. Example items include "I dislike myself" and "I get easily irritated*".* The *Neuroticism sub-scale* has excellent internal consistency in adults (α = 87-92; Asendorpf et al., 2011) as well as promising convergent validity with other measures of emotional instability (*r* = .68, Thompson et al., 2012).

Body Mass Index (BMI) and the number of hours each participant worked per week (HWPW were also self-reported, but as the strength of the relationships between these variables and

the health outcome measures were typically very weak, non-significant, and directionally inconsistent (see Table 2.1, BMI: r = .04 – .12; HWPW: r = -.08 – .04), they were not considered within the regression analyses.

2.2.3 Procedure

After providing informed consent to participate in a study titled 'Work, Health & Wellbeing Study', participants completed the measures on a survey site (Qualtrics) in the following order: job strain, neuroticism, general worry and rumination, work-related worry and rumination, somatization, burnout, health behaviours, and sleep. The median time taken to complete the survey was 14 minutes and 21 seconds (SD = 5.56 minutes). Following completion of the survey, participants were debriefed.

2.2.4 Method of Analysis

Data were analysed in R-Studio (version 3.6.2).

Data was first tested for randomness using Little's Missing Completely At Random (MCAR) test as a small amount of data was missing for Body Mass Index (BMI, N = 39). Little's test was non-significant (p = .745) and further graphical summaries confirmed there were no missing cases elsewhere in the data (see Appendix 1.2.2). As an extra safe-guard, the analyses were run with and without imputed data for this variable (i.e., BMI, using single, expectation maximization imputation). The use of imputed data did not alter the results; therefore, the non-imputed findings are reported.

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 (e.g., scatter plots, Cullen & Frey graphs, QQ-plots, PP-plots etc.), formal tests (e.g., Durbin-Watson, Goldfield-Quandt, Variance Inflation Factor etc.) 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 analysis.

The 'lm' function (Base R; Chambers, 1992) was used to calculate beta coefficients to determine if greater PC was associated with poorer health outcomes and to conduct moderated regressions to assess interactions between job strain and PC on health outcomes. Multiple R^2 was calculated to indicate the size of effects for the relationship(s) between study variables (Hypothesis 1 & 2). According to effect-size conventions for R^2, 0.02, 0.15, and 0.35 represent small, medium, and large effects (Rosnow & Rosenthal, 1989). Across all analyses, bootstrapping, with 5,000 random imputations, was used to assess robustness; bootstrapped confidence intervals and re-sampled *p*-values were generated.

Correlational analyses were conducted to assess the interrelationships between the predictor (job strain), mediator (general and work-related worry and rumination) and outcomes (burnout, somatization, health behaviours and sleep quality).

A series of hierarchical ordinary-least squares (OLS) regressions were then conducted to test if higher levels of general PC, as well as work-related PC (i.e., worry and rumination), significantly predicted poorer health outcomes (i.e., burnout, somatization, health behaviours & sleep) (Hypothesis 1). Separate regressions were performed for each construct of PC to aid in comparisons with previous research and to maximise statistical power (see Hayes, 2019) (see, Appendix 1.1.1, Tables 1 – 16).

Further OLS regressions were used to test if general PC (worry or rumination) (Hypothesis 2A), as well as work-related PC (worry or rumination) (Hypothesis 2B), significantly moderated the negative relationship between job strain and health outcomes, such that this relationship is intensified within individuals reporting higher levels of general and work-related PC. For these analyses, job strain was entered at step 1, general worry or rumination

(or work-related worry or rumination) at step 2, and the interaction between job strain and general worry or rumination (or work-related worry or rumination) was entered at step 3.

Mediation, using the product of ordinary-least-squares estimation approach, was computed to determine if general PC (Hypothesis 3A), as well as work-related PC (Hypothesis 3B), significantly mediated the job strain and health outcome relationship. The R package 'psych' (Revelle, 2015) was used to estimate the direct, indirect, and the total effects for the path from the proposed predictor(s) (i.e., job strain) to the mediator(s) (i.e., general and work-related PC), and for the path from the mediator to the outcome variable (i.e., health outcomes). In view of highlighting the precise mechanism through which job strain is exacerbated through PC, and because it is unlikely the effect of an independent variable on an outcome variable is only transmittable by one means alone (Preacher & Hayes, 2008), *both* general worry and rumination *and* work-related worry and rumination were entered into the same multiple mediation model.

Finally, an additional set of regressions were conducted to test whether each type of PC (general worry & rumination (as a set); work-related worry and rumination (as a set); both general and work-related worry & rumination (combined together)) independently predicted poorer health outcomes. This approach was employed to determine if variation across the different types of PC independently predicted poorer health across the outcome variables. This may have implications from an applied perspective, as targeting worry and rumination together (rather than one alone) may produce more favourable changes in the outcomes.

Given females were significantly more likely to suffer from burnout, $t(572) = 2.60$, $p = .009$, exercise poorer health behaviours, $t(598) = 4.48$, $p < .001$, and experience poorer sleep, $t(572) = 3.44$, $p < .001$; and because being younger was associated with significantly higher levels of burnout, $\beta = -.04$, $p = .035$, and somatization, $\beta = -.09$, $p < .001$, age and gender were considered as covariates. In addition, to assess if the relationships between PC and the outcomes were independent from established personality correlates of stress (i.e.,

neuroticism, see Enns, Cox & Clara, 2005), the analyses were also ran with (and without) neuroticism included (at step 3). These results are reported within Appendix 1.1.1.

2.3 Results

2.3.1 Participant Characteristics

An overview of participants' demographics can be found in Table 2.1. Typically, participants were white males aged around 29 years old, worked approximately 31.5 hours a week, and were educated to university level (see Table 1). Scores for both types of PC, as well as job strain, were particularly high in the present sample and the degree to which the scores were dispersed around the mean was relatively small. Further Figures, for the different participant demographics and how they were related to the different measures of PC and health, are presented in Appendix 1.2.1 (see Figures 1 – 13).

Table 2.1. Participant Demographics

Characteristics	
Age (years), M (SD)	28.9 (10.9)
Gender, % female (N)	43.23 (281)
Hours worked per week, M (SD)	31.5 (15.5)
Body Mass Index, M (SD)	25.3 (7.88)
Ethnicity, N (%)	
White	542 (83.38)
Mixed	27 (4.15)
Asian	16 (2.46)
Black	11 (1.69)
Other	54 (8.31)
Nationality, N (%)	
UK & Ireland	176 (27.08)
North or West Europe	22 (3.38)
USA or Canada	24 (3.69)
Central or Eastern Europe	186 (28.62)
South Europe	176 (27.08)
Latin America, South Pacific, Middle East, African	66 (10.15)
Occupation, N (%)	
Managers (e.g., chief executives, legislators)	33 (5.08)
Trained professionals (e.g., scientists, lawyer)	143 (22.00)
Technicians and associate professionals (e.g., health assistants, business service agents)	105 (16.15)
Clerical support workers (e.g., secretarial, clerks)	13 (2.00)
Service and sales workers (e.g., waiters, hairdressers, traders)	103 (16.85)
Skilled agriculture (e.g., crop growers, animal producers).	7 (1.08)
Craft workers (e.g., building trade, garment trade)	17 (2.62)
Plant and machine operators (e.g., mining, truck drivers).	18 (2.77)
Elementary occupations (e.g., cleaners, labourer)	203 (31.23)
Armed forces (e.g., army, navy)	8 (1.23)
Education, N (%)	
Some secondary school	118 (18.15)
GCSE (or equivalent)	53 (8.15)
A-level (or equivalent)	93 (14.31)

Foundation degree (or equivalent)	38 (5.85)
Degree	201 (30.92)
Masters	118 (18.15)
PhD	29 (4.46)
Mental health condition, N (%)	
Yes	102 (15.69)
Physical health condition, N (%)	
Yes	101 (15.54)

Table 2.2 Interrelationships between study variables (SD)

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age	28.90	10.90	-													
2. Gender	1.43	0.50	.24***	-												
3. BMI	25.30	7.88	.08	-.02	-											
4.Hours worked per week	31.50	15.5	.27***	.01	.03	-										
5.Job Strain	0.96	0.07	-.02	.06†	.09*	.05	-									
6.General worry	46.70	9.41	-.19***	.23***	.01	-.07**	.21***	-								
7.Work-related worry	11.90	4.00	-.01**	.06	.04	-.08*	.35***	.55***	-							
8.General Rumination	24.80	7.06	-.22***	.07	.06	-.10**	.25***	.62***	.52***	-						
9.Work-related Rumination	42.70	7.16	-.09**	.04	.04†	-.05	.19***	.34***	.62***	.42***	-					
10.Neuroticism	31.60	8.00	-.12***	.18***	.03	-.09*	.24***	.44***	.44***	.50***	.18***	-				
11.Burnout	21.60	4.85	-.08*	.10**	.12**	.04	.43***	.48***	.64***	.47***	.57***	.43***	-			
12.Health behaviours	49.80	12.10	-.03	-.18***	.04	.01	.03	.05	.02	-.01	-.02	.21***	.11**	-		
13.Somatization	13.70	6.39	-.17***	.07	.13**	-.08*	.29***	.42***	.46***	.49***	.38***	.40***	.46***	.02	-	
14.Sleep quality	10.00	4.34	-.01	.14***	.05	-.07*	.24***	.33***	31***	.30***	.17***	.44***	.38***	.19***	.38***	-

Note: ***p < .001 **p < .01 * p < .05; for gender 1 = male, 2 = female.

2.3.2 Hypothesis 1: Does greater PC predict poorer health outcomes?

The correlational analyses suggested that all measures of PC were related with burnout (particularly the work-related measures of PC), somatization and sleep quality, with higher levels of PC associated with higher burnout, higher somatization and poorer sleep quality (see Table 2.2). The PC measures were unrelated to the overall measure of health behaviours. The analyses also revealed that the measures of PC were modestly correlated with each other, with the exception of work-related worry and work-related rumination (i.e., all *r*'s between .34 and .62), suggesting that they are distinct constructs.

2.3.2.1 General Perseverative Cognition

In separate regressions for worry and rumination, general PC significantly predicted burnout (worry: β = .47, p <.001, R^2 = .22; rumination: β = .46, p <.001, R^2 = .21), somatization (worry: β = .41, p <.001, R^2 = .17; rumination: β = .49, p <.001, R^2 = .24), and sleep quality (β = .34, p <.001, R^2 = .12; β = .31, p <.001, R^2 = .09). These associations remained after controlling for age and gender together, as well as neuroticism alone (see Appendix 1.1). Although higher levels of general worry were initially statistically unrelated to health behaviours (β = .04, p = .258, R^2 = .01), it did become a significant predictor after controlling for age and gender (as a set), β = .09, p = .025, R^2 = .01.

Higher levels of rumination were initially statistically unrelated to health behaviours, β = -.01, p = .849, R^2 = .01), but when gender and neuroticism were controlled for (as a set) higher levels of general rumination was significantly associated to poorer health behaviours (β = -.15, p <.001, R^2 = .02). Each of these associations, for both general worry and general rumination, also stood when controlling for neuroticism (see Appendix 1.1).

2.3.2.2 Work-related Perseverative Cognition

Work-related PC significantly predicted burnout (worry: β = .64, p <.001, R^2 = .41; rumination: β = .58, p <.001, R^2 = .33), somatization (worry: β = .47, p <.001, R^2 = .22; rumination: β = .39,

p<.001, R^2 = .15) and sleep quality (worry: β = .31, *p* <.001, R^2 = .10; rumination: β = 18, *p* <.001, R^2 = .03). Higher levels of work-related PC were statistically unrelated to health behaviours (worry: β = .02, *p* = .534, R^2 = .01; rumination: β = -.04, *p* = .369, R^2 = .01).

Although higher levels of work-related worry were initially statistically unrelated to health behaviours, β = .02, *p* = .534, R^2 = .01, it did become a significant predictor after controlling for age and gender (as a set), β =.-.08, *p* <.001, R^2 = .09. Further information on each of these analyses can be found in Appendix 1.1.

2.3.2.3 Additional Analyses

An extra set of sensitivity analyses testing if each type of PC (when entered together, at the same level, as predictor variables) predicted poorer health outcomes, revealed that, with the exception of the relationship between work-related rumination and sleep quality, β = -.01, *p* = .608, R^2 = .01, each type of PC remained a significant predictor of greater burnout and somatization, as well as poorer sleep quality. As in the main analyses, no relationships were observed for health behaviours. (see, Appendix 1.1.3, Tables 21 - 23). This suggests worry and rumination (both general *and* work-related) are uniquely important predictors of ill-health.

2.3.3 *Hypothesis 2: Does Perseverative Cognition moderate the relationship between job strain and health outcomes?*

While job strain significantly predicted greater burnout across each analyses containing the different types of PC*, B* = 12.87 to 37.47, *SE* = 5.73 – 9.12, all *p* <.01, R^2 = .32 - .47, none of the PC measures (general or work-related) moderated the relationships between job strain and any of the outcome variables. The regression models for these results can be found in Appendix 1.1, Tables 17-20.

2.3.4 *Hypothesis 3: Does Perseverative Cognition mediate the relationship between job strain and health outcomes?*

The mediation models revealed significant indirect paths between job strain and burnout, somatization, and sleep quality (but not health behaviours), via total PC (general worry; general rumination; work-related worry; work-related rumination) (see Table 2.3). These paths remained significant when controlling for age, gender and neuroticism.

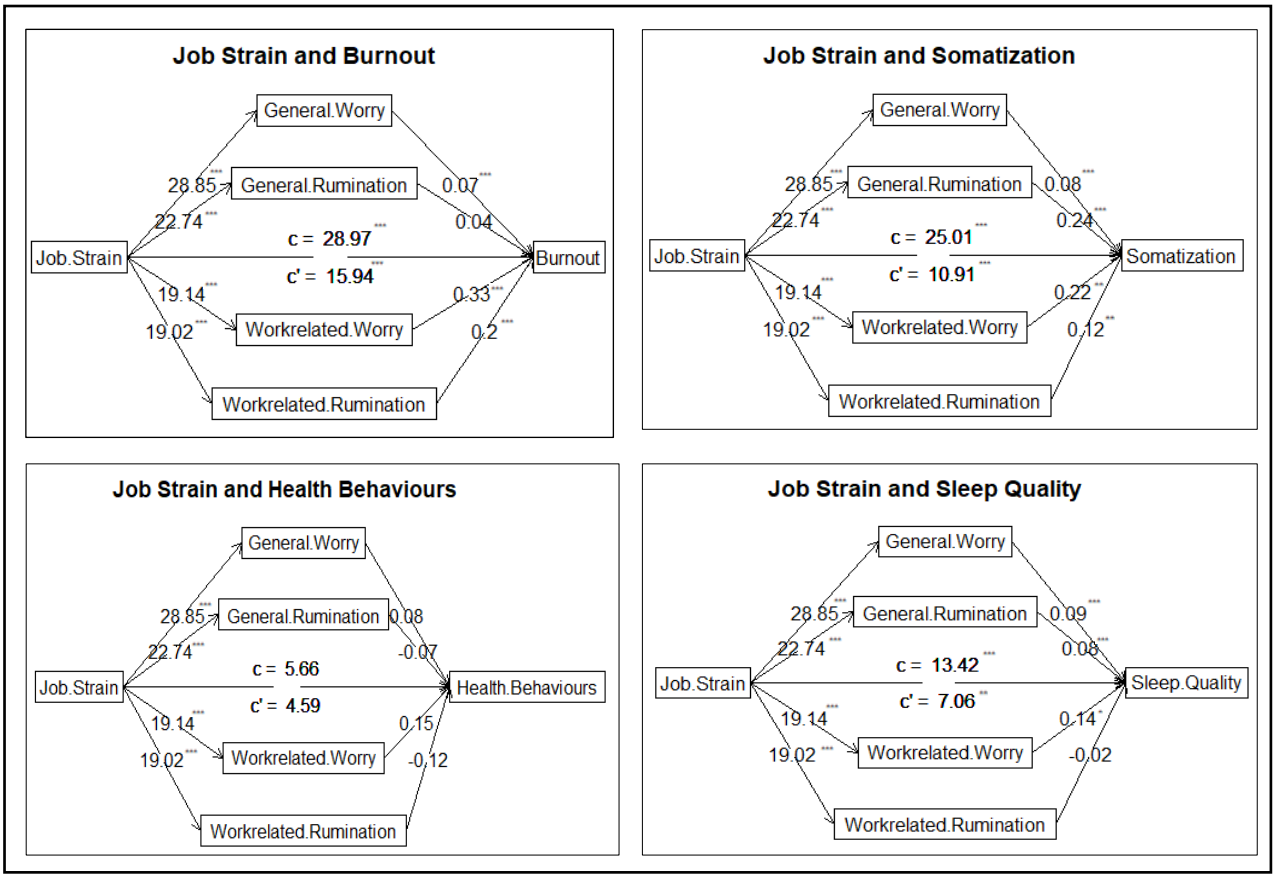
Table 2.3. Mediation Analysis for Job Strain, PC, and Health Outcomes.

Predictor	Outcome	Effect	*b (95% CI)*	*S. E*	R^2
Job Strain	Burnout	Total	28.97***	2.32	
		Direct	15.94***	1.88	
		Indirect	12.99*** (9.62 – 16.62)	1.70	.54***
Job Strain	Somatization	Total	25.01***	3.26	
		Direct	10.91***	3.00	
		Indirect	14.16*** (10.15 – 18.35)	2.04	.33***
Job Strain	Health Behaviours	Total	5.66	6.43	
		Direct	4.59	6.89	
		Indirect	1.03 (-.4.22 – 6.67)	2.79	.01
Job Strain	Sleep Quality	Total	13.42***	2.25	
		Direct	7.06**	2.28	
		Indirect	6.36*** (4.31 – 8.58)	1.09	.16***

*Note: General worry/rumination as well as work-related worry/rumination are the mediator terms in these mediation models. Beta-coefficients are unstandardized; p-values and 95% CI's for indirect effects are from 5,000 bootstrapped samples; ***p < .001 **p < .01 * p < .05.*

The individual paths from job strain to each outcome, via each type of PC, are shown in Figure 2.1. With the exception of the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were significant.

Figure 2.1. Mediation path analysis for Job Strain, PC, and Health Outcomes.



Note: Figure 2.1 shows the individual paths from job strain to each outcome, via each type of PC. With the exception of the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were significant. Numbers reflect the unstandardized regression coefficients; C = the total effect of x on y; C' = the direct effect of x on y; ***p < .001 **p < .01 * p < .05.

2.4 Discussion

The aim of the present study was to test the relative impact of both general *and* work-related worry and rumination as a mediating and/or moderating mechanism between job strain and health outcomes. The data were broadly in support of our hypotheses regarding the predictive role of PC on health and its function as a mediator between stress and ill-health, although PC and job strain did not interact to predict health outcomes. Both types of general *and* work-related worry and rumination predicted significantly higher scores in burnout and somatization, as well as lower scores in sleep quality (Hypothesis 1). Until age and gender were controlled for, no significant relationships were observed for the impact of any type of PC on health behaviours. While job strain significantly predicted greater burnout across each analysis containing the different types of PC, none of the PC measures (general or work-related) interacted with job strain to predict any of the health outcome variables (Hypothesis 2). However, for the mediation analyses, with the exception of the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were statistically significant (Hypothesis 3). Thus, together, the results of the present study provide fresh evidence for the damaging nature of PC, its role as an important mediating mechanism between exposure to stress and adverse health outcomes as well as the potential unique, additive contributions of different types of PC.

Similar to previous studies associating PC to health consequences (for reviews, see Ottaviani et al., 2018; Clancy et al., 2019), and in conjunction with Hypothesis 1, higher scores in PC predicted more adverse health outcomes. In the present study, this finding is extended to a large, multi-cultural, sample of full-time employees. Specifically, all types of PC (both general and work-related) predicted significantly higher scores in burnout and somatization, as well as lower scores in sleep quality. This is significant as, despite emerging reports associating rumination specifically about work to physiological consequences (see Cropley et al., 2017) and some contemporary evidence for the effect of

worry in the workplace (see Aasa et al., 2005 & Flaxman et al., 2012), we show, for the first time, that work-related worry *and* rumination – as well as its more general counterparts – represent a uniquely harmful threat to a range of health markers. It is also consistent with the original perseverative cognition hypothesis (see Brosschot et al., 2005), wherein the repetitive and pervasive thinking styles represented by worry and/or rumination (or in this case about work) may amplify the short-term bodily response to stress and delay the recovery and/or reactivation of the stress response following exposure to a stressor. Thus, it would follow that, employees' psychological manifestations of past and future stressors experienced either through worry or rumination about work (e.g., upcoming deadlines; fractious relationships with colleagues), or just more generally (e.g., personal feelings of inadequacy; analyzing past behaviours), likely contribute to their inability to 'switch-off' following work (through rumination) and to mentally loiter over stressors (via worry). It is also important to note that all of the observed effects held after controlling for neuroticism indicating that PC is a distinct and independent predictor of these outcomes.

While job strain, that is high job demand and low job control, did predict significant increases in burnout, our consideration of the interplay between PC and health within the context of job strain, was not in line with our hypotheses (see Hypothesis 2A & 2B). Indeed, no moderation effects were observed between any type of PC (general or work-related) and job strain on any health outcome. Some consistency can be sought, however, between this null result and of those reported by previous studies. Cropley et al. (2006), for example, found that work-rumination did not significantly moderate the relationship between job strain and sleep quality; though, this was observed in a much smaller (N = 152) and homogeneous sample (i.e., school teachers). Equally, also for sleep, Van Laethem et al. (2015) reported similar size associations between PC and sleep quality in a longitudinal study (r = .28 - .32). It is also notable that the absolute strength of the reciprocal relationships between PC and sleep in that study were much smaller than those in the present study (e.g., direct effect, B = .03 vs B = 7.06); although, crucially, moderation effects were not formally assessed.

While there is plentiful evidence showing that high job strain is related to ill-health (for review, see Amiri & Behnezhad, 2020) and some showing that coping resources do moderate the link between work stress and sleep (Åkerstedt et al., 2002; Sadeh, Keinan, & Daon, 2004), there are few studies that document the moderating effects of (the different types) of PC and job stain on the other health outcomes explored in this study (i.e., burnout; somatization; health behaviours) and what we could find was not in agreement and assessed largely homogeneous samples (i.e., school teachers). For example, Pieper et al. (2007) found that teachers reporting high job strain displayed elevated cardiac activity that was no different to teachers reporting low job strain, nor did they report daily worry episodes more frequently. Whereas earlier reports by Cropley et al. (1999) demonstrated that schoolteachers with high job strain were around twice as likely to experience worry and nearly two and half times more likely to report somatic symptoms. Further, in a recent review of 12 studies concluding that job strain is associated with lower psychological detachment from work, the authors pointed out the requirement for future work to consider other modes of perseverative thinking and its impact on a diversification of health outcomes (Türktorun, Weiher & Horz, 2020).

The present study did, however, find significant mediation effects for the associations between PC and poorer health outcomes. Our findings (in particular the mediation models) support the theoretical link between job strain, different types of PC, and burnout, somatization and sleep quality. Indeed, with the exception of the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were statistically significant. Not only does this finding broadly support Hypothesis 3 but it is consistent with a range of empirical evidence (for reviews, see; Clancy et al., 2016; Clancy et al., 2020; Ottaviani et al., 2018; Verkuil, Brosschot, Gebhardt & Thayer, 2010) and theoretical considerations advocating the causal chain through which PC influences health (see Brosschot et al., 2005 for the original PC Hypothesis & O'Connor,

Thayer & Vedhara, 2021). While it might appear axiomatic that people with high job strain, and who engage in worry and/or rumination are more prone to burnout, somatization and poorer sleep, to the best of our knowledge, this is the first time this relationship has been examined collectively.

The main findings of the current study also have implications for interventions looking to reduce the negative effects of PC on health and wellbeing. In particular, our multi-mediator models and sensitivity analyses (see Appendix 1.1.3) suggest that targeting both worry and rumination may produce more positive outcomes than targeting either alone. Further research should be conducted to identify the types of techniques that best influence worry and rumination. As noted by McCarrick, Prudenzi, Prestwich and O'Connor's (2021) review of the experimental literature, relatively few intervention techniques have been used to try reduce PC with only 7 broad types of intervention identified and worry and/or rumination were rarely considered as a primary outcome in studies. Moreover, it is not known, how well specific techniques work for work-related worry and rumination explicitly. Nevertheless, the results of the present study highlight the need for future work-based studies considering health to examine maladaptive cognitive processes, such as PC; especially considering work-related PC has now also been shown to play a damaging role in both general and work-related and general distress (see Prudenzi et al., 2021).

This study is not without its limitations. First, this study relies exclusively on self-report measures to assess both its predictor and outcome variables. Several problems have been associated with the use of self-report measures such as social desirability or retrospection (Manag, 1986). Future studies should therefore seek to not solely rely on self-report tools, but to use objective methods to assess, for instance, at least the outcome measures for health. For example, one could use actigraphy (see Van Laetham et al., 2013) to assess sleep quality or use daily-diary style methods to capture individual types of health behaviours (e.g., Clancy, Prestwich & O'Connor, 2020). In addition, we also recognise the limitations of using a cross-sectional design in terms temporal validity and issues relating to causality

(e.g., Maxwell, Cole & Mitchell, 2011). Therefore, future research ought to attempt to replicate the current findings using large scale longitudinal designs. Quasi-experimental field studies may also represent a fruitful avenue of future research to help understand the bidirectional relationships between stress, PC, work-related PC, and health outcomes. In addition, the work-based and general PC measures were not entirely matched with differences across items not just related to context (workplace vs. general). As such, differences in work-based and general PC findings cannot be entirely attributable to differences in context. Finally, while the composite measure used to tap health behaviours is useful for broadly understanding the relationships between PC and health behaviours overall, stronger relationships may arise with specific health behaviours (e.g., sleep, Radstaak et al., 2014; unhealthy snacking, Eschle & McCarrick, 2021) that are not detectable in the composite measure we used. Future studies should therefore look to explore the interrelationships between PC and individual health behaviours, ideally via prospective study designs (e.g., McCarrick, Prestwich & O'Connor, 2022).

To conclude, the present study provides supportive evidence for the PC Hypothesis and the role worry and rumination play as related, yet distinct, cognitive processes in contributing to ill-health. Both types of general *and* work-related worry and rumination predicted significantly higher scores in burnout and somatization, as well as lower scores in sleep quality, but further work is needed to understand the role of health behaviours. Job strain significantly predicted greater burnout across each analysis containing the different types of PC, but none of the PC measures (general or work-related) interacted with job strain to predict any of the health outcome variables. However, for the mediation analyses, with the exception of the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were statistically significant implying the additive roles of different types of PC. Therefore, taken together, the results of the present

study provide new evidence for the damaging nature of PC and its role as an important mediating factor between stress and health-related outcomes.

Chapter 3

Health Effects of Psychological Interventions for worry and rumination: *a meta-analysis*

3.1 Introduction

Psychological stress has consistently been related to negative health outcomes, with recent figures suggesting stress-related health care costs an estimated $300 billion per annum (American Institute for Stress, 2020). Indeed, the impact of psychological stress, that is, when the appraisal processes attached to a threat or experience exceeds an individual's perceived coping ability, has long been implicated in a variety of health and illness outcomes (e.g. neurotic symptoms, House et al., 1979; organ damage, Plante, 2002; cardiovascular disorders, Lundberg, 2005; migraines, Schoonman., 2007; diabetes, Öhman, Bergdahl, Nyberg & Nilsson, 2007; for a review see O'Connor, Thayer & Vedhara, in press). Whether directly through autonomic and neuroendocrine responses or indirectly, via changes[1] in health behaviours (Christiansen, Larsen & Lasgaard, 2016, Jones & Bright, 2007, O'Connor, Thayer & Vedhara, in press), adverse health outcomes have been noted to be of direct consequence to stress, even when the stressor is no longer present (Brosschot et al., 2006). In particular, perseverative cognition (PC) has been identified as an important mechanism that may help explain how stressful events and encounters increase the risk of ill-health and poor wellbeing. PC is thus defined as any type of stress-related, negative, repetitive thought and encompasses thoughts about feared future events (worry) and thoughts and negative feelings about distressing past experiences (rumination).

In the original perseverative cognition hypothesis (PC hypothesis), Brosschot et al. (2006) suggested that stressful thoughts activate the body's stress response in the same way as stressors in the physical environment and serve to prolong the hypothalamic-pituitary-adrenal-axis stress response. Since then, several key reviews have shown that PC is associated with a range of physiological health outcomes; including higher blood pressure

and heart rate, lower heart rate variability, as well increased cardiovascular activity, reduced secretion of antibody productions, blunted cortisol response and increased levels of somatization (for reviews, see Ottaviani et al., 2018; Verkuil, Brosschot, Gebhardt & Thayer, 2010).

Aside from evidence connecting PC with physical health, emerging work suggests PC can influence a variety of health behaviours including sleep, diet and alcohol consumption (Clancy, Prestwich, Caperon & O'Connor, 2016; Cropley et al., 2012; Frone, 2015). Importantly, these negative health behaviours are related with illness (Suris & Parera, 2005), disease and morbidity rates (Burke et al., 2007), in both adults and children cross-culturally (for review, see Mackenbach, 2014). Notably, in a meta-analytic review of health behaviours across 19 studies, Clancy et al. (2016) showed that higher levels of PC were associated with significantly more health risk behaviours. In particular, these authors found that PC was associated with greater substance use, unhealthy eating and smoking. Taken together, these findings provided evidence for an extended PC hypothesis, such that there may be scope for an additional route to pathogenic disease via poorer health behaviours.

However, the evidence base discussed thus far for the impact of PC on both health behaviours and physical health outcomes is mostly based on correlational methodologies. Reliance on this type of evidence has a number of issues as: (a) it does not account for the likelihood that negative health-outcomes may trigger variations in measures of PC and/or vice-versa; (b) it overlooks consistency biases that may inflate the strength of the relationship between stress and health outcomes, as shown in previous work (see, Arkin, Gabrenya, Appelman & Cochran, 1979; Renner, Laux, Schütz & Tedeschi, 2004); and (c) it disregards statistical considerations around the important role(s) of confounding variables on the PC and health outcome relationship; meaning the impact of a third variable, or 'spuriousness', is often not accounted for in analyses (see, Kenny, 1979; Mauro, 1990). An alternative, more valid way to strive towards understanding causality would be to observe studies whereby an experimental manipulation brings about statistically significant

differences in PC between intervention and control arms after exposure to some level of intervention, while observing the same between group differences with subsequent measures of health. This approach can be considered superior to correlational tests as: (a) standardized differences between intervention arms within measures of PC (particularly when assessed early) are attributable to an experimental manipulation and thus are not based on deviations in health accrued later; and (b) random assignment of participants to condition help to account for the influence of extraneous variables and potential biases.

A number of techniques have been used in an attempt to influence PC (e.g. mindfulness, Garland, 2011; relaxation, Andersson et al., 2012; action planning, Versluis, Verkuil, Spinhoven & Brosschot, 2018), however, these are small in number and there are few, if any, that observe health consequences. Querstret and Cropley (2013) represent the only available review exploring how PC might be reduced via psychological interventions. Across nineteen studies, comprising both face-to-face and internet-delivery formats, interventions in which participants were encouraged to detach themselves from emotional responses to PC and adopt more concrete or re-constructive ways of thinking, were reported as most promising. However, few studies in the Querstret and Cropley review were explicitly designed to target PC, it only includes studies between 2002 and 2012; and, most importantly, it did not consider the impact of changing PC on health outcomes. An up-to-date evaluation of current studies which provides a quantitative estimate of the effectiveness of interventions for reducing PC, while also accounting for moderating factors and health consequences, is thus timely and warranted.

3.1.1 The present review

Evidence for the PC-health outcome relationship has tended to be based on correlational evidence (for reviews, see Ottaviani et al., 2016; Clancy et al., 2016) and a review has not been conducted to identify the best approaches to reduce PC in a health context that captures the consequences of changing PC on health behaviours and physical health

outcomes. Thus, using the available experimental literature, in this review we examined whether: PC can be influenced by interventions (Objective 1a); and, if so, which intervention or study characteristics, following exposure to intervention content, produce larger effect sizes for PC (Objective 1b); interventions that target PC also impact health outcomes (Objective 2a); and, if so, which intervention or study characteristics, at post-intervention, produce larger effect sizes for health (Objective 2b); larger effect sizes for PC are also associated with larger, but positive, effect sizes for health outcomes at post-intervention (Objective 3). Across these objectives, PC was considered at three levels (worry, rumination and both PC types combined) and health outcomes were explored across two levels (health behaviours, physical health outcomes). Sleep (the most popular health outcome) and a composite measure for both types of health outcomes (behaviours and physical health combined, health *overall*) were also considered but these findings are reported in the Appendix section (see Appendix 2.2).

3.2 Method

This review was pre-registered with PROSPERO (CRD42019119381) and is available on the Open Science Framework (see, https://bit.ly/35X81xi).

3.2.1 Eligibility Criteria

To be eligible, studies had to: (1) involve the random assignment of participants to a treatment group that received a psychological intervention targeted at PC or to a control group who received either a control intervention or no intervention, (2) include a measure of perseverative cognition (worry and/or rumination) after exposure to an intervention, (3) contain measures of either physical health outcomes and/or health behaviours, at follow up (to reflect the PC hypothesis). Studies were excluded if: (1) they had a non-human (animal) sample, (2) they were an existing review/meta-analysis, (3) if any aspect of the intervention was pharmacological (i.e. to test the effects of a drug), or (4) participants were specifically recruited on the basis of a learning disabilities/intellectual disorders (e.g., cerebral palsy, autism, epilepsy) severe alcohol and/or substance dependency (i.e., based on author

classifications as per standardized measures), or severe psychiatric disorders (e.g., schizophrenia, bipolar disorder, depression with psychotic symptoms, psychosis, serious suicidal thoughts). However, because Generalised Anxiety Disorder (GAD) has several temporal and theoretical properties relating to PC (e.g., repetitive negative thinking, constant worrying), studies whose participants had a diagnosis of GAD (*N* = 2) were included; so long as they did not have other severe comorbid mental health disorders akin to those described above. Studies comprising participants with sleep disturbances (i.e., insomnia, *N* = 4) were also included, as we were interested in the effects of PC on parameters of sleep.

Pharmacological based interventions were not included for two main reasons. First, such interventions are very different to the psychological therapies included in this review as they trigger change at the neuroendocrinological level that are out of the control of the participant; i.e. taking a pill/tablet is not comparable to offering people a strategy to control their worry. Whereas, all the studies within our inclusion criteria offered participants a conscious opportunity to tackle their PC. Second, the participants included in pharmacological studies typically derive from samples which have several co-morbid issues that may interfere with the PC-health outcome relationship.

3.2.2 Search Strategy

Three databases were searched to maximize search sensitivity (see Montori et al., 2005): PsycINFO (1806 – present) and Medline (1806 – present) via OVID, and CINAHL (1960-present) using EBSCO. The search was last conducted on the 23rd November 2019 with search terms relating to perseverative cognition, and randomized interventions. Perseverative cognition search terms were adapted from Clancy et al. (2016). Specifically, “negative and (thought or thinking)” was removed to enhance specificity; “perseverati” with “cogniti” was replaced with “perseverative and (thought* or thinking or cognition*)”. The Eady et al. (2008) RCT filter (random*.tw) was employed as a single term to capture the best optimisation of sensitivity and specificity, complimented with the term (intervention*.tw) to

enhance sensitivity. Further, to maximise sensitivity (at the expense of specificity), search terms were not generated for health outcomes. The search was limited by the English language and human studies but not by year (see, Appendix 2.1.1). Titles, abstracts, and full-text screening were completed by the first author. The third author independently screened the titles and abstracts using a subset of 1070 studies (20% of total) (Cohen's kappa = .91). Any discrepancies were discussed and resolved. Any study identified as potentially eligible at the abstract screening stage was progressed to full-text screening. The first author then independently assessed all full-texts with 40% of full-texts independently double-screened by the third author (Cohen's kappa = .98). Discrepancies were then discussed and verbally agreed upon between both authors. Across the sets of double-screened studies, the secondary coder did not identify any eligible studies missed by the primary coder.

3.2.3 Data Extraction & Data Coding

The subsequent data were extracted and coded for each study: lead author name, publication year, country, study design (RCT or cluster RCT), measurement points (in days) for PC and health outcomes, type of PC (worry or rumination), measurement of PC and health outcomes (i.e. self-report vs non-self-report), health outcome type (behavioural or physical), participant characteristics: age, percentage female, GAD diagnosis, sleep disturbance, and number of participants included in analysis and attrition (across the entire study). We recognise health outcomes is a broad term, though for the purposes of this review, we defined health behaviours a-priori as an action(s) to maintain, attain, or regain good health and to prevent illness (Conner & Norman, 2005) and physical health outcomes as any marker indicative of, or which would impede, impact or constrain routine physiological functioning (e.g., neurological, circulatory, endocrinological, immune, digestive, muscular systems) (Corbin, Pangrazi & Franks, 2000).

The following main intervention types were extracted: pain management, PC action plans (i.e., planning interventions to help better manage PC), stress management (i.e., broad ranging therapies concerned with eliminating stress), mindfulness and relaxation (i.e., refocusing on the present moment), psychological detachment (i.e., 'switching off' from situations, such as work, that trigger negative affect), Cognitive Behavioural Therapies (CBT) and Acceptance and Commitment Therapies (ACT) (i.e., challenging unhelpful thoughts and engendering self-help strategies) and expressive writing (i.e., disclosing one's deepest thoughts and feelings). Other features of the intervention: duration (in days), number of sessions, weeks delivered across, delivery format (group or individual), mode of delivery (health-care professional, self-administered, trained facilitator) and if the intervention was delivered online or delivered in-person was also assessed. Study setting was also evaluated. Studies were classified as medical if they took place within a hospital or health-care environment, educational if within a school, or academic if they took place within a university or research unit.

Study quality and risk of bias were assessed using all items from Cochrane's Risk of Bias tool (Higgins et al., 2011), including selective outcome reporting and extra bias sources. Other important methodological or statistical features (e.g., using validated measures, reporting of satisfactory levels of internal consistency, baseline differences between groups) and if studies incorporated intention-to-treat analysis (ITT) were also considered. We approached data extraction in two phases to minimise the possibility of coding errors. The first phase was piloted on 10% of the studies in a 'training phase". For this piloted 10%, the coding for all measures was checked by a second reviewer. Inter-rater agreement levels were classified as near-perfect for items relating to health outcomes and PC (Cohen's kappa = .75 - .1) and often perfect for items relating to risk of bias and other study characterises (i.e., population, attrition, design, measure timing) (all kappas >.92; Landis & Koch, 1977). Second, we operated a 'validation phase' whereby data for all studies was first extracted by a primary coder before an extra 20% of studies were independently assessed by a second

coder. For this phase, agreement between coders was near perfect across all study items (Cohens kappa = .97 - .1). In all cases, if either coder was in any doubt, the study authors were contacted for additional clarification before making deciding upon eligibility.

3.2.4 Data Synthesis

Effect sizes were calculated based on means and standard deviations and, when not available (*k* = 6), using other statistics reported (i.e. *F* and *p* values). Effect sizes were calculated for *PC overall* (worry, rumination and measures of perseverative thinking combined), for worry and rumination separately, for health behaviours and physical health outcomes separately, as well as for sleep as it was the most common health outcome (77.3% of studies) (note, we view sleep as a health behaviour as it is an action that is under volitional control). Results pertaining to health outcomes *overall* (i.e., physical health and behaviours combined) are reported in Appendix 2.1 and 2.2. Standard errors were adjusted to account for clustering in relevant studies (*k* = 3) (see Higgins, Deeks, & Altman, 2008). Hedges' *g* was used as the main effect-size measure (see Appendix 2.2 for Hartung-Knapp-Sidik-Jonkman method) as it provides an unbiased estimate of effects (Hedges & Olkin, 1985).

When more than one intervention group was present (*k* = 5), there were four cases where we selected the arm which authors stated, or hypothesised, would outperform the other arms. However, as this was not made clear in one study (Topper et al., 2017), to avoid including the same participants more than once within the meta-analysis (to avoid unit-of-analysis error) and because the primary aim of this review was to identify the most effective methods of influencing PC, the intervention that generated the largest effect on PC was selected. For the selection of comparator groups there was just one study whereby there was more than one comparison group present (i.e., 'waitlist' vs. 'standard control'; Versluis et al., 2018). In this case, the 'standard control' was selected for our analyses because: a)

authors hypothesized that the ‘standard control’ would be more likely to reduce PC than the ‘waitlist’ and, b) because the ‘standard control’ in this particular study contained all the features of an attention-placebo control (i.e., an intervention that mimics the theoretically inactive elements, but not the active elements) which are regarded as highly valid control groups (Popp & Schneider, 2015).

Effect sizes were calculated using the first measure of PC following exposure to an intervention and the final measure of health reported in each study. We used this approach because the temporal relationship that was of primary interest was from PC to health rather than vice-versa and because the impact of interventions on PC was more likely to be detected at this initial time point (i.e., after intervention exposure), rather than in later follow-ups (i.e., in a number of weeks/months). We did not consider baseline scores within the calculation of study effect sizes because data was not always available for baseline assessments across the included studies and none of the studies reported pre-post correlations on the dependent variable which are used in the calculation of these effect sizes. Given concerns regarding additional heterogeneity with baseline scores being reported for some studies but not others, and the need to estimate correlations, effect sizes were based only on post-intervention scores. In cases where there were multiple measures of the same construct (e.g. two questionnaires for worry, total sleep time and sleep onset latency) the effect sizes were calculated and then averaged using a random effects model. All analyses were exclusively between conditions (treatment vs control) and none were within conditions.

STATA (version 13) was used to conduct random-effects meta-analyses (to produce effect size estimates for the effect of interventions on influencing PC (objective 1a) and impacting health outcomes (objective 2a). STATA was also used for sub-group analysis and meta-regressions; to assess whether the presence or absence of specific study or intervention characteristics were associated with: larger effect sizes for PC (objective 1b) and for health

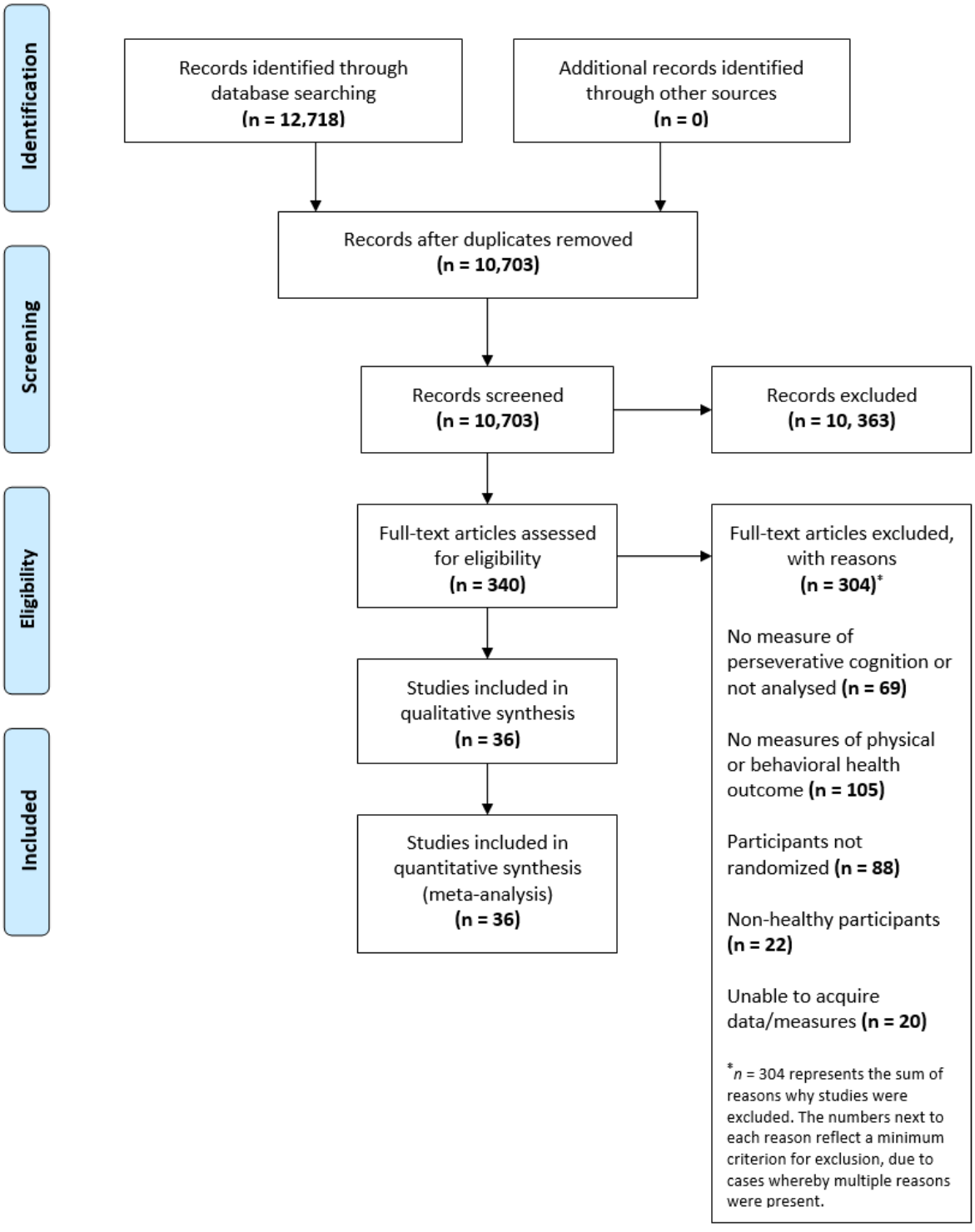
outcomes (objective 2b), as well as the association between larger effect sizes for PC and effect sizes for health outcomes at post-intervention (Objective 3). For this latter objective, the 'Metafor' package (Viechtbauer, 2010) in *R* was used to conduct permutation test(s) with 10,000 random interactions to test the robustness of effects. The package was also used to test for potential influential cases and/or outliers (using the 'influence' function) (in addition to visual plot inspections) in the relevant sensitivity analyses. All meta-regressions were univariate, except to test for confounding between two significant moderators (these exceptions can be found in Appendix 2.2).

A range of additional analyses were conducted to: (a) check data met the statistical assumptions associated with regression such as multivariate normality, low multicollinearity, lack of auto-correlation and homoscedasticity; (b) identify potential confounds that may have affected the conclusions and consider the results when the behavioural and physical health outcomes were combined as an *overall* health index; (c) assess the possible impact of two studies for which we had concerns regarding the measures of behaviour; assess the robustness of the findings when focused only on studies (d) measuring PC *immediately* post-intervention and then health at a *later* point in time and (e) measured sleep; (f) check for small-study bias; (g) assess, when an alternative study arm was available (i.e., two treatment arms/different control types), if our approach to arm selection significantly altered study effect sizes for both PC and health; h) control for the possibility that baseline between group differences influenced effect sizes; i) detect if clinical heterogeneity influenced effect sizes. The results of these ten sets of additional analyses are reported in Appendix 2.2.

3.3 Results

Studies considered for inclusion in the review are displayed in Figure 3.1. Thirty-six studies met the inclusion/exclusion criteria. Nineteen studies included measures of worry (52.7%), 9 included measures of rumination (25%) and 11 measured perseverative thinking (a composite measure of worry and rumination) (30.5%). Of these studies, two included measures of both worry and rumination (Ebert et al., 2015; Thiart, Ebert & Riper, 2015) and one study (Topper et al., 2017) included measures of worry, rumination and perseverative thinking. Regarding health outcomes, 21 studies (58.3%) included measures of physical health and health behaviours, and, of these, 6 studies included measures of both a health behaviour and physical health outcome (6%). Of all health behaviours, sleep was the most common (*k* = 17, 77.3%) and, of all physical health outcomes, pain (*k* = 3, 14.3%) was the most common.

Figure 3.1. PRISMA diagram for included studies.



3.3.1 Study Characteristics

The characteristics of included studies are summarized in below in Table 3.1. All studies were RCTs (3 cluster-trials, 33 non-cluster trials). Twenty-one studies (58.3%) obtained participants from academic research settings, seven (19.4%) sourced participants from educational environments (i.e., schools) and 8 (22.2%) drew participants from medical settings (e.g. hospitals; clinics). Nine (25%) utilised a student sample and, on average, 70.4% of participants were female. Thirty-one studies (86.1%) recruited adults (aged 18 or over) and 5 (13.8%) obtained samples of school children. Studies were conducted across 9 countries, though the most common were the USA (*k* = 9, 25%), Netherlands (*k* = 8, 22.2%) and Germany (*k* = 7, 19.4%). The mean age of all participants (*n* = 5098) was 36.52 years (*SD* = 14.32) and the average number of participants in each study, across all studies, was 142 (*SD* = 53.88). Two studies (5.5%) recruited their participants on the basis of a GAD diagnosis and a further four (11.1%) studies had participants which reported sleep disturbance (i.e., insomnia).

Table 3.1. Overview of included studies (k = 36)

Lead Author, year	Design	Location & Setting	Intervention features (*n* treatment sessions/ delivery across weeks.	PC & HO Measurement points (days after intervention exposure)	Type of PC (& measure)	Type of health outcome (& measure)	Participant characteristics	Pps included in analysis (*k*) & mean age (& *SD*)	% Female	Attrition (across entire study)
Aardoom et al., 2016	Randomized controlled trial	Netherlands, Educational	Stress management (8/8): Online based psychoeducation intervention.	PC: 56 HO: 91	Perseverative thinking (PTQ)	Binge eating (EDE-Q).	Opportunity sample of adults with dietary concerns.	*k* = 178, *M* = 24.2 (*SD* = 7.7)	98.9%	63.3%
Abbasi et al., 2012	Randomized controlled trial	Israel, Medical	Pain management (7/7): In person spouse-assisted programme to alleviate back pain.	PC: 49 HO: 365	Health Rumination (PCS - rumination subscale)	Physical pain (TSK; RDQ; VAS (1-10) of pain intensity for the week)	Referred to the GP with lower back pain of greater than 6 months duration.	*k* = 21, *M* = 45 (*SD* = 10)	87.88%	10%
Brosschot et al., 2006	Cluster randomized controlled trial	Netherlands, Educational	PC action plans (6/<1): In person Diary based worry postponement.	PC: 7 HO: 7	Worry (PSWQ & tally of daily worry).	Physical health complaints (SCH)	Volunteer sample of final grade high school students from 25 different schools.	*k* = 171, *M* = 16.7 *(range:* 15 – 19)	81.4%	29%
Buntrock et al., 2015	Randomized controlled trail	Germany, Academic	CBT (6/3): Online CBT to prevent relapse into depression	PC: 42 HO: 183	Worry (PSWQ)	Insomnia severity (ISI) & functional impairment (SF-12v1)	Volunteer sample adults with minor depression.	*K* = 366*, M, =* 45 *(SD* = 11.9*)*	73.9%	19.9%
Buntrock et al., 2016	Randomized controlled trail	Germany, Academic	CBT (6/<1): Online CBT to prevent relapse into depression	PC:40 HO:365	Worry (PSWQ)	Insomnia severity (ISI)	Volunteer sample of adults with minor depression.	*k* = 336*, M* = 45 *(SD* = 11.9*)*	73.9%	Not reported.
Carney & Waters, 2006	Randomized controlled trail	USA, Academic	PC action plans (6/4): In person experimental pre-sleep constructive worry intervention.	PC: 7 HO: 7	Worry (PSWQ; WDQ; PSAS-worry subscale).	Sleep (SOL, TST, TWT).	University students with the presence of 3 or more nights per week of sleep onset difficulty.	*k* = 33, *M* = 20.97 (*SD* = 3)	78.78%	3.1%
Christiansen et al., 2014	Randomized controlled trial	Australia, Academic	CBT (10/10): Online CBT programme to reduce anxiety.	PC: 77 HO: 183	Worry (PSWQ)	Alcohol dependence (AUDIT)	GP referred with elevated anxiety.	*k* = 133, *M* = 25.7 (*SD* = 3.1)	82.9%	35%

Conrad et al., 2008	Randomized controlled trial	America, Medical	Mindfulness & Relaxation (12/12): In person applied relaxation to reduce worry.	PC: 7 HO: 7	Worry (PSWQ)	Somatization (CSAI, somatic subscale)	Self-enrolled individuals with GAD.	*k* = 33, *M* = 44.6 (*SD* = 12.8)	59%	38%
Crain et al., 2017	Randomized controlled trial	Canada/USA; Academic	Mindfulness & Relaxation (11/8): In person group based mindfulness sessions.	PC: 91 HO: 152	Job rumination (2 Likert scales, from teacher stress scale)	Sleep (Likert scales on sleep quality, sleep quantity & daytime sleepiness).	Self-enrolling public school teachers.	*k* = 113, *M* = 46.9 (*SD* = 9.2)	89%	Not reported
Digdon & Koble, 2011	Randomized controlled trial	Canada, Academic	PC action plans (7/<1): Online constructive worry sessions to help with pre-sleep worry.	PC: 7 HO: 7	Worry (daily sleep log; PSAS, worry subscale)	Sleep (SQS; sleep onset latency, sleep quantity and sleep quality) & somatic complaints (PSAS, somatic subscale).	Self-enrolled undergraduate students with pre-sleep worries.	*k* = 22, *M* = 23.22 (*SD* = 6.11)	78.05%	51.2%
Ebert et al., 2014	Randomized controlled trial	Germany, Educational	Stress management (5/7): Online based, virtual instructor lead, problem solving therapy.	PC: 49 HO: 183	Worry (PSWQ)	Burnout (MBI-D) & physical health (SF-12-PCS subscale).	School teachers with minor depression.	*k* = 150, *M* = 47.1 (*SD* = 8.2)	83.3%	15.3%
Ebert et al., 2015	Randomized controlled trial	Germany, Educational	Detachment (6/8): Online based recovery training on work related stress.	PC: 56 HO: 56	Worry (PSWQ-PW) & work related rumination (CI, rumination subscale)	Sleep (PSQI, ISI, SSI & GSI).	School teachers experiencing poor sleep and low levels of detachment from work.	*k* = 100, *M* = 48.5 (*SD* = 9.9)	74.2%	31.17%
Ebert et al., 2016	Randomized controlled trial	Germany, Academic	Detachment (7/7): Online based, e-coach led, work detachment stress-management sessions.	PC: 49 HO: 183	Worry (PSWQ-PW)	Sleep (ISI) & burnout (MBI· emotional exhaustion subscale) & physical health complaints (SF-12)	General population with elevated symptoms of stress.	*k* = 249, *M* = 42.9 (SD = 9.8)	85.9%	50.8%

Eilenberg et al., 2016	Randomized controlled trial	Denmark, Medical	CBT (9/9): In person ACT to help with health anxiety	PC: 304 HO: 304	Illness worry (IWS)	Somatic symptoms (90-item Symptom Checklist & SCL - somatization subscale).	Opportunity sample of patients with health anxiety.	*k* = 107, *M* = 36.23 (*SD* = 8.75*)*	67%	6%
Freshour et al., 2016	Randomized controlled trial	USA, Medical	CBT (10/24): In person therapist led CBT reduce anxiety.	PC: 70 HO: 365	Worry (PSWQ)	Patient health (PHQ-8)	Later-life individuals with GAD.	*k* = 224, *M* = 66.83 (*SD* = 6.38)	54.57%	12.5%
Harvey et al., 2017	Randomized controlled trial	USA, Medical	CBT (8/8): In person CBT for chronic insomnia.	PC: 56 HO:183	Pre-sleep worry (APSQ)	Insomnia severity (ISI) and sleep diary (BTv, RTv, TIB).	Self-referred individuals with moderate insomnia.	*k* = 128, *M* = 47.4 (*SD* = 12.6)	62.23%	7.5%
Hazlett-Stevens & Oren, 2017	Randomized controlled trial	USA, Academic	Mindfulness & relaxation (10/10): In person reflection and mindfulness workshops.	PC: 70 HO: 70	Worry (PSWQ)	Physical health (WHOQOLBREF, physical subscale).	Self-enrolled students seeking stress reduction.	*k* = 68, *M* = 22.1 (*SD* = 4.7)	75%	26.1%
Jansson-Frojmark et al., 2012	Randomized controlled trial	Sweden, Academic	PC action plans (4/4): In person worry construction and behavioural therapy to aid with sleep.	PC: 7 HO: 14	Pre-sleep worry (APSQ)	Insomnia severity (ISI)	Self-enrolled individuals with primary insomnia from local care centres.	*k* = 21, *M* = 56.5 (*SD* = 12.7)	52.5%	9.1%
Jellesma et al., 2009	Cluster randomized controlled trial	Netherlands, Educational	PC action plans (7<1): In person worry postponement to stop night time worriers..	PC: 7 HO: 7	Perseverative thoughts (CERQ-K, nightly tally)	Somatic complaints (SCL)	Children from grades 7 and 8 from seven primary schools.	*k* = 227, *M* = 11.4 (*SD* = .70)	56.83%	15.4%
Lokman et al., 2017	Randomized controlled trial	Netherlands, Educational	CBT (7/4): Online CBT self-help to improve sleep and wellbeing.	PC: 91.25 HO:91.25	Worry (PSWQ)	Sleep quality (JSEQ)	Self-enrolled individuals with mild depressive symptoms.	*k* = 237, *M* = 43 (*SD* = 12.93)	75.7%	54.4%
Magan et al., 2014	Randomized controlled trial	USA, Academic	PC action plans (14/2): Online constructive plans on smoking-related consequences,	PC: 14 HO: 14	Worry (PSWQ, 2 Likert items on smoking worry)	Smoking addiction (FTND-R, and mean number of cigarettes smoked per	Volunteer sample of university students who smoke on a daily basis.	*k* = 117, *M* = 29.6 (*SD* = 12.9)	44.4%	Not reported.

			negative thoughts and worry prevention.			week at baseline, compared to post-intervention)				
McGowan & Behar, 2013	Randomized controlled trial	USA, Academic	PC action plans (14/2): In person focused worry postponement to reduce anxiety.	PC: 14 HO: 14	Worry (PSWQ)	Insomnia severity (ISI)	Volunteer sample of university students/ are high trait worriers	*k* = 46, *M* = 19.9 (*SD* = 3.8)	82.6%	16.9%
Mehlsen et al., 2017	Randomized controlled trial	Denmark, Medical	Pain management (6/6): In person, therapist led, chronic pain self-management programme to improve wellbeing.	PC: 63 HO: 152	Illness worries (Whiteley-7)	Physical health symptoms (SCL) & bodily pain (RDQ, a 1-100 pain intensity VAS).	Individuals with chronic pain for longer than 3 months from 75 different hospitals.	*k* = 399, *M* = 54 (*SD* = 13.05)	72%	8%
Michailidis and Cropley, 2019	Randomized controlled trial	England, Academic	Expressive writing (3/<1): In person self-guided, expressive writing to reduce work-related rumination.	PC: 31 HO: 91	Work-related rumination (WRRQ)	Sleep quality (ISI)	Full-time adult employees working in the UK from a wide range of occupations	*k* = 47, *M* = 34.22 (*SD* = 11.39)	50%	49%
Pech & O'Kearney, 2013	Randomized controlled trial	Australia, Academic	Stress management (5/6): In person problem solving therapy to reduce stress and improve sleep quality.	PC: 7 HO: 70	Worry (PSWQ)	Sleep quality (PSQI) and insomnia severity (ISI).	Individuals with primary insomnia for longer than 3 months.	*k* = 47, *M* = 39.21 (*Range:* 18-60)	62.8%	14.9%
Peters et al., 2017	Randomized controlled trial	Netherlands/Belgium, Medical	CBT (8/8): Online CBT to reduce pain and intrusive thoughts.	PC: 65 HO: 65	Perseverative thinking (PTQ)	Bodily pain (Likert 1-10 rating of pain intensity)	Volunteer sample of adults who had experienced musculoskeletal pain for longer than 3 months	*k* = 162, *M* = 48.6 (*SD* = 12)	85%	25.4%

Querstret et al., 2017	Randomized controlled trial	England, Educational	Mindfulness (10/4): Online instructor-led, mindfulness to reduce work-related rumination/fatigue.	PC: 28 HO: 183	Work-related rumination (WRRQ)	Sleep quality (PSQI) & work-related fatigue (OFER, 2 subscales for chronic fatigue & acute fatigue)	Self-enrolling working adults with elevated levels of work-related rumination	*k* = 87, *M* = 40.68 (*SD* = 10.45)	80.5%	25%
Sabinga et al., 2013	Randomized controlled trial	USA, Educational	Mindfulness (12/12): In person, instructor led, mindfulness based stress reduction to improve sleep and reduce negative physical health.	PC: 84 HO: 84	Rumination (AMR, mindfulness inventory, rumination subscale)	Sleep quality (nightly sleep diary, and via ACTigraph 24 h/day during the 1-week).	Self-enrolling 7th and 8th grade boys at urban middle school.	*k* = 41, M = 12.5 (*range* 11–14)	0% (all male)	2.38%
Sabinga et al., 2016	Cluster randomized controlled trial	USA, Educational	Mindfulness (12/12): In person, instructor led, mindfulness based stress reduction to improve physical health and reduce rumination.	PC: 84 HO: 84	Rumination (CRSQ, rumination subscale)	Somatization symptoms (SCL)	Volunteer sample of 5th to 8th grade students in two public schools.	*k* = 300, *M* = 12 (unclear)	50.7%	Unclear: between 25.2% and 27.2%
Sandlund et al., 2018	Randomized controlled trial	Sweden, Medical	CBT (6/10): In person, nurse-led CBT to improve daytime symptomology of insomnia.	PC: 70 HO: 70	Pre-sleep worry (1-100 VAS)	Sleep quality (USI, ISI)	Volunteer Individuals with primary insomnia.	*k* = 132, *M* = 54 (*SD* = 16)	72.7%	20%
Teismann et al., 2014	Randomized controlled trial	Germany, Academic	Expressive writing (3/<1): In person, diary based, self-guided positive writing about personal life goals	PC: 3 HO: 3	Perseverative thinking (PTQ)	Cortisol awakening response (CAR)	Volunteer sample of general population.	*k* = 64, *M* = 29.1 (*SD* = 8.42)	62.5%	0% (4 sets of missing data were excluded)
Thiart et al., 2015	Randomized controlled trial	Germany, Academic	CBT (6/8): Online, mixed intervention	PC: 56 HO: 182	Worry (PSWQ) & work-related rumination (IS,	Insomnia severity (ISI) &	Volunteer sample of school teachers	*k* = 118, M = 48 (*SD* = 9.9)	74.2%	7.2%

			based on CBT principles to improve wellbeing and sleep quality.		cognitive irritation subscale)	recuperation in sleep (SF-AR)	with sleep complaints.			
Topper et al., 2017	Randomized controlled trial	Netherlands, Academic	CBT (6/6): Online, group based, CBT to prevent anxiety and depression	PC: 56 HO: 365	Worry (PSWQ), rumination (RRS) & perseverative thinking (PTQ)	Alcohol consumption (QDS) & dietary screening (EDI-2-BU)	Self-enrolled high school children from final three grades in 13 schools.	*k* = 150, *M* = 17.43 (*SD* = 2.09)	83.7%	17%
Versluis et al., 2016	Randomized controlled trial	Netherlands, Academic	PC action plans (6/<1): Online, worry postponement to reduce health complaints.	PC: 6 HO 6	Worry (nightly diary for duration and frequency)	Subjective health complaints (SHC)	Volunteer sample of general population.	*k* = 351, *M* = 36.36 (*SD* = 12.97)	84.76%	64%
Versluis et al., 2018	Randomized controlled trial	Netherlands, Academic	PC action plans (26/4): Smartphone-based, self-guided, worry-reduction training for stress reduction and emotion regulation.	PC: 14 HO: 27	Worry (PSWQ & nightly diary recording of: duration, frequency, severity)	Cardiac activity (ambulatory measured continuously for the three test days via an ekgMove sensor).	Volunteer sample of adults who reported elevated levels of work-based stress	*k* = 79, *M* = 43.60 (*SD* = 11.39)	74%	8%
Woilizky-Taylor et al., 2010	Randomized controlled trial	USA, Academic	Mindfulness and relaxation (12/4): In person, pulsed audio-photic stimulation for relaxation to reduce worry.	PC: 12 HO: 12	Worry (PSWQ & AQW)	General health (visits to health centres in the past semester).	Self-enrolled sample of university students concerned about assessments.	*k* = 41,(not reported, undergraduate university students)	75.2%	40.7%

Note: Bedtime variability (BTv), risetime variability (RTv), time in bed (TIB), visual analogue scale (VAS). Measures (note, references can be found at the bottom of this document): Academic worry questionnaire (AWQ, Wolitzky & Telch, 2005); Cognitive irritation (CI, Mohr et al., 2007); Eating Questionnaire (EDE-1; Fairburn & Beglin, 2008) Fagerström Test of Nicotine Dependence-Revised (FTND-R; Heatherton et al., 1991); Glasgow Sleep Inventory (GSI, Broomfield & Espie, 2014); Illness worrying scale (IWS, Fink et al., 1999); Maslach Burnout Inventory (MBI, Maslach, 1986);Patient Health Questionnaire (PHQ-8, Razykov et al., 2012); Penn State Worry Questionnaire (PSWQ, Meyer, Miller, Metzger & Borkovec, 1990); Pre-Sleep Arousal Scale (PSAS, Nicassio et al., 1985); Recooperation in sleep (SF-AR, Görtelmeyer et al., 2011); Roland and Morris Disability Questionnaire (RDQ; Roland & Fairbank, 2000);Ruminative Response Scale (RRS, Topper, Emmelkamp, Watkins, & Ehring, 2014);Standardized Sleep Inventory (SSI, Görtelmeyer, 2011); Tampa Scale of Kinesiophobia (TSK, Swinkels-Meewisse et al., 2003); The Children's Response Style Questionnaire (CRSQ, Abela et al., 2007); Acceptance, mindfulness, and related processes in childhood (AMR, Greco, Dew & Ball, 2005); The Cognitive Irritation Scale (CIS, Mohr, Rigotti, & Müller, 2007); The Health Survey (SF-12, Gandek et al., 1998); The Insomnia Severity Index (ISI, Bastien et al., 2001); The Maslach Burnout Inventory for people working in human services (MBI-D, Büssing & Perrar, 1992); The Non-Productive Thoughts Questionnaire for Kids (CERQ-k, Garnefski et al., 2007); The Occupational Fatigue Exhaustion Recovery scale

(OFER, Winwood, Bakker, & Winefield, 2007); The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995); The Perseverative Thinking Questionnaire (PTQ, Ehring, Raes, Weidacker & Emmelkamp, 2012); The Pittsburgh Sleep Quality Index (PSQI, Buysse et al., 1989); The Sleep Quality Scale (SQS, Yi, Shin, & Shin, 2006); The Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999); The Somatic Complaints List (SCL, Jellesma et al., 2007); The Subjective Health Complaints Inventory (SHC, Eriksen et al., 1998); The Whiteley-7 (Conradt et al., 2006); The Work-Related Rumination Questionnaire (WRRQ, Cropley et al., 2012); The World Health Organization Quality of Life (WHOQOLBREF; World Health Organization, 2004); Upsala Sleep Inventory scale (USI, Liljenberg et al., 1988); Worry Domains Questionnaire (WDQ; Tallis, Eysenk, & Mathews, 1992). Reference list in Appendix 1.1.8.

On average, content was provided across 8 days (*SD* = 4.27), with intervention groups receiving content on more days (*M* = 9.2, SD = 3.81) than the comparison groups (*M* = 7.14, SD = 3.11). The mean time-point at which post-intervention measures were collected (from initial exposure to intervention content) was 49 days (*SD* = 52.49) for PC, 99 days for physical health outcomes (SD = 103.06) and 143 for health behaviours (SD = 130.38) (*M* = 118*, SD* = 115.59 for health outcomes *overall*). All of the interventions that were delivered in an in-person (*k* = 21, 58.3%) used printed materials, and employed a variety of delivery formats (i.e., self-administered, self-administered with support, healthcare professionals). Fifteen studies (41.66%) were hosted using an online platform (i.e., computer, mobile phone or tablet based). The most popular mode of delivery was interventions that were self-administered, with participants set a task to complete (e.g., to postpone worry) by experimenters in their own time (*k* = 16, 44.4%), followed by self-administration with support (i.e. from the experimenter) (k = 8, 22.2%). Less popular were interventions delivered with a trained facilitator (i.e. a mindfulness coach) (*k* = 6, 16.6%), or by a health-care professional (i.e. a nurse practitioner) (*k* = 4, 11.1%). Of these, three studies (8.3%) also used the telephone, two studies used mail (5.55%) and one study adopted a video to deliver part of the intervention (3.6%). The interventions tested were broadly defined as: (1) cognitive behavioural/acceptance and commitment therapies (*k* = 10, 27.7%), (2) PC action plans (*k* = 9, 25%), (3) mindfulness and relaxation (*k* = 7, 19.4%), (4) stress management (*k* = 4, 11.1%), (5) psychological detachment (*k* = 2, 5.5%), (6) expressive writing (*k* = 2, 5.5%), and (7) pain management (*k* = 2, 5.5%). While these categories do not capture the granular level nuances between interventions, they do represent the core therapy used.

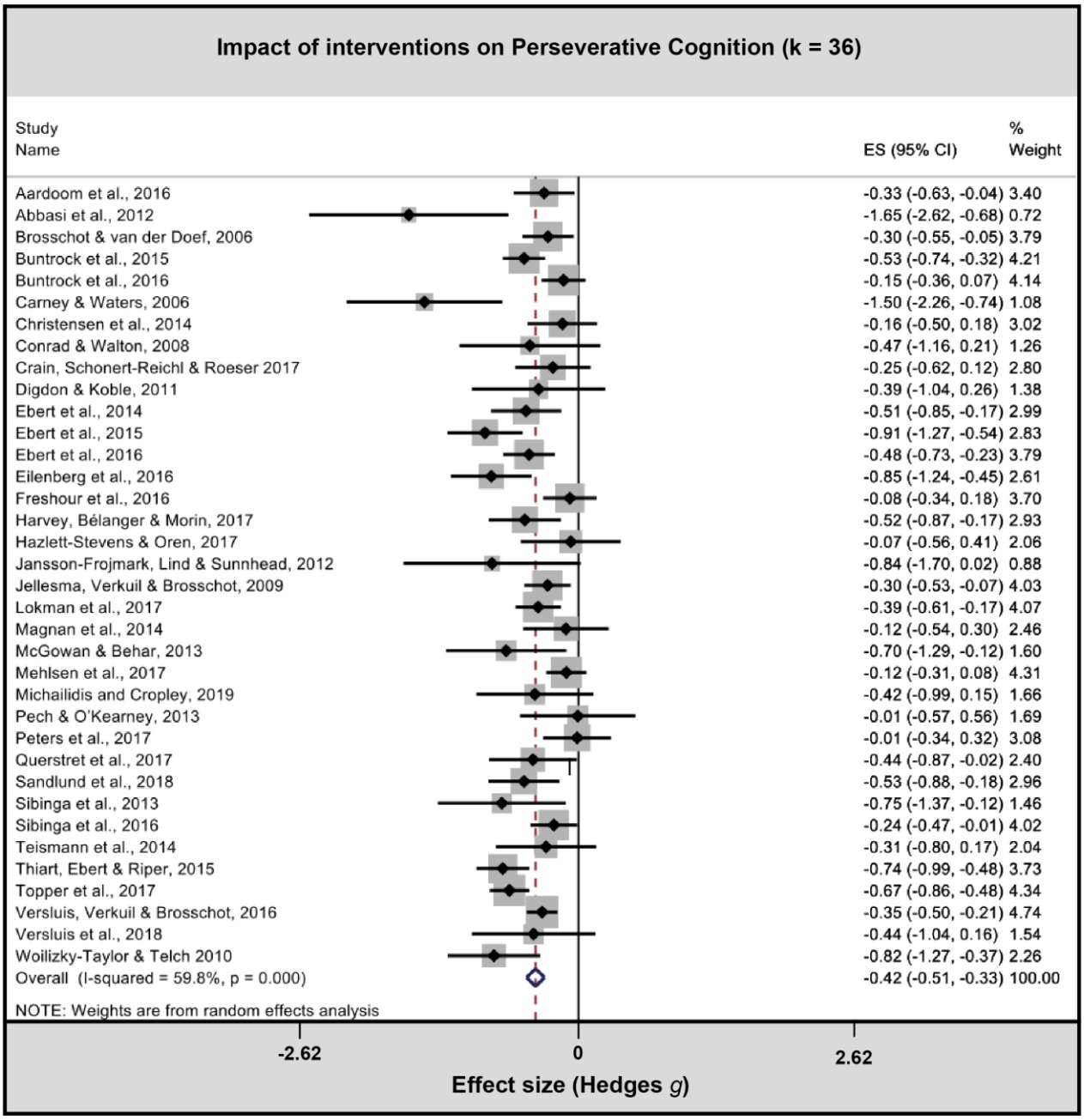
In general, studies were unclear or at high risk of bias. Although only 4 studies (11.1%) failed to report a valid method of randomization, 21 (58.3%) did not report a method of allocation concealment, 29 (80.6%) did not report adequate steps to blind the experimenter or data analyst and 34 (94.4%) did not report adequate methods to blind participants. Over 60% of studies (*k* = 20, 61.1%) did not claim contamination prevention between groups and did not

consider using ITT analysis, though only one study (3.6%) used measures of PC that were not internally reliable. The majority of studies contained information on informed consent (*k* = 32, 88.8%). Attrition rates were moderate (22.9%, *SD* = 16.63), and did not significantly influence PC effect sizes (*p* =.381). A summary of the risk of bias for each study is available via Appendix 2.3 and 2.4. Despite instances of high risk of bias across the included studies, each risk of bias item did not moderate the effects of the interventions on PC (*p* =.076 to .981; median = *p* = .432).

3.3.2 Objective 1a: Can PC (worry and rumination) be influenced by interventions?

Levels of PC were lower in the intervention group versus the comparison group at follow-up. The interventions produced, on average, a near medium-sized effect on PC, *g* = -0.42, 95% *CI* = -0.51 to -0.33 (*k* = 36, see Figure 3.2), albeit the effect sizes were heterogeneous across studies, I^2 = 59.3%; *Q*(35) = 87.17 *p* < .001. A similar-sized, and heterogeneous effect, I^2 = 47.9%; Q(18) = 34.56 *p* = .011, emerged when the analyses were repeated specifically for worry, *g* = -0.41, 95% *CI* = -0.51 to -0.30 (*k* = 19, see Appendix 2.1.2). Interventions produced a medium-sized effect on rumination, *g* = -0.58, 95% CI = -0.84 to -0.32 (*k* = 8, see Appendix 2.1.3), with the effect sizes again heterogeneous, I^2 = 66.9%; Q(7) = 21.14 *p* = .004.

Figure 3.2 Perseverative Cognition forest plot.



3.3.3 Objective 1b: Study characteristics associated with greater effect sizes for PC.

All but two of the seven intervention types (pain management and expressive writing) produced significant effect sizes for PC. However, meta-regressions indicated that none of the intervention types produced larger effects than the other interventions combined (see Appendix 2.1.4 and Appendix 2.1.5). Effect sizes were significantly larger, suggesting more

effectiveness, when interventions were delivered by healthcare professionals, *B* = 0.39, *S.E.* = 0.18, *CI* = -0.77 − -.009, *p* = .045, versus when they were not delivered by healthcare professionals. No other moderators influenced PC effect sizes across all PC related analyses.

Three intervention types, (PC action planning, psychological detachment and CBT) produced significant effect sizes for worry, though subsequent meta-regressions revealed none of these intervention types outperformed one another. Effect sizes were, however, significantly larger in studies comprising of a student sample, *B* = -0.35, *S.E.* = 0.14, *CI* = -0.65 − -0.05, *p* = .024, than in those which did not. Worry effect sizes were not influenced by any other moderators across all other worry related analyses.

Four intervention types (mindfulness, psychological detachment, CBT and pain management) produced significant post-intervention differences in rumination between the intervention and comparison conditions (see Appendix 2.1.4), though subsequent meta-regressions revealed none of these intervention types outperformed one another. These effects were not influenced by any moderators.

3.3.4 Objective 2a: Can interventions targeting PC also impact health outcomes?

The interventions targeting PC, on average, led to a small-to-medium, and heterogeneous I^2 = 51.8%; *Q*(20) = 41.50 *p* = .003, effect for health behaviours, *g* = 0.31, 95% *CI* 0.21 to 0.42 (*k* = 21, see Figure 3.3). A similar-sized, but non-significant and homogeneous I^2 = 24.7%; *Q*(20) = 26.57 *p* =.148, effect, *g* = 0.23, 95% *CI* = 0.15 to 0.31, was detected for physical health outcomes (*k* = 21, see Figure 3.4).

Figure 3.3. Forest plot for Health Behaviours.

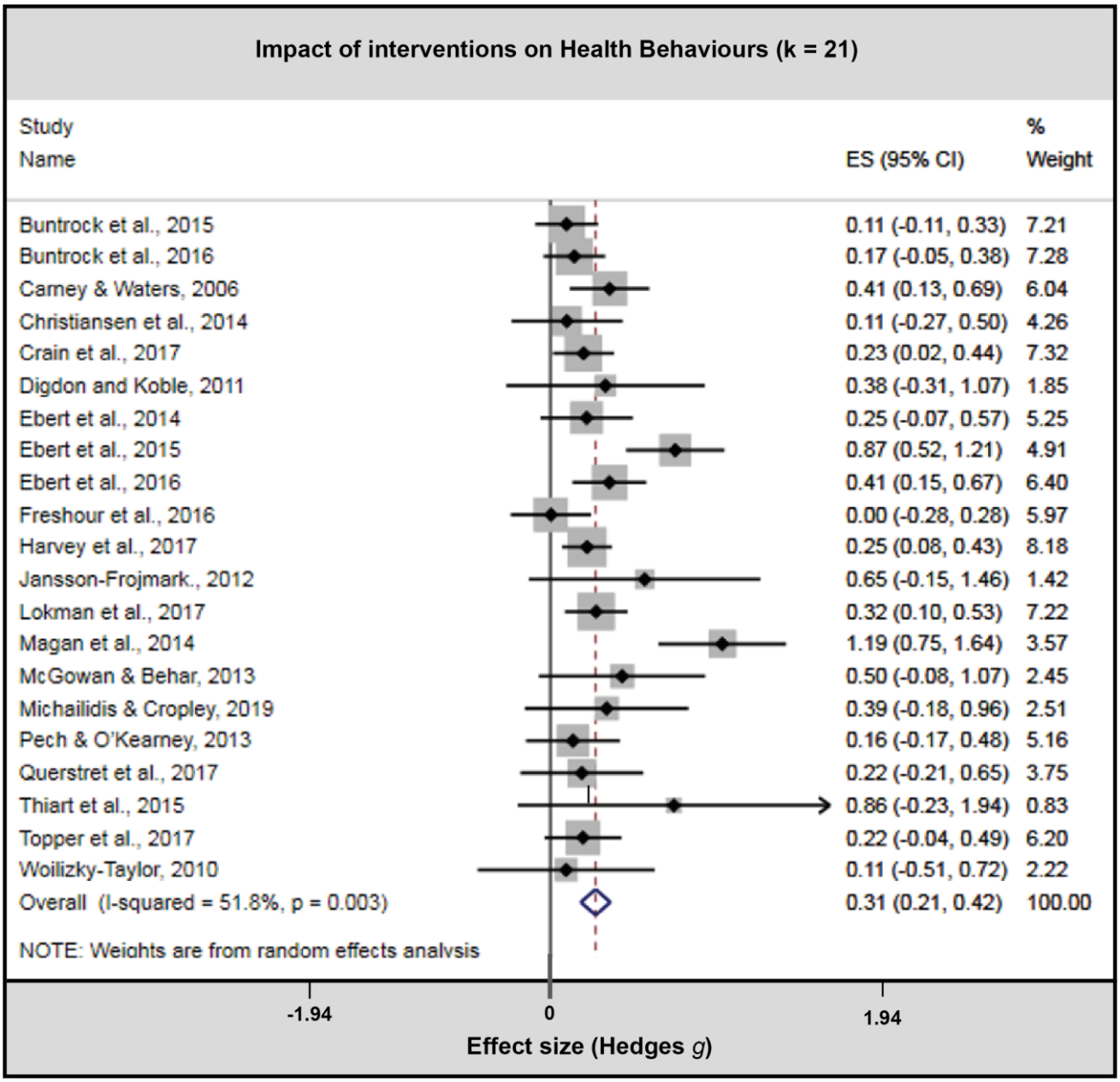
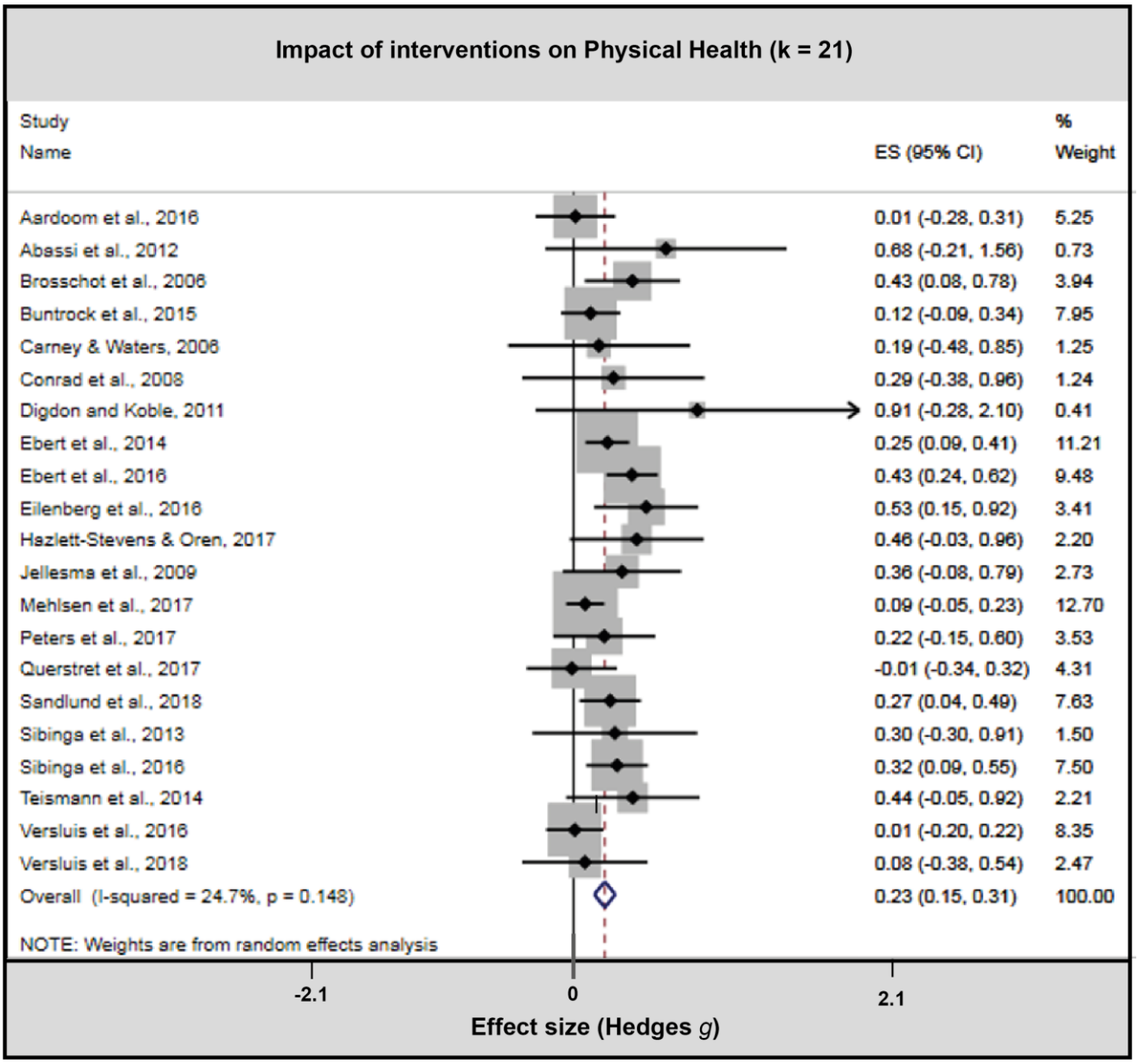


Figure 3.4. Forest plot for Physical Health.



3.3.5 Objective 2b: Study characteristics associated with larger effect sizes for health behaviours and physical health.

A range of study characteristics were significantly associated with effect sizes for both health behaviours and physical health outcomes. These are reported in full within Appendix 2.1 (see, 2.2.4 – 2.1.6) and Appendix 2.2; where we also consider the impact of confounding. In brief, all intervention types had a significant, positive effect on health behaviours with the

exception of pain management strategies. However, the effect sizes in studies testing psychological detachment style interventions, *B* = 0.33, *S.E.* = 0.16, *CI* = -.007 − 0.67, *p* = .05, and PC action plans, *B* = 0.37, *S.E.* = 0.14, *CI* = 0.08 − 0.66, *p* = .016, produced significantly larger effect sizes than studies not testing this intervention type for health behaviours. In addition, effect sizes were significantly larger when interventions were self-administered, *B* = 0.26, *S.E.* = 0.09, *CI* = 0.07 − 0.45, *p* = .01, delivered at an individual level rather than group-level, *B* = -0.25, *S.E.* = 0.11, *CI* = -0.49 − 0.006, *p* = .045, and when health behaviours were assessed closer to the conclusion of an intervention, *B* = -0.001, *S.E*. = .0003, *CI* = -.002 − -.0003, *p* = .01 (*k* = 21) (see Appendix 2.2, for further consideration).

While no particular intervention type was related to significantly larger effect sizes for physical health outcomes, interventions were at their most effective when delivered in educational, *B* = 0.19, *S.E.* = 0.07, *CI* = 0.48 − 0.32, *p* = .01, and academic settings, *B* = -0.17, *S.E.* = 0.08, *CI* = -0.35 − 0.06, *p* = .043, as opposed to delivered in medical settings, *B* = 0.009, *S.E.* = 0.10, *CI* = -0.19 − 0.21, *p* = .919.

Objective 3: Are larger effect sizes for PC associated with positive effect sizes for health outcomes?

Initially, effect sizes for PC were unrelated to effect sizes for health behaviours *B* = -0.21, *S.E.* = 0.15, *CI* = -0.54 − 0.12, *p* = .212 (*k* = 21). However, after the removal of a multivariate influential case (Magnan et al., 2014), medium-sized effects for PC, *g* = -.43, were associated with a small, but positive, *g* = .27, effect for health behaviours, *B* = -0.28, *S.E.* = 0.10, *CI* = -0.50 − -0.07, *p* = .012. Importantly, this effect was upheld in subsequent permutation tests with 10,000 random computations, *B* = -0.28, *S.E.* = 0.24, *CI* = -0.75 − 0.19, *p* = .019. Marginal associations between both worry and health behaviour, as well as between rumination and health behaviour were also revealed (see Appendix 2.2).

Effect sizes for PC were unrelated to effect sizes for physical health, *B* = -0.18, *S.E.* = 0.16, *CI* = -0.52 − 0.15, *p* = .264 (*k* = 21), even after the removal of an influential case (Digdon & Koble, 2011), *B* = -0.18, *S.E*. = 0.10, *CI* = -0.52 − 0.15*, p* = .261. There were no significant associations between specific effect sizes for either worry or rumination and physical health outcomes (see Table 3.2).

Table 3.2. Associations between PC effect sizes and health outcome effect sizes.

Predictor	Outcome	Studies	*k*	Statistic	
				B	*S.E*
PC	Health behaviours	Full	36	-.21	.15
		Exc.outliers	35	-.28*	.10
PC	Physical health	Full	36	-.18	.16
		Exc.outliers	35	-.18	.10
PC	Sleep	Full	17	-.29*	.10
		Exc.outliers	16	-.19*	.11
Worry	Health behaviours	Full	14	-.45†	.21
Worry	Physical health	Full	9	-.35	.61
		Exc.outliers	8	-.67	.53
Worry	Sleep	Full	10	-.76	.28
		Exc.outliers	9	-.94**	.23
Rumination	Health behaviours	Full	5	-.71†	.27
Rumination	Physical health	Full	4	-27	.36
Rumination	Sleep	Full	5	-62	.34

Note: **p* < .05; ***p* < .01; ***; † = *p*>.05 -.08; PC = perseverative cognition; Exc. = exclude.

3.4 Discussion

The findings of this systematic review and meta-analysis revealed that interventions produce medium-sized effect sizes for worry and rumination and that these correspond to small, but positive, effect sizes for health behaviours (and small-medium positive effect sizes for sleep, see Appendix 2.2). Interventions did not, however, produce significant differences for physical health outcomes. Interventions produced significantly larger effect sizes for PC when interventions were delivered by healthcare professionals compared to all other alternative methods, and despite no intervention type producing larger effect sizes for PC (when directly compared against other types), there was evidence that studies incorporating

psychological detachment style and PC action planning interventions generated significantly larger effect sizes for health behaviours.

This review provides the first meta-analytic evidence that a range of psychological interventions can be used to influence PC. Consistent with a previous narrative review (see, Querstret & Cropley, 2013), a broad variety of interventions encouraging participants to challenge their thinking style, or to disengage from the emotional response brought on by worry or rumination, can significantly decrease PC. Larger effect sizes were observed for rumination (g = .58, k = 8) than for worry, but worry was represented by far more studies and therefore subject to a wider variety of intervention types (g = .41, k = 19) and, promisingly, the majority of studies used the same well-validated measures (i.e., PSWQ; RRS) for these constructs. Further, the Querstret and Cropley review promoted the utility of CBT and mindfulness approaches, which was in line with our moderation analyses highlighting both approaches as useful strategies to mitigate against PC. Interestingly, however, in the current meta-analysis, no particular intervention type produced significantly larger PC effect sizes, but this is likely attributable to considerable heterogeneity belonging to the specific intervention content adopted by the studies. Therefore, despite the need for future research to understand the mechanisms of action in more detail, these findings show that these brief, inexpensive, and often self-administered interventions represent a useful safeguard against the harmful consequences brought on by worry and/or rumination.

The theoretical significance of the current findings are twofold as: a) they represent the first synthesis of experimental studies testing Brosschot et al.'s (2006) original PC hypothesis; and b) they document fresh evidence for the extension of the PC hypothesis to one that includes health behaviours, given that effect sizes for PC (following intervention) are positively associated with health behaviours, but not physical health outcomes. The original PC hypothesis proposed that worry, rumination and related thought processes mediate the relationship between stress and disease as, when stressors are perseverated upon in

thought, the damaging physiological activation associated with stress is also protracted, thus increasing susceptibility to stress-related ill-health (see, Brosschot et al., 2006, O'Connor et al., 2013). Therefore, the absence of effects for physical health outcomes in this review does not support the original PC hypothesis, though a number of contextual factors relating to this meta-analysis may account for these findings. First, the intervention content was delivered over a relatively short period (*M* = ~ 8 days) and very few of the studies reviewed here set out to improve physical health, with almost all studies listing their physical health outcome as a secondary measure (i.e., with the exception of the pain management studies). Second, as many interventions targeted determinants of behaviour, it would follow that they are more likely to produce larger effect sizes for health behaviours than in physical health outcomes; highlighting that the null effect observed for physical health may not be a reflection of PC failing to mediate the association between stress and physical disease, but rather that the intervention content was misaligned to significantly impact physical health. Third, it is notable that there was significantly more heterogeneity among physical health outcomes than for health behaviours, indicating that the observed intervention effects for physical health contained greater differences and more 'noise' among the data and, fourth, health behaviours were largely represented by a number sleep studies which yielded significant effects. It must therefore be noted that while the currently available evidence does not support the original PC hypothesis, such a conclusion may change; given the relationship between PC and physical health is theoretically viable, the effects were in the predicted direction, and potentially confounded by the aforementioned factors. Combined with the fact that previous published work drawing comparisons between PC and physical health is sparse, we are not ruling out that the effects for physical health outcomes may have been different with a greater number of studies and with interventions which more carefully targeted this particular facet of health. This does, however, highlight the need for future research to design carefully controlled studies with robust intervention arms to explicitly investigate further the relationship between PC and subsequent improvements (or otherwise) in physical health outcomes.

However, the current findings do support the recent extension of the PC hypothesis to include health behaviours as an additional pathway to disease (see, Clancy et al., 2016; 2020). These findings are an important milestone for the extended PC hypothesis, and for the stress literature more generally as they show, for the first time across a range of studies, that effect sizes for PC following randomised experimental manipulations (taken, on average, 41 days after intervention exposure) are positively associated with health behaviours (taken, on average, at 143 days post-intervention). Further to what has been previously revealed in correlational tests by Clancy and colleagues – who first showed that the effects for health behaviours were most strongly associated with rumination (Clancy et al., 2016), before a second meta-analysis demonstrated that both types of PC were robustly associated with poorer sleep (Clancy et al., 2020) – here, using experimental evidence, we show that a more negative health behaviour profile (and sleep in particular) are related to larger effect sizes for the maladaptive characteristics of *both* worry and rumination. This is not only theoretically important, as this finding supports the view that worry and rumination, though separate and related constructs, are likely underpinned by related cognitive processes (as the same intervention content yielded the similar treatment effects), but also affords further clarity to healthcare professionals and other interventionists to help make more informed treatment choices in the knowledge that both constructs are sensitive to similar interventions. Therefore, given the prominence of PC in the aetiology of illness and disease, the interventions included in this review can be used to attenuate the impact of both worry and rumination on health behaviours.

Promisingly, the findings for PC were not exclusive to a particular population (age or gender), setting or participant format (group vs. individual), and did not vary across duration of delivery or the number of sessions (single session vs. multi-session); suggesting that similar results could be achieved through brief and long interventions as well as single and multi-session interventions. Effect sizes also did not vary for PC across time possibly

indicating the interventions might have a longer term impact on PC. However, despite our best efforts to identify and control for confounding, it is not possible to remove all sources and it must be remembered that the number of studies reviewed here was relatively small especially when accounting for potential confounds in multivariate analyses. Equally, although all but pain management and expressive writing intervention types yielded significant effect sizes for PC, no intervention type was found to outperform another by producing significantly larger effect sizes. However, significantly greater differences between intervention and comparator groups for health behaviours and sleep, were attributable to psychological detachment style interventions (see, Appendix 2.2) and, for health behaviours in particular, PC action planning interventions were more effective than interventions not utilising this approach. Interpreting and understanding the impact of these interventions is a challenging task that is influenced by a range of moderators and factors that are difficult to explain. It is interesting, however, that the two most successful interventions yielding larger health behaviour effect sizes (psychological detachment & PC action planning) do share one common feature in that both place emphasis on the appraisal of metacognitions that urge the participant to discover internal goals and use environmental cues to either ‘switch-off’ or ‘offset’ their intrusive thoughts (e.g., Brosschot & van der Doef, 2006; Ebert et al., 2015).

A number of potential moderators were identified which may be helpful in identifying means to maximise intervention effects. For example, larger effect sizes for PC were found when interventions were delivered by healthcare professionals (for all results, see Appendix 2). Overall, however, these findings are consistent with recent observations suggesting a range of study characteristics, beyond behaviour change techniques, can influence the magnitude of change in health contexts (Prestwich, Kenworthy & Conner, 2017) and thus should be carefully considered within prospective interventions targeting similar or related mechanisms of influence.

Surprisingly, few studies in this meta-analysis explicitly targeted rumination, which is notable given its long-standing role in the aetiology of adverse mental health conditions (see, Kraft, 2019; Mezulis, Priess & Hyde, 2011; O'Connor, O'Connor & Marshall, 2007; Nolen-Hoeksema, 2000; Pugach, Campbell & Wisco, 2020; Thomsen et al., 2004). As a result, power issues were present in some of the rumination related analyses and should be therefore interpreted with caution (Cochrane, 2020). Indeed, an insufficient number of studies did not allow for a thorough exploration of the specific facets of rumination (e.g., positive vs. negative rumination, brooding vs. self-reflection, relationships with catastrophic thinking) that may be more likely to mediate the relationship between stress and ill health. Therefore, while the studies in this review are important and highlight the impact of rumination on subsequent health-related outcomes and behaviours, we strongly advocate future work exploring rumination.

We recognise that there are a number of limitations of the current meta-analysis. First, as with any meta-analysis, the effect sizes reported only represent estimates of the true effects. Second, the majority of measures for both PC and health outcomes were based on self-report methods. Although some work does exist documenting the impact of PC on objective measures of health (e.g., Teisman et al., 2014 & Versluis et al., 2018), this review highlights the pressing requirement for future interventions to incorporate more objective measures of health within their designs. Third, formal tests of mediation are required to further examine whether PC mediates the effects of interventions upon health behaviours. Fourth, studies were generally at unclear or high risk of bias (See, Appendix 2.3 and 2.4). Although synthesising evidence across studies noted to have different sources of bias can be problematic, the risk of bias factors did not significantly moderate the effectiveness of any of the interventions on PC or health variables. Equally, it was reassuring that small study or publication bias had no impact on any study effect sizes (see Appendix 2.1.7). Fifth, although they did not meaningfully influence the main objectives there was some evidence for confounding across the assessed moderators (see Appendix 2.1.4 – 2.1.6) and, sixth,

this meta-analysis did not address all sources of heterogeneity contributing towards effect sizes despite testing a range of moderators (see Appendix 2.1.8). Future research is thus required to understand the mechanisms of action relating to the types of intervention content most likely to produce larger PC effects.

In conclusion, this systematic review and meta-analysis reveals interventions can produce medium-sized effect sizes for worry and rumination and that these correspond to small, but positive, effect sizes for health behaviours (and small-medium effect sizes for sleep) but not physical health. This casts new light on the original PC hypothesis and offers fresh support for its extension, placing greater emphasis on the role of health behaviours as an important mediating factor in the relationship between stress and disease.

Chapter 4

Effects of worry postponement on daily worry and sleep: A randomised controlled trial.

4.1 Introduction

Recent data from the United Kingdom suggest 74 per cent of adolescents have reported feeling stressed, worried, or anxious at some point in the past year, to the extent that they have felt overwhelmed or unable to cope (Mental Health Foundation, 2022). Worry, defined as a 'chain of thoughts and images that are negatively affect-laden and relatively uncontrollable' (Borkovec, Robinson, Pruzinsky, & DePree, 1983, p. 10), has long been studied as a cardinal aetiological element in several psychological/psychiatric conditions and as an aspect of everyday life (e.g., Hawkes, Houghton & Rowe, 2009). For example, worry is a key feature in generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), and depressive disorders (Borkovec, Robinson, et al., 1983; Chelminski & Zimmerman, 2003). Importantly, there is also empirical evidence showing that worry is a significant determinant of heightened physiological activity and neuroendocrine dysregulation (Brosschot, Gerin, & Thayer, 2006; O'Connor, Thayer & Vedhara, 2021; for review see, Ottaviani, 2018) and, more recently, studies have associated worry to the enactment of poorer health behaviors, including adopting a less balanced diet, poorer sleep, and increased alcohol consumption (Clancy, Prestwich, Caperon & O'Connor, 2016; Clancy, Prestwich & O'Connor, 2022; Cropley et al., 2012; Frone, 2015). It is well established that these negative health behaviors are related to illness (Suris & Parera, 2005), disease, and morbidity rates (Burke et al., 2007) in both adults and children globally (for review, see Mackenbach, 2014).

A conceptual model now exists – the Perseverative Cognition Hypothesis - which portrays how worry can have a detrimental impact on health. Central to this hypothesis is the notion that perseverative cognition (PC), the cognitive representation of past stressful events

(rumination) or feared future events (worry), is a key mediator through which psychosocial stress leads to negative health outcomes. PC is thought to prolong the physiological activation beyond the presence of a direct stressor, and this prolongation of the stress response can lead to health problems (Brosschot et al., 2006). This is a concern, given that prolonged physiological activity carries severe health risks; for example, prolonged increases in heart rate has been shown to be predictive of coronary heart disease and cardiovascular death (Palatini & Julius, 1997; Kubzanskyet al., 1997; Fisher & Newman, 2013). Further, Ottaviani et al. (2018) reviewed neuroimaging data suggesting that heart rate variability (HRV) reduction from pre- to post-induction of PC is associated with both structural and functional brain abnormalities, showing that PC leads to impaired prefrontal inhibitory control over subcortical structures (e.g., diminished prefrontal amygdala functional connectivity). As such, there is a growing need for interventions that aim to prevent the onset and experience of PC due to its function as a transdiagnostic risk factor for both psychological and somatic health.

A recent meta-analysis of 36 randomized controlled trials that aimed to reduce PC, while subsequently measuring health outcomes, revealed that psychological interventions (relative to control groups), on average, produced medium-sized effects on rumination (g = -0.58), small-to-medium sized effects on worry (g = -0.41) and health behaviors (g = .31), and small-sized effects on physical health outcomes (g = 0.23) (McCarrick et al., 2021). Crucially, these findings suggest that PC can be influenced by relatively brief psychological interventions and that these treatment methods also hold beneficial downstream health effects. Of the interventions reviewed the most common was ‘PC action plans’, an approach focusing on proactively planning to deal with PC before encountering it; importantly, ‘PC action plans’ were responsible for significant reductions in the frequency of worry (g = -0.40). While there was some diversity in the practical implementation of this technique, the most prominent approach, coined ‘worry postponement’ (see, Brosschot et al., 2006), aims to confine the negative consequences of stress – attenuated through worry – within a restricted

time period reserved for such negative thoughts, rather than them being freely dispersed throughout the day (see, Brosschot et al., 2005). In turn, it is argued that the negative physiological and neuroendocrine dysregulation, as well as the unhealthier behaviors that are associated to worry, will be equally confined to the same window of time (i.e., the 20-30 minutes worry window).

Despite the reported effectiveness of worry postponement interventions, there are, however, ostensible issues that must not be overlooked. First, from a perspective of temporal validity, worry postponement interventions have not been tested over a period of more than 7 days, which is significant given worry episodes in those clinically affected are known to be temporally sensitive (Mackintosh, Lean & Hardy, 2021). Second, there are methodological limitations related to measurement strategies adopted in previous studies; change scores (i.e., baseline > follow-up) do not account for daily within-person variation in worry (and health behaviors). Each of these issues are important for the prospective design and implementation of interventions containing 'postponement' features that are built on this evidence base. Therefore, as worry postponement techniques have shown some promise in previous studies, further exploration of this method, combined with novel ways to boost adherence over an extended timeframe (e.g., techniques that include methods such as planning) with a more precise methodological lens (e.g., utilizing daily-diary designs), are both timely and warranted.

One solution, to boost intervention adherence, may be represented by implementation intentions (Gollwitzer, 1993). Implementation intentions involve individuals identifying appropriate cues for action (e.g., time of day, particular TV program) and response (intended action) and linking them in the form of an if-then plan (If I encounter X then I will do Y). A robust body of evidence shows that implementation intentions are an effective strategy for bridging the well-established gap between intentions and behavior (i.e., the intention-behavior gap; see Godin, Conner, & Sheeran, 2005 for a review). This strategy could be paired with worry postponement to increase the likelihood that individuals engage in worry

postponement. By enhancing the likelihood of engaging in worry postponement, there is greater likelihood that worry postponement would reduce worry and, in turn, outcomes associated with worry.

To our knowledge, implementation intentions and methods of worry postponement are yet to be combined within a randomized controlled trial. Therefore, given worry is a central facet of the PC Hypothesis, and considering the health-risks it poses, there is a need for innovative interventions that aim to reduce worry that may also improve health behaviors. Sleep, in particular, is an important health behavior due to its role regulating physiological repair and recuperation processes (Buysee, 2014; Carskadon & Dement, 2005). Indeed, people who sleep less are more likely to report a poorer quality of life (Groeger, Zijlstra & Dijk, 2004), are at higher risk of cardiovascular disorders (Brostrom et al., 2004) and ongoing sleep deficiency has been associated to kidney disease, high blood pressure, diabetes, and stroke (Stenholm et al., 2010; 2011). The properties of worry make it a key mediator between stress and sleep quality (Brosschot, Van Dijk & Thayer, 2007), principally this is because of worry's ability to prolong the psychological experience of stress (i.e., with 'what-if' self-assertions) and due to the frequently reported presence of worrying before bedtime (c.f., Pillai & Drake, 2015). Consequently, a direct attempt to attenuate worry may indirectly also positively impact upon sleep and sleep quality.

Therefore, in this study, we tested the relative effectiveness of two worry postponement interventions arms: an 'augmented' worry postponement intervention (i.e., including implementation intentions), and a 'standard' worry postponement arm, compared to two control arms (active & non-active control) at reducing (state-level) worry (duration & frequency) and improving sleep parameters over a 14-day period in an (online) randomized controlled trial. We tested the following hypotheses:

The intervention arms, combined, will outperform both of the control arms; such that participants in the augmented and standard worry postponement arms will report, on average, significantly lower levels of worry (Hypothesis 1A; H1A, relative to an active-

control), and significantly improved sleep outcomes (Hypothesis 1B; H1B, relative to control arms combined).

The augmented worry postponement arm will outperform the standard worry postponement arm; such that participants in the augmented arm will report, on average, significantly lower levels of worry (Hypothesis 2A; H2A), and significantly improved sleep outcomes (Hypothesis 2B; H2B).

4.2 Method

4.2.1 Design

A four-armed (online) randomized controlled trial (RCT), conducted between September 2021 and December 2021, was used to compare standard and 'augmented' worry postponement arms (i.e., a standard worry postponement arm and an augmented worry postponement arm, including implementation intentions) to two control arms (active and non-active control). We chose to include two control arms as, in typical worry postponement trials, control groups typically have an 'active' role, in that participants are either asked to complete worry diaries or itemized questionnaires (e.g., Brosschot et al., 2009). Given this task has been shown to trigger further worry (see, Carney & Waters, 2006), we also included a non-active control arm in view of having a 'pure' control arm, whose participants were solely required to complete daily measures of sleep. An interval-contingent design was utilized, where self-report measures of worry each night (duration & frequency for that day) and sleep (onset latency; disturbance, quality) each morning (for the previous night) were collected across the trial period. End-of-day/following morning diaries, rather than event contingent diaries were used to reduce participant burden and to help maintain adherence to the diary protocol (Broderick & Stone, 2006; Tennen et al., 2006). The study was preregistered on AsPredicted and can be found here.

The survey was hosted on Qualtrics (2022). In addition, for the sleep measures only (across all arms), text messages were sent to participants on each morning of the study prompting

their response. The timing of these texts was pre-determined based on the question "*what time do you usually wake up?"*; the texts were then scheduled within 30 minutes of this waking time. Text prompts were not used for the worry measures as this would violate the hypotheses testing the adherence aspect of the 'augmented' worry arm.

4.2.2 Participants and procedure

Participants were recruited via an online purposeful sample using email distribution, social media and adverts on university webpages. A power calculation (in G*Power version 3.1, Faul et al., 2009) revealed 252 participants were required (in total, post attrition, across groups; 63 in each arm) to detect a moderate change (effect size of f = 0.21 (based on the association between worry and sleep; see, McCarrick et al., 2021)) at post-treatment based on a power of .80 in a two-tailed test with alpha set at .05. This calculation was based on comparisons between all 4 arms of the intervention via a one-way omnibus analysis of variance[1].

The interventions were evaluated in adults (18 & older) who answered 'yes' to both of the following screening questions: (i) "Do you find yourself worrying a lot?; (ii) "Does your worry keep you awake at night?". These questions were used in view of recruiting individuals who concurrently experienced issues with worry and sleep (see, Digdon & Koble, 2011, for previous validation).

Participants who took sleep medication were not excluded from the study but were requested to keep their medication constant during the study period (e.g., Querstret & Cropley, 2013). Participants who presented with either chronic medical conditions (e.g., cardiovascular disease, musculoskeletal disorders, neurological disorders), or who have a

[1] A post-hoc sensitivity analysis, with alpha = .05, power 80% and the study sample of 186 participants, indicated the minimum effect size to which the study was sensitive was f = .24 (3, 182, critical f = 2.65) (see, Bloom, 1995) which only slightly exceeded the original anticipated effect size of f = .21. Moreover, (i) other daily-diary studies have reported effect sizes between worry and sleep that have exceeded the minimum effect size to which our study was sensitive (Weise et al., 2013); (ii) this study was initially powered on the relationship between worry and sleep, although had it been powered on the effect of the intervention on worry (g = .42; see, McCarrick et al., 2021) the minimum power requirements would have been achieved.

new-born baby, were excluded from the study as these have been shown to negatively impact sleep (Parish, 2009; Bulck, 2004). Participants were required to own a mobile phone at the time of the study to participate.

All eligible participants were first directed to Qualtrics (2022) where, following providing informed consent, they were enrolled onto the study. Qualtrics (2022) 'Randomizer' performed the randomization process, randomly allocating participants into one of the four arms (1:1:1:1) at the 'block' level, in order to reduce potential selection bias and to ensure methodologically adequate allocation concealment (Cochrane, 2022); as such, the researchers were blinded to allocation but the participants in the active arms were not. Specifically, while participants were not aware that other arms exist, the participants in the worry intervention arms were provided with procedural instructions about how to manage worry and the active-control was provided instructions on how to log their sleep. To mask this, participants were told they were taking part in a study investigating the general sleep quality of adults for the purposes of designing a prospective intervention to improve sleep. Participants were told that they would be asked to provide responses every day for the next 14 days (on measures specific to their study arm). At this stage, participants in the active arms were asked to make note of a specific hyperlink that would take them to their worry diary and save it in the notes section of their phone. When completing the diary, participants were asked to provide a consistent and unique codeword for which they could be identified. Upon completion of the study, participants were compensated with a £5 gift voucher, were informed of the aims of the study and provided with documents detailing how they can utilize the worry postponement techniques. Leaking of study information was highly unlikely given this study was hosted online and participants never met one another or knew who else was participating in the trial. The precise contents of each arm and the procedural differences are described below. The study was approved by institutional ethical procedures (PSYC-310).

4.2.3 Intervention arms

4.2.3.1 Worry Postponement (WP)

This arm of the intervention aimed to replicate the classic worry postponement intervention put forward by Brosschot et al. (2006). Participants were thus provided the same instructions afforded to those in the original Brosschot study of postponing their worrying every time they realize they are doing so to a special 20-30min period in the early evening (4-5 hours before sleep). As such, participants were instructed: *'every time you realize that you are worrying, terminate them right away, and 'postpone' them to this special period".* Participants received no further instruction for the timing or content of this special period.

4.2.3.2 Augmented Worry Postponement (AWP)

The augmented arm adopted the same central feature of worry postponement as described above, but as specific goal-orientated plans have been consistently shown to promote greater automated goal-directed responses (for review, see Gollwitzer & Sheeran, 2006), implementation intentions (i.e., an 'if-then' plan) were utilized within this group as a supplement to the standard worry postponement approach. Accordingly, the first aspect of instructions to participants was identical to the worry postponement arm. They were instructed: ": *'every time you realize that you are worrying, terminate them right away, and 'postpone' them to this special period".* However, unlike the worry postponement arm, participants were then told to form a specific plan framed to aid their adherence in executing this instruction.

Next, participants were asked to highlight an activity they routinely engage around 4-5 hours before bed and to use the period to trigger the onset of their special period of worry reflection. This took the form of an if-then plan (i.e., IF X happens, THEN I will do Y). As an initial aid, participants were provided examples such as: '*watching 10pm news', 'putting the kids in bed', 'eating supper'* or '*reading my favourite book'* .They were also asked if this period/activity would be appropriate for every night of the week and, in instances where participants report otherwise, an alternative plan was requested. Once agreed, participants were instructed to complete the following checklist to ensure their plans meet the criteria for an implementation intention: (a) does your plan(s) contain the words IF, THEN and I?; (b)

does your plan(s) identify ample situations for you to do 30 minutes of worry reflection every night of the week?; (c) does your plan(s) identify how you will undertake worry postponement in the situations identified in your plans? Participant must have answered yes to all responses to proceed with the study.

4.2.3.3 Active Control (A-C)

The active control arm aimed to replicate the control group from the original Brosschot et al. (2006) study to provide a neutral reference point for the direct comparison(s) between the relative efficacy of the augmented arm compared to, and combined with, the standard worry postponement arm found in the original Brosschot et al. (2006) study. In this group, participants were asked to register their worry (in their online diary) on each night of the study. They were instructed: *"On each night of this study, please provide an estimate on the total number and total frequency of your worries today via your worry diary".* These participants were not asked to postpone their worries and no implementation intentions were made.

4.2.3.4 Non-Active Control (NA-C)

Completing a nightly worry diary may confound the accuracy of worry recall, or worse, result in further worries (for example, see Szabó & Lovibond, 2006). Therefore, a fourth arm was incorporated in view of providing a 'pure' control. In this group, participants were only asked to provide an estimate of their sleep parameters on each morning of the study via text message. These participants were not asked to postpone their worries and no implementation intentions were made. As a way of masking the intervention, participants were instructed *"on each morning of this study, please provide as close to as possible your best estimate of your previous night's sleep quality"* and were asked to complete the battery of sleep questions outlined below.

4.2.4 Measures

4.2.4.1 Worry

An online worry diary was used in the three active arms (i.e., augmented worry postponement, standard worry postponement, active-control) to record the total duration and frequency of worry episodes experienced (retrospectively) that day. The questions asked to participants were as follows: "*How many times did you find yourself worrying today?*" and "*How long did these periods of worry, on average, last for?*". Responses were requested in minutes. The worry diary used in this study was replicated from the original Brosschot et al. (2006) study and has since been validated in more recent studies both in terms of its content validity and sensitivity to change (e.g., Fisher, Keogh & Eccleston, 2017; Clancy, O'Connor & Prestwich, 2020; Narmandakh et al., 2021).

4.2.4.2 Sleep

The following items were extracted from the Consensus Sleep Diary (Carney et al., 2012), Time in Bed: "*At what time did you get into bed last night?*; Sleep Onset Latency (SOL): *How long do you think it took you to fall asleep?*; Sleep Disturbance (SD): "*How many times did you wake up during the night?;* Sleep Quality (SQ): "*On a scale of 1-5, how would you rate the overall quality of last night's sleep?"* (1 = very poor to 5 = very good). This instrument has been used widely, and has been extensively validated, in a variety of sleep trials (e.g., Jungquist et al., 2015; Dietch & Taylor, 2021) and show good convergent validity (*r* = .615; Maich, Lachowski & Carney, 2018).

4.2.5 Statistical Analysis

The primary outcome was variability in worry duration and frequency across the 14-day study period; parameters of sleep (SOL; SQ; SD) were assessed as a secondary outcome. Responses were analysed in Stata 18 and SPSS 28. These data were analysed using Generalized Estimating Equations (GEE) models to account for within subject component (times) and the between component (study arm). For frequency data (number of times reporting worry or waking) negative binomial model were estimated and for continuous data

(sleep onset latency and worry variables) OLS models. Outliers were dealt with in several ways according to the variable properties. For sleep onset latency, the data ranged from 4 minutes to just under 9 hours for one participant, then 7 hours for another and then 6 hours, with a mean of 39 minutes. This distribution was positively skewed (*Z* for skew = 4.51/0.060) with a long tail. In line with the pre-registration, as the extremely high values were very likely implausible, symmetrical 90th percentile winsorization was applied (see Wilcox, 2005). Sleep quality was marginally negatively skewed (*Z* for skew = -0.23/0.052). For the frequency of daily worry, the range of data was from 0 to 100. For daily worry duration, the data ranged from 0 to 240 with a modal value of 20 minutes analysed using negative binomial regression. These are all plausible values. The minimum number of days completed was 1, the maximum was 14 and the median was six. Eighty-six participants completed all 14 days. We included in the analyses all days when participants provided diary data of 3 days or more, as simulation studies show that provides reliable estimates (Griffiths et al., 2018; Griffiths, Williams & Brohan, 2022)[2]. In addition, for the analyses relating to each of the hypotheses, age and gender were entered into the models as covariates.

4.3 Results

4.3.1 Descriptive Statistics

A total of 265 participants enrolled were onto the study, completed baseline measurements, were (block) randomized to condition, and provided their informed consent; fulfilling the power requirements set out by the a-priori calculation. However, 79 participants were excluded from the study by not providing a minimum of 3 days data (for worry variables) across the 14-day worry period (AWP: *n* = 31,45.58%; WP: *n* = 28, 41.79%; A-C: *n* = 11, 20.75%; NA-C: *n* = 9, 12%). Consequently, the final sample comprised 186 participants (WP: *n* = 39; AWP: *n* = 37; A-C: *n* = 42; NA-C: *n* = 66) who were aged, on average, 30.34

[2] The analyses were replicated on a non-winsorized dataset (i.e., with participants who provided less than 3 days data) and the results did not significantly change.

years (SD= 7.89) (63% female; 71.24% from United Kingdom & Ireland; 81.52% of white ethnicity; mean BMI = 23.95 (SD = 9.89)). Table 4.1 displays the baseline participant characteristics across the four study arms (there is no systematic difference across arms on any of these variables), Table 4.2 shows the means and SDs for the study variables across the study period.

Table 4.1. Means (SD) of Baseline Characteristics Across Study Arms

	AWP (*n* = 37)	**WP (*n* = 39)**	**A-C (*n* = 42)**	**NA-C (*n* = 66)**
Age (SD)	30.43 (7.77)	29.97 (7.72)	30.95 (8.26)	30.03 (7.81)
% Female	65%	62%	62%	63%
BMI (SD)	23.16 (9.49)	24.27 (9.10)	24.22 (10.01)	24.18 (10.98)
% from UK and Ireland	70%	71.%	73%	70%
% White Ethnicity	82%	81%	81%	82%
Average units of alcohol consumed per week	19.21	18.39	19.06	18.89
% of current smokers	21%	19%	20%	17%

Note: WP = worry postponement; AWP = augmented worry postponement; A-C = active control; NA-C = non-active control.

Table 4.2 Means (SD) of (level 1) daily study variables across the study period (*n* = 186).

	Total Sample	**AWP**	**WP**	**A-C**	**NA-C**
Worry Duration	40.40 (34.42)	29.82 (27.04)	47.53 (38.32)	43.13 (33.35)	N/A
Worry Frequency	18.60 (13.35)	16.40 (12.68)	23.83 (15.09)	13.40 (7.10)	N/A
Sleep Onset Latency	35.34 (31.03)	35.03 (34.19)	32.10 (26.89)	34.98 (29.18)	39.23 (32.88)

Sleep Disturbance	6.73 (2.01)	6.61 (2.12)	6.83 (2.08)	6.82 (1.91)	6.70 (1.96)
Sleep Quality	3.27 (1.07)	3.27 (1.11)	3.39 (1.07)	3.18 (1.01)	3.27 (1.04)

Note: WP = worry postponement; AWP = augmented worry postponement; WP = worry postponement; A-C = active control; NA-C = non-active control; N/A as not measured in this arm. These values reflect the winzorized data.

4.3.2 Do the worry interventions (combined) influence worry or sleep outcomes?

4.3.2.1 *Worry (Hypothesis 1A)*

The augmented worry postponement and standard worry postponement arms (combined) did not significantly differ, relative to the active control, on worry duration. However, worry frequency significantly increased, relative to the active-control, for the combined worry arms (Table 3, Panel A & C) Inspection of the means in Table 4.1 suggest that the standard worry postponement arm drove this effect on worry frequency. Neither age nor gender were significantly associated with worry duration or frequency (Table 4.3, Panel A & C).

4.3.2.2 *Sleep (Hypothesis 1B)*

The augmented worry postponement and standard worry postponement arms (combined) did not lead to any significant improvements in the sleep variables, relative to the control groups (combined), in terms of sleep quality, sleep onset latency, or sleep disturbance frequency (Table 4, Panels A, C & E). Neither age or gender significantly influenced the findings for sleep quality or sleep disturbance frequency. There was no significant effect of age on sleep onset latency, however a significant effect for gender was present, with females reporting significantly poorer (i.e., delayed) sleep onset latency (Table 4.4, Panel A, C & E).

4.3.3 Do either of the worry interventions (augmented & standard) have a unique influence worry or sleep outcomes?

4.3.3.1 Worry (Hypothesis 2A)

The augmented worry postponement arm yielded significantly lower worry duration across the study period compared to the active-control arm (Table 4.3, Panel B). The regression coefficient reflects an average reduction in worry duration of 14 minutes and 34 seconds (reported by participants in the augmented worry arm), relative to the active-control arm. There were no significant effects of age or gender on worry duration (Table 4.3, Panel B). We further tested if this positive effect of the augmented worry postponement arm was significantly greater than the standard worry postponement arm. To do this, the coefficients were contrasted for the augmented worry postponement and standard worry postponement arms in Panel B of Table 3. This was significant ($\chi^{2,}$ = 9.84, p = .002) indicating that the augmented worry postponement arm has a significantly greater effect reducing worry duration than the standard worry postponement arm

For worry frequency, the standard worry postponement arm significantly increased the frequency of worries across the study period, relative to the active-control arm (by ~ half a worry episode) (Table 4.3, Panel D). Neither age or gender were significantly associated with worry frequency (see Table 4.3, Panel D). We further tested if this negative effect of the standard worry postponement arm was significantly greater than the augmented worry postponement arm. To do this, the coefficients were contrasted for for the augmented worry postponement and standard worry postponement arms in Panel D of Table 3. This was significant ($\chi^{2,}$ = 4.54, p = .033) indicating that the standard worry postponement arm has a significantly greater effect in terms of increasing the frequency worry compared to the augmented worry postponement arm.

4.3.3.2 Sleep (Hypothesis 2B)

The augmented worry postponement arm did not yield any significant improvements, relative to the control groups (combined), in terms of sleep quality, sleep onset latency, or sleep disturbance frequency. Neither age nor gender significantly impacted the findings for sleep

quality, sleep disturbance frequency. There was no significant impact of age on the findings for sleep onset latency, however a significant effect for gender was present, with females reporting significantly poorer (i.e., delayed) sleep onset latency (see Table 4.4, Panel B, D & F).

Table 4.3 GEE analysis for effects of interventions on worry variables.

			Worry	
	Duration			
	Panel A			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
Combined Arms	-3.390 (7.001)	.628	-17.110	10.330
Demography				
Gender	10.576 (6.430)	.100	-2.026	23.179
Age	0.255 (0.379)	.67	-0.487	0.997
Constant	33.357 (13.433)	.013	7.028	59.686
n Groups (*observations*), M-days	91 (849), 9.3			
	Panel B			
	B (se)	**p**	**Lower**	**Upper**
Intervention Arm				
WP	6.393 (7.323)	.383	-7.959	20.747
AWP	-14.833 (7.568)	.049	-29.717	-0.050
Demography				
Gender	10.397 (6.080)	.087	-1.519	22.312
Age	0.282 (0.359)	.431	-0.4208	0.985
Constant	32.504 (12.724)	.011	7.566	57. 442
n Groups (*observations*), M-days	91 (849), 9.3			
	Frequency			
	Panel C			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
Combined Arms	0.447 (0.206)	.030	0.043	0.852
Demography				
Gender	-0.009 (0.181)	.965	-0.361	0.342
Age	0.001 (0.011)	.907	-0.019	0.022
Constant	2.552	<.001	1.770	3.335
n Groups (*n*), R^2	89 (846), 9.5			
	Panel D			
	B (se)	**p**	**Lower**	**Upper**

Intervention Arm				
WP	0.631(0.223)	.005	0.194	1.068
AWP	0.196 (0.230)	.394	-0.255	0.647
Demography				
Gender	-0.008 (0.184)	.964	-.368	0.352
Age	0.003 (0.011)	.815	-.0186	0.024
Constant	2.514 (0.389)	<.001	1.751	3.276
n Groups (*observations*), M-days	89 (846), 9.5			

Note. The active-control is the reference arm; Gender (0 = Men, 1 = Women); WP = worry postponement; AWP = augmented worry postponement; A-C = active control; NA-C = non-active control.

Table 4.4 GEE analysis for the effects of interventions on sleep variables.

	Sleep			
	Sleep Quality			
	Panel A			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
Combined Arms	0.113 (0.092)	.220	-0.067	0.293
Demography				
Gender	-0.071 (0.094)	.452	-0.255	-0.255
Age	-0.001 (0.006)	.978	-0.012	0.011
Constant	3.242 (0.190)	<.001	2.869	3.615
n Groups (*observations*), M-days	184 (2089), 11.4			
	Panel B			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
WP	-0.061 (0.121)	.281	-0.109	0.377
AWP	0.0426 (0.126)	.735	0.377	0.289
Demography				
Gender	-0.073 (0.094)	.448	-0.255	0.113
Age	0.001 (0.006)	.990	-0.0113	0.011
Constant	3.259 (3.259)	<.001	2.880	3.638
n Groups (*observations*), M-days	184 (2089), 11.4			
	Sleep Onset Latency			
	Panel C			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
Combined Arms	-3.537 (3.179)	.266	-9.768	2.693
Demography				
Gender	7.517 (3.255)	.021	1.137	13.897
Age	0.151 (0.200)	.450	-0.241	0.544
Constant	29.086 (6.550)	<.001	16.248	41.925

n Groups (*observations*), M-days	184 (2044), 11.2			
	Panel D			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
WP	-7.006 (4.273)	.101	-15.381	1.369
AWP	-2.892 (4.332)	.504	-11.383	5.598
Demography				
Gender	7.269 (3.239)	.025	0.920	13.620
Age	0.159 (0.200)	.426	-0.233	0.551
Constant	30.470 (6.657)	<.001	17.421	43.518
n Groups (*observations*), M-days	184 (2046), 11.1			
	Sleep Disturbance			
	Panel E			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
Combined Arms	-0.007 (0.096)	.943	-0.194	0.180
Demography				
Gender	-0.010 (0.098)	.917	-0.202	0.181
Age	-0.001 (0.006)	.955	-0.012	0.012
Constant	1.934 (0.198)	<.001	1.547	2.321
n Groups (*observations*), M-days	184 (2089), 11.4			
	Panel F			
	B (SE)	**p**	**Lower**	**Upper**
Intervention Arm				
WP	0.024 (0.130)	.853	-0.230	0.277
AWP	-0.030 (0.131)	.833	-0.285	0.230
Demography				
Gender	-0.009 (0.098)	.924	-0.201	-0.201
Age	-0.001 (0.006)	.953	0.953	0.012
Constant	1.928 (0.202)	<.001	1.533	2.323
n Groups (*observations*), M-days	184 (2089), 11.4			

Note. The active and non-active control (combined) is the reference arm; Gender (0 = Men, 1 = Women). WP = worry postponement; AWP = augmented worry postponement. Confidence intervals are 95% upper and lower.

4.3.4 Discussion

The aim of the present study was to test the relative effectiveness of two worry postponement interventions; an 'augmented' worry postponement arm, featuring

implementation intentions, and a ‘standard’ worry postponement intervention, compared to an active-control arm at reducing worry (duration and frequency) and improving sleep parameters (relative to active and non-active control groups) over a 14-day period in an (online) randomized controlled trial. Hypothesis 1 (A & B) received no support, as participants in the intervention arms (combined) did not experience any significant reductions in worry (H1A) (relative to the active control) or any improvements in sleep (H1B) (relative to control groups combined). The findings for hypothesis 2A were more complex, whereas hypothesis 2B received no support. Specifically, participants in the augmented worry postponement arm experienced significantly lower worry duration (by ~15 minutes) across the study period, relative to the active-control arm (H2A). Furthermore, when compared to the active-control, the standard worry postponement arm significantly increased the frequency of worry (by ~ half a worry episode) (H2A). However, when testing the intervention arms against each another, the augmented worry postponement arm yielded both significantly shorter worry duration and significantly less worry frequency. Neither of the intervention arms, augmented or standard worry postponement, produced significant improvements in any of the sleep parameters (H2B). Accordingly, these preliminary set of findings shed new light on the value of implementation intentions at enhancing traditional approaches to the management of worry, while also highlighting the growing requirement for future avenues of research to test intervention methods that not only impact worry but also engender positive changes in health behaviors, such as sleep.

Psychological interventions aiming to reduce the adverse effects of worry on health behaviors have been sparsely researched until recent years. Of the varied and pervasive health consequences attributable to worry (for review, see Ottaviani, 2018), research concerning its negative effects on health behaviors has only come to light in recent years (see, Clancy et al., 2016) and interventions directly targeting worry to, in turn, improve sleep are rare. A meta-analysis by McCarrick et al. (2021) found PC can be influenced by psychological interventions and that, in line with the PC Hypothesis (Brosschot et al., 2005),

they may yield beneficial effects on health behaviors (e.g., sleep outcomes), with the most effective strategy being ‘PC action plans’. However, several issues were raised with the technique relating to adherence barriers, limited testing periods (i.e., studies restricted to 7 days), in addition to methodological limitations underpinned by study designs that do not account for daily within-person variation in worry and that overtly rely on change scores (i.e., baseline > follow-up assessments; see McCarrick et al., 2021). Therefore, the present finding, that shows beneficial treatment effects for the augmented worry postponement arm, extends previous knowledge in three key ways: (i) via complementing a standalone worry postponement intervention with implementation intentions in an attempt to boost adherence, (ii) expanding the testing period to 14 days to assess robustness, and (iii) adopting a multi-level daily-diary design to capture not only how worry varies within-participants, but also how these variations relate to sleep outcomes measured the previous evening.

Despite the efficacy of the augmented arm at reducing the duration of worry, it must also be discussed as to why the standalone worry postponement arm increased worry frequency. Interestingly, this finding is somewhat consistent to what other authors have reported in relation to worry frequency. For example, Brosschot and van der Doef (2006) found postponers reported lower worry duration than controls (152 minutes, relative to 222 minutes), however this effect did not significantly extend to worry frequency (23.7 worry episodes versus 30.0 worry episodes). Similarly, Versluis, Verkuil and Brosschot (2015) reported no difference between postponers and controls at reducing worry frequency in an online trial (null effects were also present for worry duration) (see also, Mobach, Schie & Naring, 2018, for similar conclusions). One possibility for these findings, consistent with the present study, is that the worry postponement intervention is successful at inspiring people to think about their worry differently but is less successful at stopping worries from accumulating, which is reflected in the significant increase in worry frequency (in the WP arm), here. It seems plausible that the instruction to postpone a worry may prevent

experiencing the one that is presently being experienced, however be less useful in preventing worry recurrence or the onset of new worries.

Another finding from the present study was the unanimous null effects of the interventions on sleep outcomes. Sleep onset latency, sleep disturbance and subjective sleep quality were unaffected by both the interventions combined (H1B), and when tested in isolation of one another (H2B). While the true cause for these findings is not clear, one possible explanation is that while the properties of worry make it a key mediator between stress and sleep outcomes (Brosschot, Van Dijk & Thayer, 2007), the change in worry was not large enough to impact sleep. Despite being contrary to the hypotheses, this is hardly surprising as dedicated sleep interventions show mixed efficacy (for meta-analysis, see Griggs et al., 2020) and effect sizes for mediation models including the indirect effects of worry on sleep are generally small (e.g., Pillia & Drake, 2015). Another explanation may be sought from the measurement approach used in this study. Sleep data was self-reported and obtained via text message each morning of the study, posing two complications. First, descriptive analysis of this data shows people took, on average, 28.21 minutes (SD = 12.14) to provide an estimation of their sleep parameters from the previous night, raising questions over the accuracy of recall for at least some of the participants. Second, while self-report methods of sleep estimation were used due to logistical implications in this study, they suffer from varying degrees of reporting error (see, Manconi et al., 2010) and have been associated to other demand characteristics such as over estimation of an idealized sleep schedule (see, Short et al., 2013) mainly because recall relates to timeframes confined to unconscious thought. It is thus a possibility that the nuances of individualized sleeping patterns were not accurately captured by the measures used in this study. Accordingly, future research should not only adopt more multifaceted intervention methods that target specific aspects of worry that may be sensitive to sleep outcomes (e.g., temporal relations and bi-directionality between worry & sleep), but that they do so using objective measures of sleep associated to superior ecological validity (e.g., such as Actigraphy; see, Martin & Hakim, 2011).

The present study was not without its limitations. First, as noted above, this study relied entirely on online self-reported measures for all the variables examined, raising questions over measurement accuracy. Second, there is some possibility the results here are under-powered, as despite recruiting the required participants to meet the a-priori power calculation (*n* = 252) the final sample (post-attrition, post study) consisted of 186 participants. Third, despite randomisation being completed in random blocks at a ratio of 1:1:1:1, attrition meant that there was unequal participants in each group, raising some concerns over internal validity (see, Hey & Killelman, 2014); although attrition patterns were consistent with expectations (wherein the more participants were asked to do, relatively across groups, the more drop-outs were present); fourth, the interventions in this study were self-administered and delivered online, meaning future studies are required to ascertain their replicability when delivered using offline methods and when facilitated by health-care professionals or in group-based settings. However, despite these shortcomings, this study is one of the few RCTs available to test the impact of a psychological intervention on both mental and behavioral health outcomes (i.e., worry and sleep). It is also the first to test a worry postponement intervention augmented with implementation intentions, across a period greater than 7-days, and via a daily-diary multi-level design. Also, some of the concerns regarding statistical underpowering can be alleviated. Firstly, a post-hoc sensitivity analysis, with alpha = .05, power 80% and the study sample of 186 participants, indicated the minimum effect size to which the study was sensitive was *f* = .24 (3, 182, critical *f* = 2.65; equivalent to d = .48) (see, Bloom, 1995) which only slightly exceeded the original anticipated effect size of f = .21. Secondly, other daily-diary studies (i.e., not the pre-post designs in McCarrick et al., 2021) have reported effect sizes between worry and sleep that have exceeded the minimum effect size to which our study was sensitive (Weise et al., 2013).Thirdly, despite the study being initially powered on the relationship between worry and sleep, had it been powered on the effect of the intervention on worry (i.e., the primary outcome; *g* = .42; see, McCarrick et al., 2021) the minimum power requirements would have been achieved.

In sum, the present study considered, for the first time, the relative effectiveness of two worry postponement interventions; an ‘augmented’ worry postponement arm, featuring implementation intentions, and a ‘standard’ worry postponement intervention, compared to two control arms (active & non-active control) at reducing worry and improving sleep parameters. Participants in the augmented worry postponement arm experienced significantly lower worry duration (by ~15 minutes) across the study period, relative to the active-control arm. Yet, when compared to the active control, the standard worry postponement arm significantly increased the frequency of worry (by half a worry episode). However, importantly, when the interventions were tested against each other it was revealed that participants in the augmented worry postponement arm experienced significantly shorter worry duration and lower worry frequency. Neither of the intervention arms, augmented or standard worry postponement, had any impact on sleep. Accordingly, these new set of findings shed new light on the value of implementation intentions at enhancing traditional approaches to worry postponement, while also opening new avenues of research to test intervention methods that not only impact worry but engender equally positive changes in health behaviors, such as sleep.

Chapter 5

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

5.1 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 associated 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 (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 (Larsen & Christenfeld, 2009; Appel et al., 2021; 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 et al., 2018; Verkuil, Brosschot, Gebhardt & Thayer, 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, Prestwich, Prudenzi & O'Connor, 2021). More evidence then soon followed, Clancy, Prestwich & O'Connor (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 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 howdoes 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, Milne, Webb & Gollwitzer, 2005). Crucially, some explanation of the PC-health behaviour association 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; Fishbein & Ajzen, 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, Aubrey, & Kellett, 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 associationss 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).

5.2 Method

5.2.1 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).

5.2.2 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- β) 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, 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.

5.2.3 Measures

5.2.3.1 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 (α = .77–.93) and coded so that high values indicated high levels on the variable of interest.

5.2.3.2 Perseverative Cognition

At T1, worry and rumination were assessed via brief versions (Topper et al., 2014) of the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the Ruminative Responses Scale (RRS; Nolen-Hoeksema, 1991). In this study, brief versions have acceptable to high internal consistency (brief PSWQ: α = .89; brief RRS: α = .79) and have been shown correlate highly with the full questionnaires (brief PSWQ: r = .91–.94; brief RRS: r = .88–.91; Topper et al., 2014). In addition, they show high sensitivity (brief PSWQ: r = .90–.92; brief RRS: r = 90–.93), and high specificity (brief PSWQ: r = .88–.90; brief RRS: r = .80–.87) at detecting excessive worry and rumination. 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.

5.2.3.3 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 (α = .77–.92). This measure has been validated (see, Godin-Shephard, 2011) and widely employed in previous prospective TPB/health behaviour change studies (e.g., Lesser & Neinhous, 2020; Marker, Steele & Noser, 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, have been validated in previous work (e.g., Espie et al., 2012), showed acceptable internal consistency in the present study (α = .91 - 96).

Sedentary Activity

At T2, the Self-Report Sedentary Behaviour Questionnaire (Gardiner, 2011) 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. Total sedentary time has acceptable test-retest reliability and validity and is as responsive to change as accelerometer-derived sedentary time. In this study, total sedentary time showed acceptable internal consistency (α = .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 (*RS* = .47), and responsive to change as accelerometer-derived sedentary time (*RS* = .39) (for review, see Gorman et al., 2014).

Unhealthy Snacking

At T2, unhealthy snacking was measured using a pre-defined food frequency questionnaire for 21 snack foods (Gardner, 2015). 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, Inauen et al.,

2016; Hagger et al., 2019). In this study, acceptable internal consistency was found across each of the snacking outcomes (α = .74 – .91).

5.2.4 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).

5.2.5 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, Curran & Bauer, 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 $p < .0125$.

5.3 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 PBS, and the rest of study variables are reported in Table 5.1.

Table 5.1. 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	-.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	-.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.

5.3.1 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, β = -.257, $p < .001$, R^2 = .066 (H1a), and rumination, β = -.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.

5.3.2 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 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, β = -.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, β = .483, *SE* = 0.06, *p* < .001, the relationship was weaker at moderate, β = .382, *SE* = 0.04, *p* < .001, and weaker again at high levels of worry, β = .280, *SE* = 0.06, *p* < .001. The results from these analyses are displayed in full in Appendix 3.1 – 3.4.

5.3.3 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 Table 5.2 & 5.3).

Table 5.2 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	
			Direct	-.058***	.009	
			Indirect	-.002 (-.006 – .003)	1.70	.001
Worry	Intentions	Total Sleep Time	Total	-.009	.007	
			Direct	-.007	.007	
			Indirect	-.002 (-.007 – .004)	.0003	.001
Worry	Intentions	Sleep Onset Latency	Total	.559	.419	
			Direct	.543	.418	
			Indirect	.016 (-.037 – .070)	.027	.006
Worry	PBC	Sleep Quality	Total	-.059***	.009	
			Direct	-.045***	.008	
			Indirect	-.014 (-.023 – -.005)**	.005	.285
Worry	PBC	Total Sleep Time	Total	.007	.007	
			Direct	.003	.006	
			Indirect	-.013 (-.021 – -.005)***	.004	.280

Worry	PBC	Sleep Onset Latency	Total	.559	.419	
			Direct	.411	.420	
			Indirect	.148 (.007 – .290)†	.072	.018

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

Table 5.3 Mediation Analysis for Rumination, Intentions & PBC, and sleep behaviours.

Predictor (T1)	Mediator (T1)	Outcome(s) (T2)	Effect	*b (95% CI)*	*S. E*	R^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; S.E = Standard Error, T1 = Timepoint 1, T2 = Timepoint 2; SQ = Sleep Quality, TST = Total Sleep Time

5.4 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 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, Rydstedt, Devereux, & Middleton, 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 PBC, 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 (Radstack 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. Belair 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 & McCarrick, 2021; Eschle, Wale & McCarrick, 2022).

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., Clancy, O'Connor & Prestwich, 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 associationss 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 associated 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.

Chapter 6

General Discussion

6.1 Chapter Summary

In this chapter, first, the findings from the meta-analyses (Chapter 3) and original research studies (Chapters 2, 4 & 5) will first be considered within the context of the thesis aims. Second, the original PCH (see, Figure 1.1) and the extended PCH (see, Figure 1.2) will be reviewed in light of the evidence generated from this thesis. Third, the strengths and limitations of the research presented in this thesis will be considered and suggestions will be made for how future research can improve and extend these findings. Finally, general conclusions are presented distilling the main findings of this thesis.

6.2 Summary of Thesis Findings

6.2.1 Aim I

The sensitivity of PC to environments marked by high stress, such as the workplace, has received little research attention and even less is known about how PC relates to health in this context. Given that previous work has evidenced that job strain (i.e., stress at work) is the most common source of stress in the UK affecting over 79% of adults (HSE, 2022) and the unique potential of PC to prolong the impact of work-related stressors (e.g., Cropley et al., 2006), through work-related worry and rumination, the first aim of this thesis was to '*test the potential role of both general, and work-related PC as a mediating, or potentially moderating, mechanism between job strain and a variety of physical health outcomes and health behaviours'*. Accordingly, this aim was addressed in Chapter 2.

The findings from the first study outlined in Chapter 2, gathered via a cross-sectional survey design, highlighted the potentially damaging nature of PC in the job strain-health outcome relationship. Both types of general *and* work-related worry and rumination predicted significantly higher scores in burnout and somatization, as well as lower scores in sleep quality. Job strain also significantly predicted greater burnout (across each analysis

containing the different types of PC). However, when age and gender were controlled for no significant relationships were observed for the impact of any type of PC on health behaviours. While the moderation hypotheses received little support, such that none of the PC measures (general or work-related) interacted with job strain to predict any of the health outcome variables, the mediation hypotheses received greater support. Indeed, except for the paths from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout, all paths from job strain to PC and from PC to health outcomes (and the indirect paths) were statistically significant.

6.2.2 Aim II

In light of previous work pointing to the damaging and pervasive function of PC as a mediator between stress and disease (Brosschot et al., 2005), a key purpose of this thesis was to shed new light on the experimental evidence testing methods to change PC and their subsequent relationship(s) with health outcomes. Therefore, the second aim was to '*synthesise current experimental evidence aiming to reduce PC and test whether, and how, PC can be changed, along with the subsequent impact on physical health outcomes and health behaviours'*. The findings from this systematic review and meta-analysis can be found in Chapter 3.

The findings noted in Chapter 3 provide the first meta-analytic evidence that a range of psychological interventions can be used to influence PC. Thirty six studies testing (non-pharmacological) interventions produced medium effect sizes for worry and rumination, corresponding to small, but positive, effect sizes for health behaviours (and small-medium positive effect sizes for sleep). Interventions did not, however, produce significant differences (relative to control arms) for physical health outcomes. Interventions produced significantly larger effect sizes for PC when interventions were delivered by healthcare professionals compared to all other alternative methods, and despite no intervention type producing larger effect sizes for PC (when directly compared against other types), there was evidence that studies incorporating psychological detachment style and PC action planning

interventions generated significantly larger effect sizes for health behaviours. Another key finding was the scarcity of studies including measures of rumination in the intervention design (*n* = 7) and, promisingly, the findings for PC were not exclusive to a particular population (age or gender), setting or participant format (group vs. individual), and did not vary across duration of delivery or the number of sessions (single session vs. multi-session. Limitations identified included the objective validity of many of the health outcome measures, as self-report questionnaires were used in most studies; studies were at high risk of bias; the meta-analysis featured a significant degree of heterogeneity across most outcomes. However, the finding that PC can be reduced, and that this has positive downstream benefits for health behaviours, reflects a novel and promising finding for the stress literature.

6.2.3 Aim III

A large focus of this thesis has been to not only understand the extent to which PC relates to ill-health but how to stop this damaging relationship. Therefore, the third aim of this thesis was to '*to test the relative efficacy of an 'augmented' (i.e., including implementation intentions) and 'standard' worry postponement intervention (alongside active and non-active controls) at reducing (state-level) worry and improving sleep over a 2-week period in an (online) 4-armed randomized controlled trial)'*. Given worry and sleep are inherently associated in the literature, and in keeping with the sleep outcomes assessed elsewhere in this thesis, the effect of the interventions was also measured in terms of sleep outcomes. The findings in Chapter 4 show that participants in the augmented arm reported significantly lower worry duration (by ~15 minutes, on average, per day), relative to the active-control arm. Participants in the augmented arm reported significantly shorter worry duration and lower worry frequency, relative to the standard arm. However, the intervention arms did not produce significant improvements in any of the sleep outcomes, relative to the control groups. As such, this thesis has uncovered that creating specific 'if-then' plans for when and how to engage in a worry postponement can produce favourable outcomes for worry

reduction. Yet, future studies are needed to understand how to best translate these effects into positive outcomes for sleep.

6.2.4 Aim IV

The fourth aim of this thesis was to provide a fresh consideration of how PC may serve as a moderator between the intention-behaviour gap (see, Sheeran & Webb, 2016), and to uncover how TPB variables may mediate the relationship between PC and a range of health behaviours. It is possible, given their role in emotion regulation in the intention-health behaviour relationship, that Theory of Planned Behaviour (TPB) variables may attenuate the association between PC and health behaviours. Therefore, the fourth aim of this thesis was to '*test whether PC moderates the relationship(s) between the TPB constructs and behaviour, as well as whether the relationships between PC and health behaviours are mediated by intentions and perceived behavioural control*. The findings from this study are presented in Chapter 5.

In a prospective design, 650 participants completed baseline measures of TPB constructs and PC (worry and rumination) and 590 completed follow-up (Time 2) measures of health behaviours (physical activity, sleep, sedentary activity, unhealthy snacking) one week later. The findings revealed that worry and rumination (at T1) predicted significantly poorer sleep quality; however, both types of PC were statistically unrelated to all other health behaviours. Worry, but not rumination, and PBC interacted to predict significantly lower physical activity frequency, yet no other moderation effects were observed. Specifically, while PBC was positively associated with physical activity frequency at low levels of worry, the relationship was weaker at moderate, and weaker again at high levels of worry. Further, consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant. These findings provide novel longitudinal support that PC is associated with poorer sleep quality, while also revealing new relationships between the components of PC, PBC and health behaviours. It was of specific note that the mediation

models received full support in relation to the sleep outcomes, supporting previous work that PC poses a particular threat for sleep behaviours (for review, see Clancy et al., 2020).

6.3 The Perseverative Cognition Hypothesis and The Extended Perseverative Cognition Hypothesis: Consideration of the Evidence.

The findings from this thesis offer some support for the EPCH and varying degrees of support for the original PCH. In the main, many of the research findings align with the central conceptual tenet tested in this thesis, that the relationship between stress and ill-health is intensified when a stressor is subject to repetitive thought, through perseverative cognition such as worry and/or rumination. However, the findings in this thesis place greater emphasis on the association between PC and health behaviours, and sleep in particular, than between PC and physical health outcomes. Throughout this thesis, PC serves as an important predictor for unhealthier behaviours and is a significant determinant of poorer sleep (see, Chapters 3 & 5), although fewer significant relationships emerged for physical health outcomes (see Chapters, 3 & 4) There were, though, a handful of exemptions to this high-level overview of findings: both work-related worry and rumination predicted significantly higher instances of burnout and somatization (Chapter 2); neither of the worry postponement interventions produced significant changes in any of sleep outcomes (Chapter 4); null effects were present for the prospective relationship between worry/rumination and health behaviours (Chapter 5). Equally, there are methodological arguments to be made as to how precise the measurements of physical health outcomes were in this thesis, and two of the three empirical studies did not include any measures of physical health, all of which will be discussed at length later (see section 6.4).

Several findings in this thesis support the proposal that there may be scope for an additional route to pathogenic disease due to PC modifying health behaviours. Consistent with previous research, showing PC is associated to health-risk behaviours (see Clancy et al. 2016), both general and work-related PC predicted significantly poorer sleep quality. The

mediation models for these relationships showing PC mediates the indirect pathway between job strain (a form of stress) and sleep quality were also significant, indicating that PC plays an important role in understanding how (work-related) stress influences sleep quality (Chapter 2). There was also some support for the PCH in this chapter. Both work-related worry and rumination predicted significantly higher instances of burnout and somatization, while the same associated mediation models were also significant, indicating PC contributes to the association between (work-related) stress and markers of physical ill-health. Yet, there was little support for the PCH in Chapter 3, wherein psychological interventions did not yield significant effects for physical health outcomes. However, the EPCH did gain substantial backing. Indeed, interventions that produced significant (medium-sized) reductions in PC also produced significant (small-sized) improvements in health behaviours. Given these interventions were brief (around 8 days, on average), and often self-administered, this finding is all the more notable. While it was true no significant relationships were observed for physical health outcomes, it should be stated that many of these interventions did not target determinants of behaviour (e.g., planning activities; goal orientated tasks), so this is of little surprise. Equally, these interventions were primarily developed to reduce worry/rumination and not the physical health outcomes. Therefore, it's likely that if future interventions aim to target the PC-health behaviour relationship they are likely to be more successful. Also, for many of the physical health outcomes, a longer follow-up period would be needed in order for any beneficial effects to emerge, while shorter follow-up periods (i.e., daily-diary designs) are needed for health behaviours.

Further, certain types of interventions that targeted specific facets of PC (such as worry action planning and psychological detachment) produced significantly greater improvements in health behaviours, showing further support for the potential link between PC and health behaviours attested in the EPCH (see, Chapter 3). And while sleep outcomes were unaffected by the interventions featured in Chapter 4, this was likely due to the characteristics of the intervention content employed, rather than the raw association

between these variables. As such, caution should be taken before drawing conclusions between these findings and how they relate to both the PCH and EPCH. What is noteworthy, however, is that participants in the augmented intervention arm reported significantly shorter worry duration and lower worry frequency (on average) across the 14-day intervention period. As such, it is important that prospective interventions wishing to establish associationss between PCH variables and health outcomes consider the potential utility of health behaviour change techniques to do so, such as the implementation intentions used in this thesis. In turn, this may pave the way for interventions that not only have a positive influence on PC but also on health behaviours and physical health outcomes alike.

Chapter 5, on the other hand, featured clear support for the EPCH, with both worry and rumination predicting (prospective) reductions in sleep quality. Additionally, the relationship between PBC and physical activity weakened as worry increased and, in the mediation models, the relationship between worry/rumination and sleep outcomes was mediated by PBC. In this sense, PC adversely affected PBC which, in turn, reduced the likelihood of a behaviour being enacted, which is significant in the case of sleep given its close ties with health and wellbeing (Broomfield & Espie, 2005). Few, if any, inferences can be drawn from Chapter 5 in relation to the PCH; indeed, no measures of physical health were included in this study as the focus was on the behavioural mechanisms that may interact with PC to predict downstream health behaviours. Axiomatically, predicting physical health outcomes from determinants of the TPB model was not thought to be a methodologically viable decision given they relate to behaviour(s) and not physiological outcomes. Nonetheless, when considering the new set of findings provided by the research in this thesis wholistically, they document the connection between PC and health behaviours and the dual risk factor they can pose to health outcomes, supporting the EPCH.

On the other hand, however, there were a handful of inconsistencies from the results within this thesis that challenge some of the theoretical predictions of the PCH and the EPCH. Indeed, they pose the question over how much support there is for the EPCH in absence of

the significant effects found for sleep in this thesis. For instance, in Chapter 2, with the exception of sleep, no significant relationships were observed for the impact of any type of PC on health behaviours. Also, none of the PC measures (general or work-related) interacted with job strain to predict health behaviours. Equally, in Chapter 4, neither the augmented worry postponement intervention or the standard worry postponement intervention yielded beneficial reductions in sleep outcomes. However, it is possible that this null effect was related to the intervention properties rather than the relationship between worry and sleep as a health behaviour. Indeed, the finding that the augmented arm resulted in larger reductions in worry duration than the active-control, and that this effect was unique to the augmented arm, provides some support for the notion that intervention properties are key in determining the associationss between stress states (such as PC) and health outcomes. Equally, in Chapter 5, while prospective relationships were present between worry/rumination and sleep outcomes, null effects were observed for physical activity and sedentary behaviours, and unhealthy snacking. As pointed out in Chapter 5, this finding was not entirely inconsistent with previous studies exploring these outcomes in relation to PC; however, they do oppose the EPCH in that, as was the case with sleep, it would be expected that increases in PC would lead to respective increases in each of these health behaviours. In this sense, these findings indicate that the pathway between PC and health behaviours is complex. As such, in order to disentangle the types of health behaviours that constitute support for the EPCH, future studies are needed which include specialised measures of distinct behaviours. For example, daily-dairy studies that include ambulatory assessed sleep, physical activity and food diaries to capture unhealthy snacking would be a significant advance for the field and offer a new test for the EPCH.

Some of these caveats may be explained by the measurement error often reported in health behaviour research. Unlike the predictably stable way in which physiological stress systems respond to psychological stress (see, Cannon, 1939; McEwen, 1998; Selye, 1950), models of health behaviour are less static and incorporate an array of other factors that may

influence behavioural enactment (e.g., capability (related to PBC), motivation (related to intention), or opportunity; Michie et al., 2011). Indeed, a plethora of research spanning several decades has shown that health behaviours reflect distinct variables which require careful consideration in their own right (Emery, 1980; Calnan & Rutter, 1986; Prestwich et al., 2015). Therefore, health behaviours are of greater influence to individual differences and from the environmental circumstances they exist in, and therefore, cumulatively, more research is required to understand this interplay further . Indeed, as is noted by Conner and Norman (2017), a great challenge facing health psychologists is determining the most optimal methods to not only influence health behaviour change but to maintain them in the face of competing priorities and daily living.

Irrespective of the degree to which these findings support either the EPCH or PCH, what is clear is that the evidence generated from this thesis has significant implications for population health and has potential to contribute toward the prevention of health behaviour-related disease. In particular, the findings from the meta-analysis and RCT provide advances in our understanding of PC as a transdiagnostic risk factor for health behaviours and show that, when left untamed, PC can have a negative influence on health behaviours. In Chapter 3, it was shown, for the first time, that reductions in PC (following experimental intervention) lead to improvements in health behaviours. Further, the findings from the RCT (in Chapter 4) provide health care professionals with a new intervention strategy to target PC with. Here, the augmented worry postponement arm resulted in larger reductions in worry duration than the standalone worry postponement arm, equating to around 15 minutes less worry (each day) across the two-week study period. As such, prospective interventions should carefully consider the utility of implementation intentions not only as a means to promote adherence to interventions with planning components, but as a therapeutic method in their own right. The structure provided by the implementation intentions tested in Chapter 4 likely provide participants with a framework of how and when to manage worry, rather than allowing it to be freely dispersed throughout the day. This, alone, is a significant finding for health care

professionals to contemplate especially considering worry postponement interventions are a commonly used method to treat psychological disorders such as GAD (Centre for Clinical Interventions, 2022). Equally, also in Chapter 4, the finding that participants in the augmented arm reported significantly shorter worry duration and lower worry frequency, relative to the standard arm, sheds new light on the utility of worry postponement interventions. Prospective studies would therefore do well to consider the benefit of other health behaviour change approaches (such as the implementation intentions used here) to enhance other traditional methods of stress reduction. Relatedly, results in Chapter 5 provide further conceptual considerations for behaviour change interventionists; indeed, as targeting either worry or PBC may result in downstream changes in physical activity, it is possible this may also be true for other health behaviours tested in different contexts to those used in this thesis. Thus, discovering how, and when, the mechanisms behind PBC interact with cognitive processes such as PC provide researchers with new opportunities to explore how best to manage worry and have beneficial effects on health. Accordingly, these findings position PC as another important determinant of health behaviours and highlight its viability as a potentially fruitful intervention target for engendering positive changes in various aspects of population health.

6.4 Strengths and Limitations of the Thesis & Future Avenues for Research

The research conducted in this thesis has several strengths. First, up until the completion of this thesis, PC research had an overreliance on cross-sectional measurements and overlooked the unique potential utility of psychological interventions at influencing PC and health-related constructs. Here, the findings of this thesis extend our understanding of PC and how it relates to health outcomes and behaviours. Importantly, it does so via more robust methodological techniques, such as meta-analysis, randomized trials with various intervention arms, and prospective designs. This therefore provides us with a greater deal of confidence in the inferences we draw from these studies and the rigour they represent. Put

simply, the findings of this thesis represent an important step forward for PC research and the stress literature more generally by significantly enhancing the evidence-base in the area of perseverative cognition. As outlined in section 6.3, future research can build upon these findings to discover new ways of tackling the pervasive nature of PC and the potentially damaging impact it holds for health outcomes. For example, implementation intentions may be used as a technique to reduce worry, while other elements of an intervention could focus on how PBC unlocks the potential to adopt healthier lifestyles.

Second, despite some of the null effects observed for the EPCH, the research conducted here significantly extends a conceptual framework still in its infancy. The meta-analysis (Chapter 3) suggesting a possible association between PC and health behaviours within experimental studies and the fact PC variables interact with constructs from other conceptual models (i.e., PBC, from the TPB; Chapter 5) reflects a fruitful opportunity for future studies to unpick this relationship in greater detail. The measurement of several sleep variables within the context of an RCT (Chapter 4) also reflect new territory covered for testing of the EPCH and Chapter 2 contains new knowledge on how work-related PC functions under the umbrella of the EPCH for the first time. Further, the contents of Chapter 4 feature methodological advances for the PC-intervention field. For the first time, a standalone worry postponement intervention was complemented with implementation intentions, where a significant reduction in worry duration and frequency was found. The testing period for a worry postponement intervention was also expanded to 14 days, which was a new test for this technique. A multi-level daily-diary design to capture not only how worry varies within-participants, but also how these variations relate to sleep outcomes measured the previous evening, was also used. These advances have significant implications not only for the findings of this thesis in terms of their precedence over measurement sensitivity, but also in terms of their increased validity for future studies that aim to measure state-level constructs over time. On another note, where they were included (e.g., neuroticism, Chapter 2; baseline clinical heterogeneity, Chapter 3), the relevant covariates, interaction terms, and sensitivity

analysis highlighted by this thesis unveil new considerations for the EPCH that would have otherwise been left uncovered.

Third, the existing literature was searched and synthesized rigorously via systematic review and meta-analysis, resulting in a publication in *Health Psychology* (i.e., McCarrick et al., 2021)*;* importantly, this article is attracting a strong citation rate reflecting the importance of these findings and their significance for the field. It has also featured in newspaper reports and several blogs internationally, demonstrating the public appeal for the work completed as part of this thesis. Fourth, the associations between various types of PC (worry, rumination, brooding) and multiple health outcomes have been examined, and new ways to tackle PC have been uncovered, addressing a number of significant gaps in the existing literature. A key finding is that only seven studies exist that specifically aim to reduce rumination in the context of a randomised intervention, which is not only hugely surprising given its role in the aetiology of several mental health conditions (see, O'Connor, O'Connor & Marshall, 2007) but serves as a timely call to action for future studies.

Fifth, beyond the obvious inferences related to health psychology regarding the theoretical significance of these findings, there are other implications that are worthy of mention. A consistent finding in this thesis was that increased PC predicts poorer sleep, yet few of the interventions identified included it as a primary outcome. Other research points to the significance of the association between PC and sleep outcomes too (e.g., Clancy et al., 2022), emphasizing the need for future research with more sensitive instruments, such as polysomnography (e.g., Croy, Smith, Gidlog-Gunnarsson & Persson-Waye, 2016), to explore the intricate interrelationships between PC and sleep across time and in different populations. Sixth, Chapter 2 describes the first piece of empirical research to examine the associations between markers of occupational stress (i.e., job strain), PC, and a variety of health outcomes. The fact PC serves as a key mediator in this relationship supports the PCH and also demonstrates the far-reaching impact PC can have, particularly in environments susceptible to increased stress. It is therefore interesting that the 'psychological detachment'

interventions in the meta-analysis, which rest upon the core principle of 'switching off' or 'cancelling out' the environment stressors, contained no measures of job strain. As shown in Chapter 2, high demands and low control at work creates an environment for PC to flourish, making job strain a prime candidate for prospective occupational interventions. Seventh, the findings from Chapter 5, showing PC and PBC interact to predict physical activity and that, consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant, unlocks new knowledge as well as future opportunities for research. Eighth, all the studies in this thesis were preregistered and the data are available via open repositories, so not only does this thesis's findings extend knowledge via enhancing the evidence threshold for PC research but they do so in way in which champions open science and reproducibility.

However, as with any research, there are several limitations to this thesis that must be acknowledged. The first, and perhaps most detrimental, relates to the measurement of PC and health outcomes. While it has been noted that relatively few studies on rumination exist that also consider measures of health, those that do adopt a range of scales or single-item measurements that retract from the validity and reliability of these research findings. It is also a likely reason, more broadly, why there are mixed findings in the literature in relation to rumination. Similar issues exist for worry. Despite the introduction of the Penn State Worry Questionnaire (Meyer et al., 1990) as the predominant (gold-standard) measure for trait worry, an equivalent state-level measure has not reached the same psychometric standards. The variety of worry measures employed by studies in the meta-analysis reflects this, and it is also of concern that few validated scales exist for specific sub-types of worry, despite their prevalence in various health models of disease-specific progression (for review, see Khodayarifard, Mansouri, Besharat & Lavasani, 2017). The meta-analysis (Chapter 3) highlights the extent of this problem, as in the studies measuring worry about pain, sleep, and work, single-item visual analogue scales were the most commonly used measures. As

such, improved measurement specificity of PC and its associated components would enable more meaningful conclusions to be drawn from the cumulative literature.

Relatedly, another factor that must be considered is the measurement properties of the health behaviours. As ‘health behaviour’ is a broad term that encapsulates a wide number of constructs (e.g., sleep; physical activity; diet; alcohol consumption; drug use etc.), there is a significant degree of heterogeneity associated to its measurement (Schneider, Pfarr, Schneider & Ulrich, 2012). This problem is also reflected in the relative lack of well-defined, validated, and time-sensitive instruments freely available to capture meaningful change in these constructs within studies. To counteract this, instruments to collect data on a large range of health behaviours have been developed to provide higher-level overviews on how people behave in regard to their health. One of these, the Good Health Practices scale (Hampson, Edmonds & Goldberg, 2017) captures sixteen health promoting behaviours and, as such, was employed as a measure in this thesis (see, Chapter 2). However, null effects were observed for the relationship between PC and health behaviours. A possible reason for this may be that the diverse range of health behaviours presented in the instrument do not converge in the same way that health specific scales do, a topic that has attracted much broader academic debate (see, Mew et al., 2020; Machielsen et al., 2021).

Therefore, care must be taken before dismissing the relationship between PC and health behaviours in Chapter 2, as the time efficiency and the low participatory burden of this scale was prioritised over the superior measurement properties of other more established behaviour-specific psychometric instruments available (e.g., see Conner & Norman, 2017, for review). This was a central reason why behaviour-specific scales were used in Chapter 5 which, interestingly, led to a number of significant associations between PC and health behaviours. Furthermore, in Chapter 4, daily-diary assessments of sleep, as a health behaviour, were used in view of maximising measurement sensitivity. Text messages were sent to participants each morning (for 14 days) asking them to rate their sleeping experience. Although no effects for sleep were observed across each of the relevant

outcomes (sleep onset latency; sleep quality; sleep disturbance), descriptive analysis of this data revealed people took, on average, 28.21 minutes (SD = 12.14) to provide an estimation of their sleep parameters from the previous night, added to the fact text messages were scheduled to be sent 30 minutes post-estimated wake-time. Therefore, while this does not invalidate these responses by any means, it would have been preferable to receive responses sooner in view of gaining the most accurate estimation possible. Thus, this reinforces the message that while null effects are present in Chapter 4, consideration must be given to these measurement issues. Future studies are needed that employ objective measures of sleep with superior ecological validity (e.g., such as Actigraphy; see, Martin & Hakim, 2011).

There are also a number of points that must be acknowledged in relation to how physical health outcomes were operationalized and measured in this thesis which have important implication for the strength of the inferences that can be directly made to the PCH. It is important to remember that, according to Brosschot et al. (2006) PC moderates the health consequences of stressors because it can prolong stress-related affective and *physiological activation*, both in advance of and following stressors. This thesis did not contain any direct measures of physiological activation. Instead, due to practical and funding limitations proxy measures were adopted such as somatization and physical symptoms of burnout (see Chapter 2). Moreover, just two studies in the meta-analysis took measurements of physiological activation (i.e., cortisol awakening response, Teismann et al., 2014; ambulatory assessed heart rate, Versluis et al., 2018) but the samples of interest were small and predominantly female, calling into question the generalisability of the findings. Brosschot et al. (2006) notes that PC acts upon somatic disease via affecting the cardiovascular, immune, endocrine, and neurovisceral systems. As such, excluding these aforementioned exceptions, none of the studies in this thesis provided a direct test of the PCH, meaning caution should be exercised in interpreting the impact of PC on physical health outcomes in this thesis; particularly as previous (more precisely designed) studies have found consistent significant

effects for the association between PC and markers of physiological functioning (e.g., worry & chronic HR elevation, Palatini & Julius, 1997; worry & myocardial infarctions, Brosschot et al., 2006; Ottaviani et al., 2018). Future studies should therefore look to build upon these shortcomings via including more objective measures of physiological activation in prospective interventions as a lens to directly test the PCH using experimental methods. This limitation leads closely onto another, the fact that all of the measurements from the empirical studies in this thesis were collected using self-reported, online, methods. Partly owing to the restrictions brought about by the Coronavirus pandemic in 2019, plainly, there was no other viable option at the time this research was conducted.

There is one final methodological and statistical limitation that must be considered, not only in acknowledgement of how this may limit the findings of this thesis, but in the best interests of advancing psychological science more generally . There may be a bi-directional relationship between PC and health outcomes (see Clancy et al., 2016). We cannot rule out the possibility of 'reverse causality', whereby PC correlates with negative health outcomes only because poorer health status produces more PC, particularly given the overreliance of cross-sectional methodologies in the PC literature. While the approach to detect how changes in PC lead to changes in health outcomes is a promising step forward (see Chapter 3) and the randomised interventions provide more robust tests, they are also not free from measurement bias. For instance, the studies in the meta-analysis, considered as the most methodologically rigorous, may still be subject to various degrees of confounding and selection bias. This could mean that PC and health relationships are conditionally associated with confounding variables, or within levels of a confounder. A typical example may be when worry is associated to somatic outcomes, but when these inferences are based only on trait measures. Thus, daily (state-level) variations in worry obscured by participant-level heterogeneity may confound these relationships, either inflating or downplaying the influence of the predictor variable.

A common to approach for how to deal with confounding bias in psychological science is to 'adjust for it' by including certain covariates in a multiple regression model and alter the effect estimates calculated thereafter; indeed, this was done in Chapter 2 when controlling for neuroticism and in the meta-regressions noted in Chapter 3. However, this has its own problems as multiple regression models treat predictors on equal merit when, conceptually, we place varying degrees of emphasis/importance of the predictors in our models (e.g., prevalence of worry, relative to rumination in specific contexts; inclusion of several demographic variables), and sometimes it is difficult to highlight which variable requires adjustment (Arnold et al., 2022; Gilthorpe et al., 2009). Directed acyclic graphs (DAGs) that represent causal effects between multiple variables is a relatively recent methodology that future research may benefit from considering. This method aims to understand whether bias (i.e., confounding) is potentially reduced or increased as a function of a covariate within a causal inference model (see, Tennant et al., 2021). Thus, DAGs can be used to identify a minimal sufficient set of variables to be used in a multivariable regression model for the estimation of a causal effect, which may be particularly useful given the complexity of the relationship between stress, PC, and health outcomes. Consequently, it is recommended, on a broader note, that future studies aim to push the experimental, methodological and statistical boundaries commonly associated to the PC literature in view of enhancing our understanding of how cognitive processes, such as PC, interact with health outcomes to predict pathogenic disease.

6.5 General Conclusions

This thesis aimed to expand the evidence base testing the original PCH and the EPCH in view of ascertaining the pathways which stress, through PC, influences physical and behavioural health outcomes. In parallel, the aim was to do this while advancing current methodological practices in the field, via using meta-analysis, prospective daily-diary designs, and randomised controlled trials to enhance the existing evidence base. Cumulatively, the research findings from this thesis provide some support for the EPCH, with

regard to sleep in particular, and varying degrees of support for the original PCH. In the main, many of the research findings align with the central conceptual tenet tested in this thesis, that the relationship between stress and ill-health is intensified when a stressor is subject to repetitive thought, through perseverative cognition such as worry and/or rumination, and that this translates to poorer health behaviours (and in some cases in this thesis, more damaging physical health outcomes, i.e., burnout and somatization, see Chapter 2). Specifically, throughout this thesis, PC serves as an important predictor for unhealthier behaviours and is a significant determinant of poorer sleep (support from, Chapters 2, 3 & 5); General and work-related worry and rumination significantly mediated, often independently, the relationship between job strain and burnout, somatization, and sleep quality (see Chapter 2); meta-analysis revealed that psychological interventions can reduce PC and that these positive changes often translate into downstream benefits for health behaviours (see Chapter 3). Worry, but not rumination, moderated PBC-physical activity frequency relations and consistent with mediation, the indirect paths from both worry and rumination, through PBC, to sleep quality and total sleep time were significant (Chapter 6). However, while the augmented worry postponement intervention in Chapter 4 resulted in significant reductions in worry duration (by ~15 minutes per day across the 2 week study period), it was unsuccessful in impacting sleep. Therefore, together, the overall weight of evidence in this thesis points to the same conclusion set out by Clancy et al. (2016) in the EPCH: that PC may pose a serious, indirect, risk for disease processes via modifying health behaviours. Further work is needed to elucidate how PC interacts with other components known to predict (or influence) disease processes and to uncover new interventions that can attenuate the now axiomatic relationship between PC and ill-health.

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(References with an Asterix are primary studies contained in the meta-analysis; Chapter 3)

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Appendices

Appendix 1.1 – Chapter 2 Supplementary Material A

Appendix 1.1.1: Covariate Analyses

First, we assess the additional contribution of age, gender, and neuroticism to the PC-health outcome models. For these hierarchical regression models, the Perseverative Cognition (PC) measure (i.e., general worry/rumination/work-placed worry/rumination) is entered at step 1, age and gender at step 2, and neuroticism at step 3. These can be found below in Tables 1-16 (Hypothesis 1).

Appendix 1.1.2: Moderation Analyses

Second, Tables 17-20 contain the output from the moderation analyses, showing that none of the PC measures (general or work-related) moderated the relationships between job strain and any of the outcome variables (Hypothesis 2).

Appendix 1.1.3 Sensitivity Regression Analyses

Third, an additional set of sensitivity regressions were used to test whether each type of PC (general worry & rumination (as a set); work-related worry and rumination (as a set); both general and work-related worry & rumination (combined together)) independently predicted poorer health outcomes. This approach was employed to determine if variation across the different types of PC independently predicted poorer health across the outcome variables. This may have implications from an applied perspective, as targeting worry and rumination together (rather than one alone) may produce more favourable changes in the outcomes.

Appendix 1.1.4 Brooding Analyses

Fourth, in order to determine that the effects of rumination on health are accurately reflected via the ‘overall’ measure used for the Rumination Response Scale (Treynor et al., 2003), and are not primarily driven by the subscale of Brooding, we re-assessed all of the analyses with the Brooding subscale exclusively, in place of the ‘overall’ measure.

Across the regression analyses for each predictor variable can be found its corresponding section and table. In these tables; *beta* indicates the standardized regression weights; *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively; and * indicates $p < .05$. and ** indicates $p < .01$. When more than one significant predictor was present the largest change R^2 was used to determine the best fitting model.

Appendix 1.1.1 Covariate Analyses

General Worry

Appendix 1.1.1: Table 1

Hierarchical regression: general worry on Burnout (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Worry	0.47**	[0.40, 0.54]	
			R^2 = .222**
			95% CI[.17,.28]
Step 2			
Worry	0.47**	[0.40, 0.54]	
Age	0.01	[-0.07, 0.08]	
Gender	0.00	[-0.07, 0.07]	
			R^2 = .223**
			95% CI[.17,.27]
Step 3			
Worry	0.33**	[0.25, 0.42]	
Age	0.01	[-0.06, 0.09]	
Gender	-0.01	[-0.09, 0.06]	
Neuroticism	0.24**	[0.15, 0.32]	
			R^2 = .258**
			95% CI[.20,.31]

Appendix 1.1.1: Table 2

Hierarchical regression: general worry on Somatization (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Worry	0.41**	[0.34, 0.48]	
			R^2 = .168**

			95% CI[.12,.22]
Step 2			
Worry	0.39**	[0.31, 0.46]	
Age	-0.10**	[-0.17, -0.02]	
Gender	0.02	[-0.06, 0.09]	
			R^2 = .176**
			95% CI[.12,.23]
Step 3			
Worry	0.25**	[0.17, 0.34]	
Age	-0.09*	[-0.17, -0.02]	
Gender	0.00	[-0.07, 0.08]	
Neuroticism	0.23**	[0.14, 0.31]	
			R^2 = .209**
			95% CI[.15,.26]

Appendix 1.1.1: Table 3

Hierarchical regression: general worry on Health Behaviours (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Worry	0.04	[-0.03, 0.12]	
			R^2 = .002
			95% CI[.00,.01]
Step 2			
Worry	0.09*	[0.01, 0.17]	
Age	0.03	[-0.05, 0.11]	
Gender	-0.20**	[-0.28, -0.12]	
			R^2 = .038**
			95% CI[.01,.07]
Step 3			
Worry	-0.08	[-0.18, 0.01]	
Age	0.04	[-0.04, 0.11]	
Gender	-0.22**	[-0.30, -0.14]	
Neuroticism	0.30**	[0.21, 0.39]	
			R^2 = .094**
			95% CI[.05,.13]

Appendix 1.1.1: Table 4

Hierarchical regression: general worry on Sleep Quality (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Worry	0.34**	[0.27, 0.41]	
			R^2 = .115**

			95% CI[.07,.16]
Step 2			
Worry	0.33**	[0.26, 0.41]	
Age	0.03	[-0.05, 0.11]	
Gender	0.06	[-0.02, 0.13]	
			R^2 = .120**
			95% CI[.07,.16]
Step 3			
Worry	0.12**	[0.03, 0.21]	
Age	0.04	[-0.03, 0.12]	
Gender	0.03	[-0.04, 0.11]	
Neuroticism	0.37**	[0.28, 0.45]	
			R^2 = .206**
			95% CI[.15,.25]

General Rumination

Appendix 1.1.1: Table 5

Hierarchical regression: general rumination on Burnout (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Rumination	0.46**	[0.39, 0.53]	
			R^2 = .212**
			95% CI[.16,.26]
Step 2			
Rumination	0.46**	[0.39, 0.54]	
Age	0.03	[-0.05, 0.10]	
Gender	0.07	[-0.00, 0.14]	
			R^2 = .219**
			95% CI[.16,.27]
Step 3			
Rumination	0.34**	[0.26, 0.41]	
Age	0.04	[-0.04, 0.11]	
Gender	0.03	[-0.04, 0.10]	
Neuroticism	0.27**	[0.19, 0.35]	
			R^2 = .271**
			95% CI[.21,.32]

Appendix 1.1.1: Table 6

Hierarchical regression: general rumination on Somatization (N = 650).

Predictor	*beta*	95% CI	Fit

		[LL, UL]	
Step 1			
Rumination	0.49**	[0.42, 0.55]	
			R^2 = .237**
			95% CI[.18,.29]
Step 2			
Rumination	0.47**	[0.40, 0.54]	
Age	-0.06	[-0.13, 0.02]	
Gender	0.06	[-0.01, 0.13]	
			R^2 = .242**
			95% CI[.19,.29]
Step 3			
Rumination	0.38**	[0.30, 0.45]	
Age	-0.05	[-0.12, 0.02]	
Gender	0.03	[-0.04, 0.10]	
Neuroticism	0.20**	[0.12, 0.27]	
			R^2 = .270**
			95% CI[.21,.32]

Appendix 1.1.1: Table 7

Hierarchical regression: rumination on Health Behaviours (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Rumination	-0.01	[-0.08, 0.07]	
			R^2 = .000
			95% CI[.00,.01]
Step 2			
Rumination	0.00	[-0.08, 0.08]	
Age	0.00	[-0.08, 0.09]	
Gender	-0.17**	[-0.25, -0.10]	
			R^2 = .030**
			95% CI[.01,.06]
Step 3			
Rumination	-0.15**	[-0.24, -0.06]	
Age	0.02	[-0.06, 0.10]	
Gender	-0.23**	[-0.30, -0.15]	
Neuroticism	0.32**	[0.24, 0.41]	
			R^2 = .105**
			95% CI[.06,.15]

Appendix 1.1.1: Table 8

Hierarchical regression: general rumination on Sleep Quality (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit

Step 1			
Rumination	0.31**	[0.23, 0.38]	
			R^2 = .094**
			95% CI[.06,.14]
Step 2			
Rumination	0.31**	[0.23, 0.39]	
Age	0.04	[-0.04, 0.12]	
Gender	0.11**	[0.03, 0.18]	
			R^2 = .109**
			95% CI[.07,.15]
Step 3			
Rumination	0.13**	[0.05, 0.22]	
Age	0.06	[-0.02, 0.13]	
Gender	0.05	[-0.02, 0.12]	
Neuroticism	0.37**	[0.29, 0.45]	
			R^2 = .210**
			95% CI[.15,.26]

Work related Worry

Appendix 1.1.1: Table 9

Hierarchical regression: workplace worry on Burnout (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Workplace Worry	0.64**	[0.58, 0.70]	
			R^2 = .415**
			95% CI[.36,.46]
Step 2			
Workplace Worry	0.64**	[0.58, 0.70]	
Age	-0.03	[-0.10, 0.03]	
Gender	0.07*	[0.01, 0.13]	
			R^2 = .420**
			95% CI[.36,.47]
Step 3			
Workplace Worry	0.56**	[0.50, 0.63]	
Age	-0.01	[-0.07, 0.05]	
Gender	0.04	[-0.02, 0.10]	
Neuroticism	0.18**	[0.11, 0.24]	
			R^2 = .444**
			95% CI[.39,.49]

Appendix 1.1.1: Table 10

Hierarchical regression: workplace worry on Somatization (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit

Step 1			
Workplace Worry	0.47**	[0.40, 0.53]	
			R^2 = .217**
			95% CI[.16,.27]
Step 2			
Workplace Worry	0.45**	[0.38, 0.51]	
Age	-0.14**	[-0.21, -0.07]	
Gender	0.08*	[0.01, 0.15]	
			R^2 = .238**
			95% CI[.18,.29]
Step 3			
Workplace Worry	0.36**	[0.28, 0.43]	
Age	-0.11**	[-0.18, -0.05]	
Gender	0.04	[-0.03, 0.11]	
Neuroticism	0.21**	[0.14, 0.29]	
			R^2 = .273**
			95% CI[.21,.32]

Appendix 1.1.1: Table 11

Hierarchical regression: workplace worry on Health Behaviours (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Workplace Worry	0.02	[-0.05, 0.10]	
			R^2 = .001
			95% CI[.00,.01]
Step 2			
Workplace Worry	0.04	[-0.04, 0.11]	
Age	0.01	[-0.07, 0.09]	
Gender	-0.18**	[-0.26, -0.10]	
			R^2 = .031**
			95% CI[.01,.06]
Step 3			
Workplace Worry	-0.08*	[-0.17, -0.00]	
Age	0.04	[-0.03, 0.12]	
Gender	-0.23**	[-0.31, -0.15]	
Neuroticism	0.29**	[0.21, 0.37]	
			R^2 = .096**
			95% CI[.05,.14]

Appendix 1.1.1: Table 12

Hierarchical regression: workplace worry on Sleep Quality (N = 650).

Predictor	*beta*	95% CI	Fit

		[LL, UL]	
Step 1			
Workplace Worry	0.31**	[0.24, 0.39]	
			R^2 = .097**
			95% CI[.06,.14]
Step 2			
Workplace Worry	0.30**	[0.23, 0.38]	
Age	-0.02	[-0.09, 0.06]	
Gender	0.12**	[0.05, 0.20]	
			R^2 = .111**
			95% CI[.07,.15]
Step 3			
Workplace Worry	0.15**	[0.07, 0.23]	
Age	0.03	[-0.04, 0.10]	
Gender	0.05	[-0.02, 0.13]	
Neuroticism	0.37	[0.29, 0.45]	
			R^2 = .215**
			95% CI[.16,.26]

Work related Rumination

Appendix 1.1.1: Table 13

Hierarchical regression: workplace rumination on Burnout (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
(Intercept)			
Workplace Rumination	0.58**	[0.52, 0.64]	
			R^2 = .334**
			95% CI[.28,.39]
(Intercept)			
Workplace Rumination	0.57**	[0.51, 0.63]	
Age	-0.06	[-0.13, 0.00]	
Gender	0.10**	[0.03, 0.16]	
			R^2 = .344**
			95% CI[.29,.39]
(Intercept)			
Workplace Rumination	0.52**	[0.46, 0.57]	
Age	-0.00	[-0.06, 0.06]	
Gender	0.03	[-0.04, 0.09]	
Neuroticism	0.34**	[0.28, 0.40]	
			R^2 = .447**
			95% CI[.39,.49]

Appendix 1.1.1: Table 14

Hierarchical regression: workplace rumination on Somatization (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Workplace Rumination	0.39**	[0.32, 0.46]	
			R^2 = .155**
			95% CI[.11,.20]
Step 2			
Workplace Rumination	0.38**	[0.31, 0.45]	
Age	-0.16**	[-0.24, -0.09]	
Gender	0.10**	[0.03, 0.17]	
			R^2 = .183**
			95% CI[.13,.23]
Step 3			
Workplace Rumination	0.33**	[0.26, 0.39]	
Age	-0.11**	[-0.18, -0.04]	
Gender	0.03	[-0.04, 0.10]	
Neuroticism	0.31**	[0.24, 0.38]	
			R^2 = .272**
			95% CI[.21,.32]

Appendix 1.1.1: Table 15

Hierarchical regression: workplace rumination on Health Behaviours (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Workplace Rumination	-0.04	[-0.11, 0.04]	
			R^2 = .001
			95% CI[.00,.01]
Step 2			
Workplace Rumination	-0.03	[-0.11, 0.05]	
Age	-0.00	[-0.08, 0.08]	
Gender	-0.17**	[-0.25, -0.09]	
			R^2 = .031**
			95% CI[.01,.06]
Step 3			
Workplace Rumination	-0.07	[-0.15, 0.00]	
Age	0.04	[-0.03, 0.12]	
Gender	-0.23**	[-0.31, -0.15]	
Neuroticism	0.26**	[0.19, 0.34]	
			R^2 = .095**
			95% CI[.05,.13]

Appendix 1.1.1: Table 16

Hierarchical regression: workplace rumination on Sleep Quality (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Workplace Rumination	0.18**	[0.10, 0.25]	
			R^2 = .031** 95% CI[.01,.06]
Step 2			
Workplace Rumination	0.17**	[0.09, 0.24]	
Age	-0.04	[-0.12, 0.04]	
Gender	0.14**	[0.06, 0.22]	
			R^2 = .049** 95% CI[.02,.08]
Step 3			
Workplace Rumination	0.10**	[0.03, 0.17]	
Age	0.03	[-0.04, 0.10]	
Gender	0.05	[-0.02, 0.12]	
Neuroticism	0.42**	[0.34, 0.49]	
			R^2 = .207** 95% CI[.15,.26]

Appendix 1.1.2 Moderation Analyses

Appendix 1.1.2: Table 17

Moderation Analyses for General Worry (N = 650).

	Dependent variable:			
	Burnout	Somatization	Health Behaviours	Sleep Quality
	(1)	(2)	(3)	(4)
Job Strain	20.633**	2.069	-11.262	-3.729
	(9.918)	(14.304)	(30.348)	(10.126)
General Worry	0.152	-0.072	-0.262	-0.124
	(0.195)	(0.282)	(0.597)	(0.199)
General Worry*Job Strain	0.052	0.332	0.325	0.275
	(0.203)	(0.293)	(0.621)	(0.207)
Observations	650	650	650	650
R^2	0.340	0.210	0.003	0.141
Adjusted R^2	0.337	0.206	-0.002	0.137
Residual Std. Error (df = 646)	3.947	5.692	12.076	4.029

F Statistic (df = 3; 646)	110.829***	57.106***	0.654	35.472***
Note:				*p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.2: Table 18

Moderation Analyses for General Rumination (N = 650).

	Dependent variable:			
	Burnout	Somatization	Health Behaviours	Sleep Quality
	(1)	(2)	(3)	(4)
Job Strain	22.488***	-0.545	10.215	2.874
	(7.298)	(10.011)	(22.155)	(7.476)
General Rumination	0.237	-0.227	0.121	-0.097
	(0.267)	(0.367)	(0.812)	(0.274)
Job Strain*General Rumination	0.023	0.656*	-0.156	0.272
	(0.278)	(0.381)	(0.844)	(0.285)
Observations	650	650	650	650
R^2	0.328	0.272	0.002	0.121
Adjusted R^2	0.325	0.269	-0.003	0.116
Residual Std. Error (df = 646)	3.981	5.461	12.085	4.078
F Statistic (df = 3; 646)	105.245***	80.621***	0.325	29.509***
Note:				*p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.2: Table 19

Moderation Analyses for Work-related Worry (N = 650).

	Dependent variable:			
	Burnout	Somatization	Health Behaviours	Sleep Quality
	(1)	(2)	(3)	(4)
Job Strain	12.872**	3.170	-9.558	-3.052
	(5.732)	(9.042)	(19.523)	(6.602)
Work-related Worry	0.429	-0.053	-1.095	-0.580
	(0.425)	(0.670)	(1.447)	(0.489)
Job Strain* Work-related Worry	0.258	0.749	1.184	0.903*
	(0.441)	(0.695)	(1.501)	(0.508)
Observations	650	650	650	650
R^2	0.467	0.236	0.002	0.117
Adjusted R^2	0.464	0.233	-0.002	0.113
Residual Std. Error (df = 646)	3.547	5.595	12.080	4.085
F Statistic (df = 3; 646)	188.486***	66.647***	0.502	28.652***

Note: *p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.2: Table 20

Moderation Analyses for Work-related Rumination (N = 650).

	Dependent variable:			
	Burnout	Somatization	Health Behaviours	Sleep Quality
	(1)	(2)	(3)	(4)
Job Strain	37.471***	5.516	-15.000	12.216
	(9.577)	(15.170)	(32.021)	(11.123)
Work-related Rumination	0.684***	0.009	-0.568	0.092
	(0.211)	(0.334)	(0.705)	(0.245)
Job Strain*Work-related Rumination	-0.350	0.314	0.511	-0.009
	(0.217)	(0.344)	(0.726)	(0.252)
Observations	650	650	650	650
R^2	0.447	0.202	0.004	0.070
Adjusted R^2	0.445	0.198	-0.001	0.066
Residual Std. Error (df = 646)	3.610	5.719	12.071	4.193
F Statistic (df = 3; 646)	174.373***	54.551***	0.820	16.234***

Note: *p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.3 Sensitivity Regression Analyses

Appendix 1.1.2: Table 21

General Worry and Rumination as independent predictors of health outcomes (N = 650).

	Dependent variable:		
	Burnout	Somatization	Sleep Quality
	(1)	(2)	(3)
General Worry	0.161***	0.136***	0.113***
	(0.021)	(0.028)	(0.020)
General Rumination	0.197***	0.340***	0.105***
	(0.028)	(0.037)	(0.027)
Observations	650	650	650
R^2	0.280	0.265	0.135
Adjusted R^2	0.277	0.263	0.132
Residual Std. Error (df = 647)	4.119	5.483	4.041
F Statistic (df = 2; 647)	125.592***	116.816***	50.526***

Note: *p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.2: Table 22

Work-related Worry and Rumination as independent predictors of health outcomes (N = 650).

	Dependent variable:		
	Burnout (1)	Somatization (2)	Sleep Quality (3)
Work-related Worry	0.563***	0.577***	0.355***
	(0.044)	(0.070)	(0.051)
Work-related Rumination	0.198***	0.153***	-0.015
	(0.025)	(0.039)	(0.029)
Observations	650	650	650
R^2	0.468	0.236	0.098
Adjusted R^2	0.466	0.233	0.095
Residual Std. Error (df = 647)	3.541	5.593	4.127
F Statistic (df = 2; 647)	284.224***	99.701***	34.997***
Note:			*p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.2: Table 23

All types of PC as a function of health outcomes (N = 650).

	Dependent variable:		
	Burnout (1)	Somatization (2)	Sleep Quality (3)
General Worry	0.072***	0.079***	0.088***
	(0.019)	(0.029)	(0.022)
General Rumination	0.052**	0.248***	0.081***
	(0.025)	(0.038)	(0.029)
Workplace Worry	0.429***	0.283***	0.178***
	(0.050)	(0.076)	(0.058)
Workplace Rumination	0.190***	0.115***	-0.027
	(0.024)	(0.037)	(0.028)
Observations	650	650	650
R^2	0.491	0.314	0.148
Adjusted R^2	0.488	0.310	0.143
Residual Std. Error (df = 645)	3.468	5.305	4.016

F Statistic (df = 4; 645)	155.492***	73.913***	28.091***

Note: *p<0.1; **p<0.05; ***p<0.01

Appendix 1.1.4 Brooding Analyses

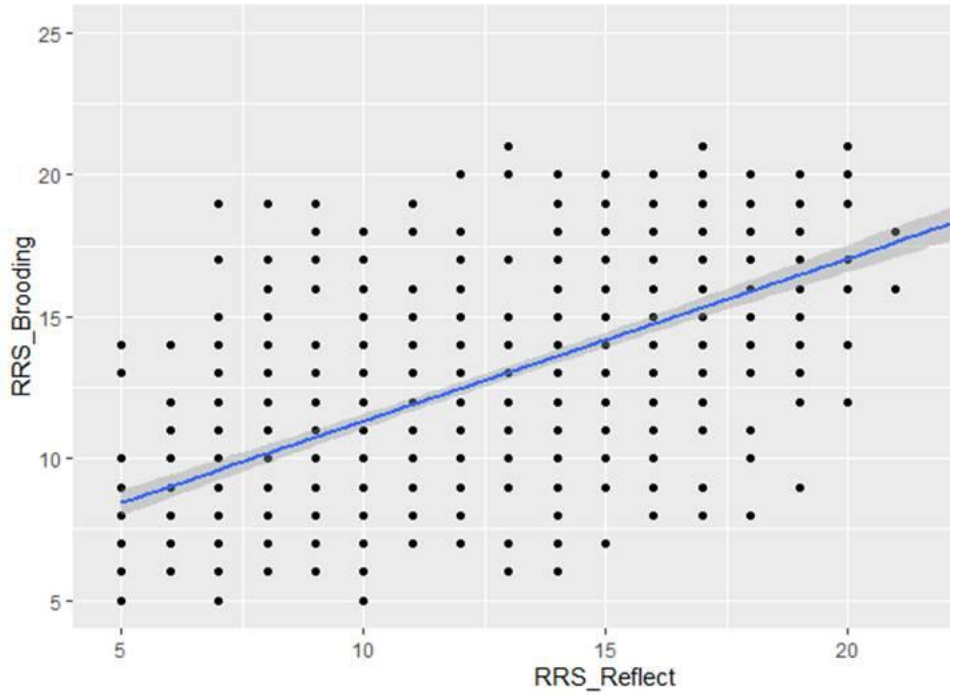
As a precaution, we re-analysed all primary analysis based exclusively on the Brooding subscale exclusively. The regression tables are shown on the page below (Tables 24 – 27). The results are virtually identical to those using the 'overall' rumination measure.

Hypothesis 1

Correlations

The correlation between Brooding and Reflection was *r* = .63.

Relationship between Brooding and Reflection subscales.



Appendix 1.1.4: Table 24

Hierarchical regression: Brooding on Burnout (N = 650).

Predictor	*beta*	95% CI	Fit

		[LL, UL]	
Step 1			
Brooding	0.44**	[0.37, 0.51]	
			R^2 = .196**
			95% CI[.15,.25]
Step 2			
Brooding	0.45**	[0.37, 0.52]	
Age	0.02	[-0.05, 0.10]	
Gender	0.08	[0.01, 0.15]	
			R^2 = .204**
			95% CI[.15,.25]
Step 3			
Brooding	0.31**	[0.23, 0.39]	
Age	0.03	[-0.04, 0.10]	
Gender	0.03	[-0.04, 0.10]	
Neuroticism	0.27**	[0.19, 0.35]	
			R^2 = .256**
			95% CI[.20,.31]

Appendix 1.1.4: Table 25

Hierarchical regression: Brooding on Somatization (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Brooding	0.47**	[0.40, 0.53]	
			R^2 = .218**
			95% CI[.16,.27]
Step 2			
Brooding	0.45**	[0.37, 0.52]	
Age	-0.06	[-0.14, 0.01]	
Gender	0.07	[-0.00, 0.14]	
			R^2 = .224**
			95% CI[.17,.27]
Step 3			
Brooding	0.35**	[0.26, 0.43]	
Age	-0.06	[-0.13, 0.02]	
Gender	0.04	[-0.03, 0.11]	
Neuroticism	0.20**	[0.12, 0.28]	
			R^2 = .251**
			95% CI[.19,.30]

Appendix 1.1.4: Table 26

Hierarchical regression: Brooding on Health Behaviours (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Brooding	0.02	[-0.06, 0.10]	
			R^2 = .000

			95% CI[.00,.01]
Step 2			
Brooding	0.03	[-0.05, 0.11]	
Age	0.01	[-0.07, 0.09]	
Gender	-0.18**	[-0.26, -0.10]	
			R^2 = .031**
			95% CI[.01,.06]
Step 3			
Brooding	-0.13**	[-0.22, -0.04]	
Age	0.02	[-0.06, 0.10]	
Gender	-0.23**	[-0.31, -0.15]	
Neuroticism	0.32**	[0.23, 0.40]	
			R^2 = .101**
			95% CI[.06,.14]

Appendix 1.1.4: Table 27

Hierarchical regression: Brooding on Sleep Quality (N = 650).

Predictor	*beta*	95% CI [LL, UL]	Fit
Step 1			
Brooding	0.30**	[0.23, 0.38]	
			R^2 = .093**
			95% CI[.05,.14]
Step 2			
Brooding	0.31**	[0.23, 0.39]	
Age	0.04	[-0.04, 0.12]	
Gender	0.11**	[0.04, 0.19]	
			R^2 = .109**
			95% CI[.07,.15]
Step 3			
Brooding	0.12**	[0.04, 0.21]	
Age	0.05	[-0.02, 0.13]	
Gender	0.05	[-0.02, 0.12]	
Neuroticism	0.37**	[0.29, 0.46]	
			R^2 = .207**
			95% CI[.15,.26]

Hypothesis 2

Appendix 1.1.4: Table 28

Moderation results: Brooding and Job Strain on Health Outcomes (N = 650).

	Dependent variable

	Burnout	Somatization	Health Behaviours	Sleep Quality
	(1)	(2)	(3)	(4)
Job Strain	24.109***	7.720	5.287	4.185
	(6.881)	(9.497)	(20.791)	(7.010)
Brooding	0.492	0.009	0.040	-0.133
	(0.493)	(0.680)	(1.488)	(0.502)
Job Strain*Brooding	-0.021	0.739	-0.002	0.459
	(0.510)	(0.704)	(1.541)	(0.519)
Constant	-7.568	-2.811	43.199**	2.058
	(6.582)	(9.083)	(19.885)	(6.705)
Observations	650	650	650	650
R^2	0.322	0.256	0.001	0.122
Adjusted R^2	0.319	0.253	-0.003	0.118
Residual Std. Error (df = 646)	4.000	5.521	12.086	4.075
F Statistic (df = 3; 646)	102.109***	74.249***	0.285	29.811***
Note:	*p<0.1; **p<0.05; ***p<0.01			

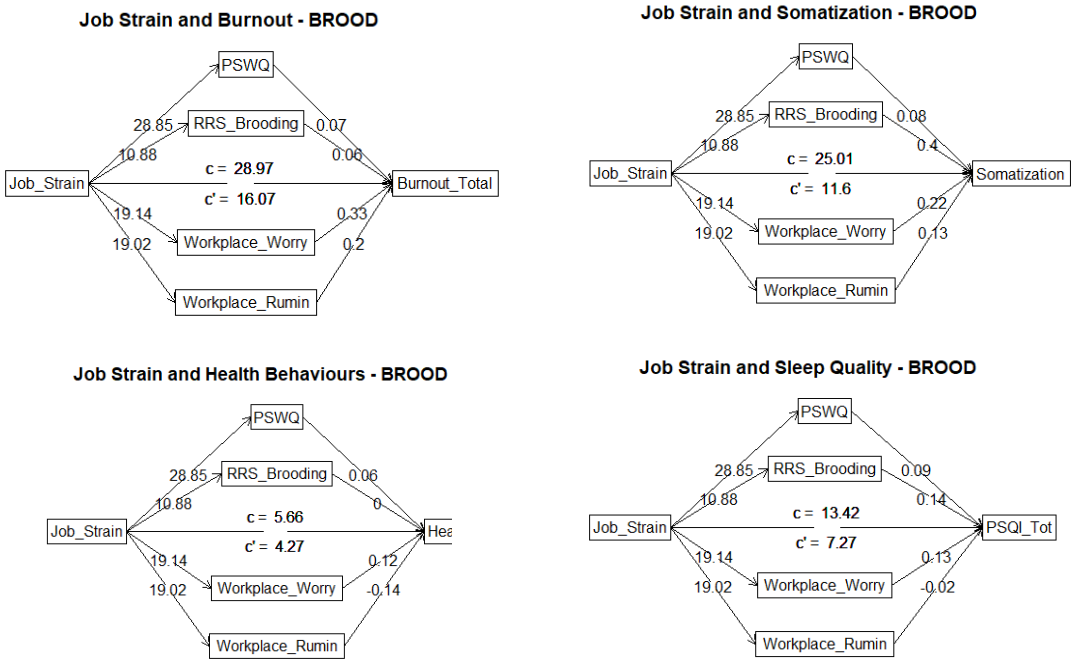
Hypothesis 3

Mediation Analyses:

The multi-meditor model was re-tested but with the terms for brooding, rather than the ‘overall’ rumination measure. Again, this did not meaningfully impact the results. The only thing to note from this is the betas are almost halved for the path from job strain to what was the total rumination measure; imply more variance is actually explained by the ‘overall’ measure and further justifying its use in the main manuscript

Appendix 1.1.4: Figure 1

Multiple Mediation Model for PC, Job Strain and Health Outcomes (with Brooding) (N = 650).



Note: All paths are significant expect those running from all types of PC to health behaviours, as well as the path from work-related rumination to sleep, and general rumination to burnout. Significance is NOT denoted via asterisk's in this figure. Numbers reflect the unstandardized regression coefficients; C = the total effect of x on y; C' = the direct effect of x on y. PSWQ (PennStateWorryQuestionnaire) = General Worry; RRS_Brooding (RuminationResponseStyle) = Brooding; PSQI_Tot (PittsburghSleepQualityIndex) = Sleep Quality.

Appendix 1.2 – Chapter 2 Supplementary Material B

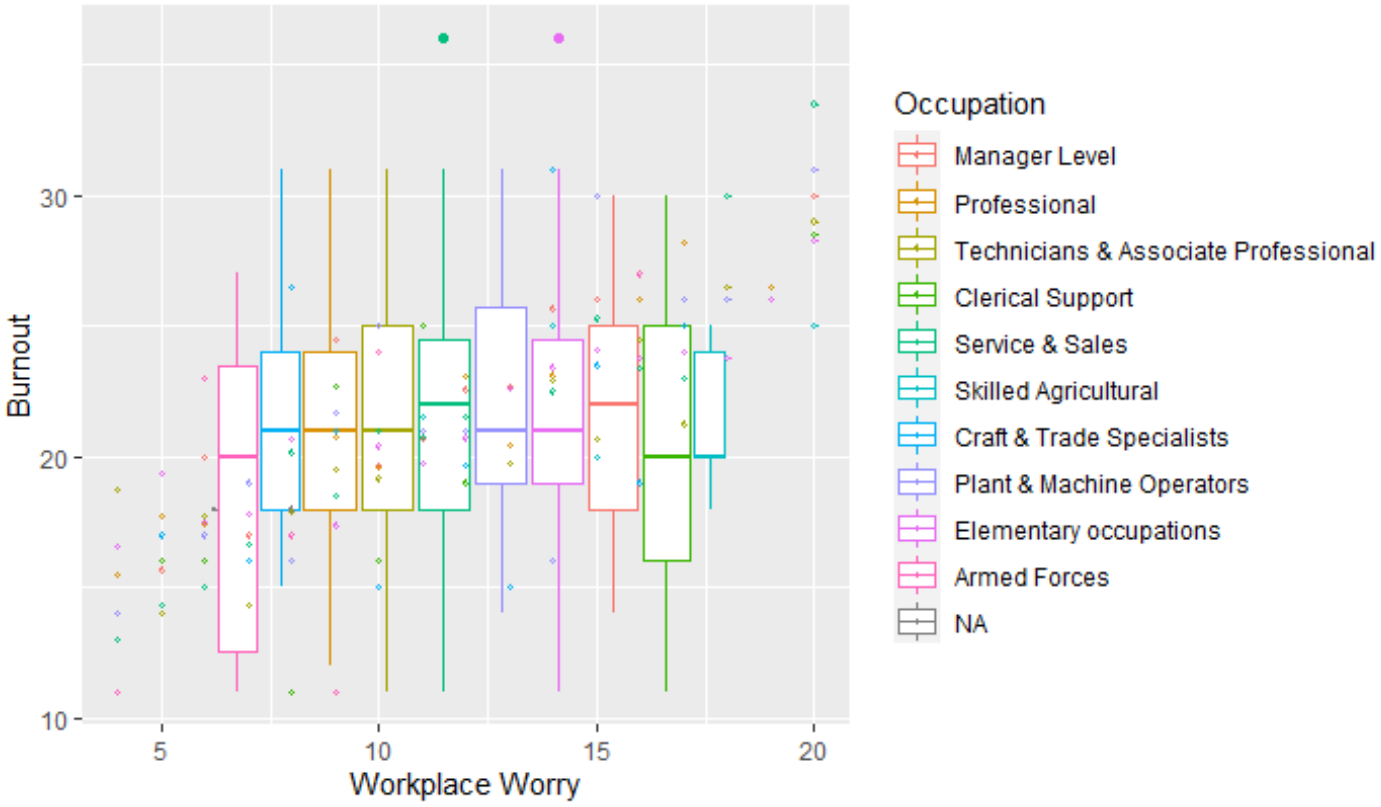
There are 2 sections to this supplementary appendix.

The first section shows various Figures (1-16) concerning the different participant demographics and how they were related to the different measures of PC and health. Few differences emerged for the relationship between occupational group on levels of PC and burnout, somatization, and sleep; suggesting that PC has an adverse effect on health regardless of occupational group; the Figures below reflect these group-wise comparisons.

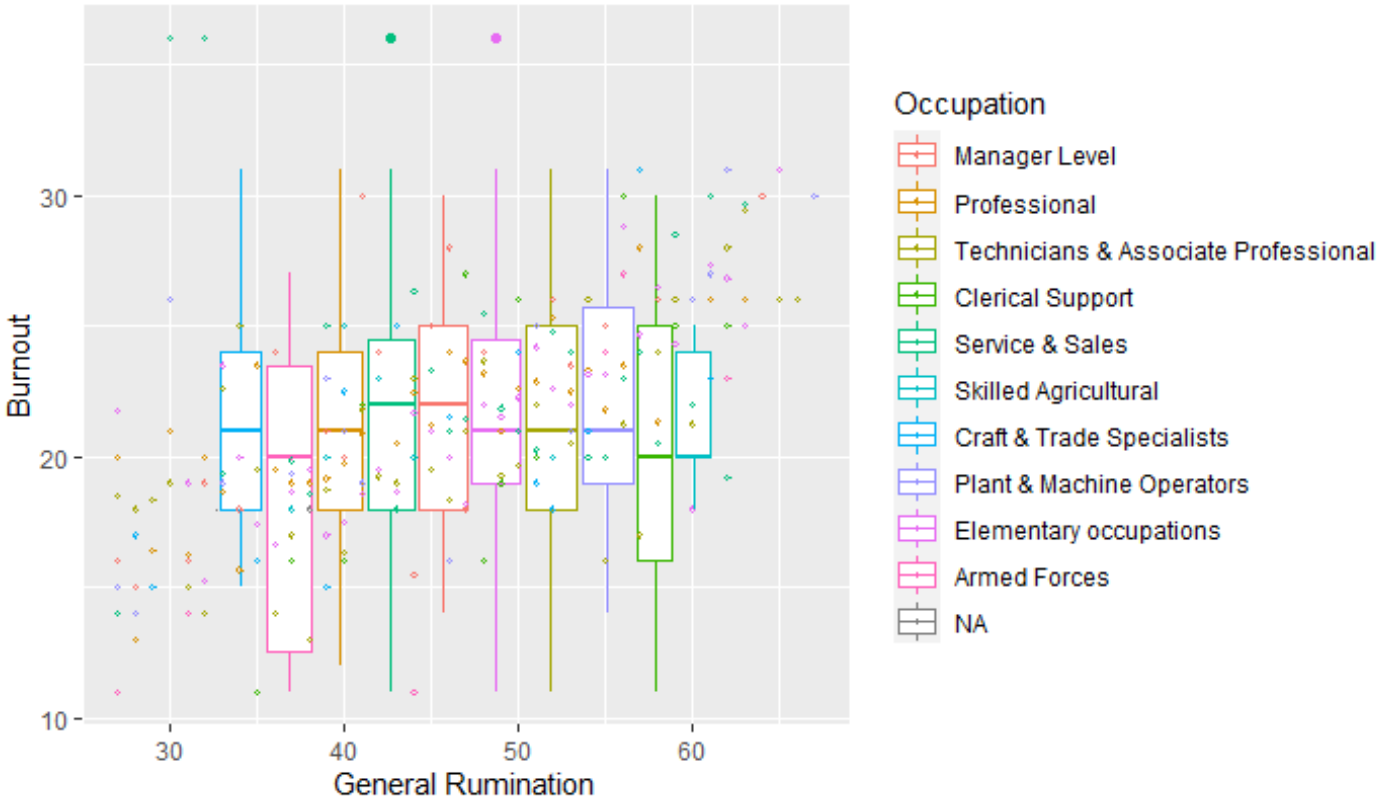
The second section contains the missing-case analysis used in conjunction with Littles test for missing data.

Appendix 1.2.1 Sub-group Analyses

OSM Figure 1. Workplace Worry and Burnout by Occupation Group

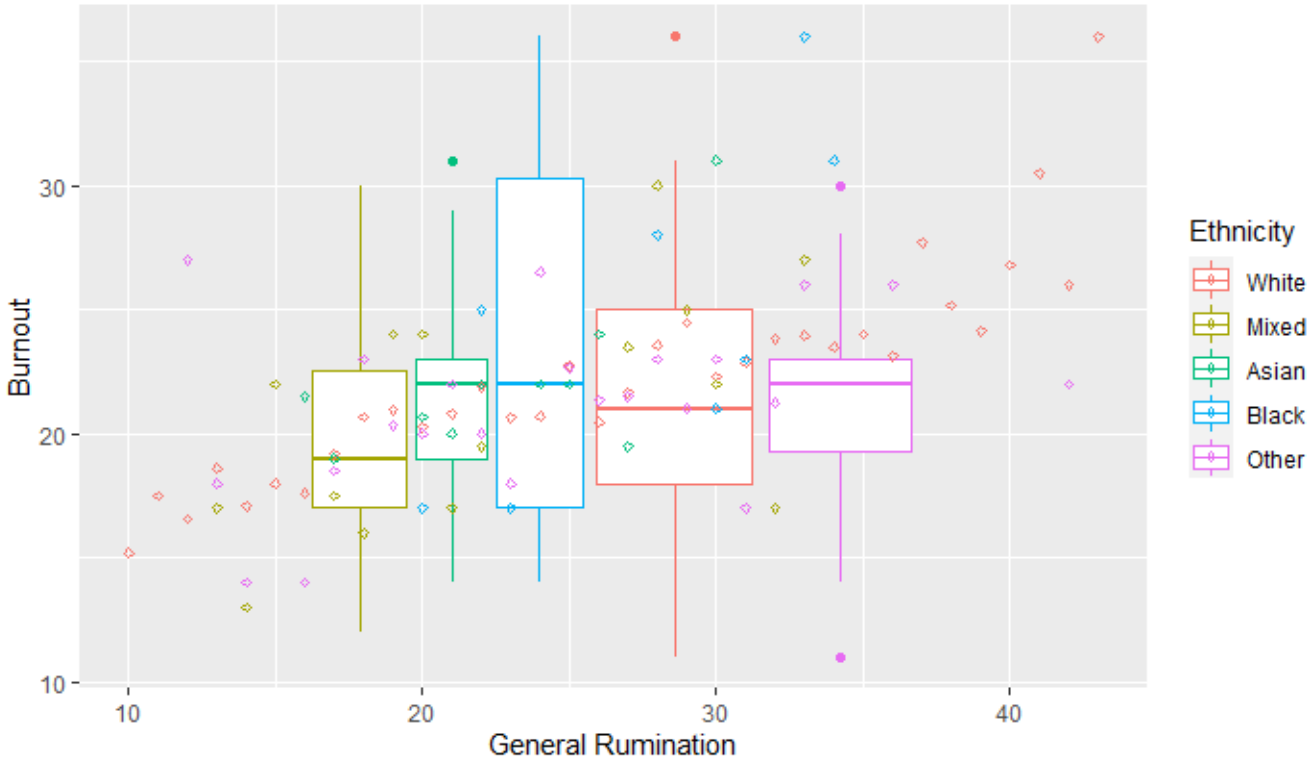


OSM Figure 2. Workplace Rumination and Burnout by Occupation Group



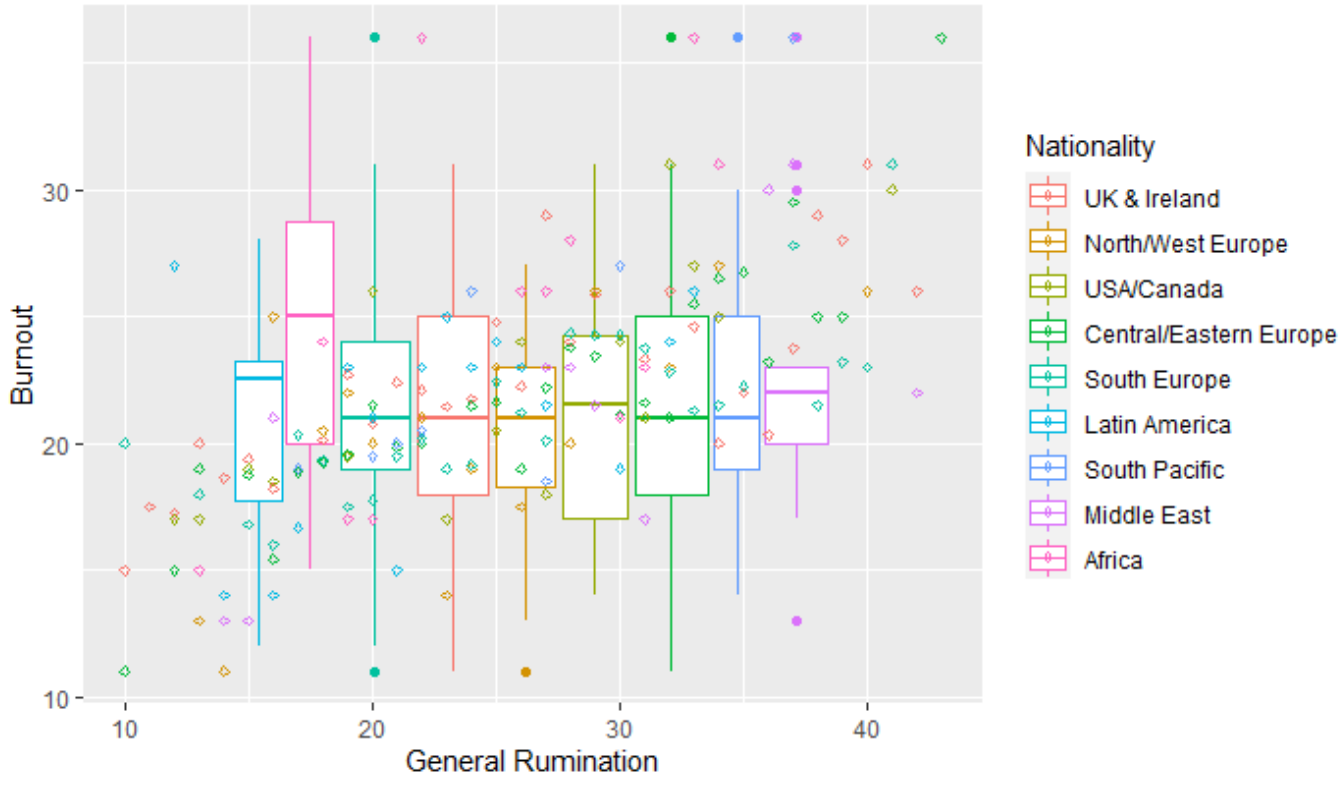
OSM Figure 3. Worry and Burnout by Ethnic Group

OSM Figure 4. Rumination and Burnout by Ethnic Group

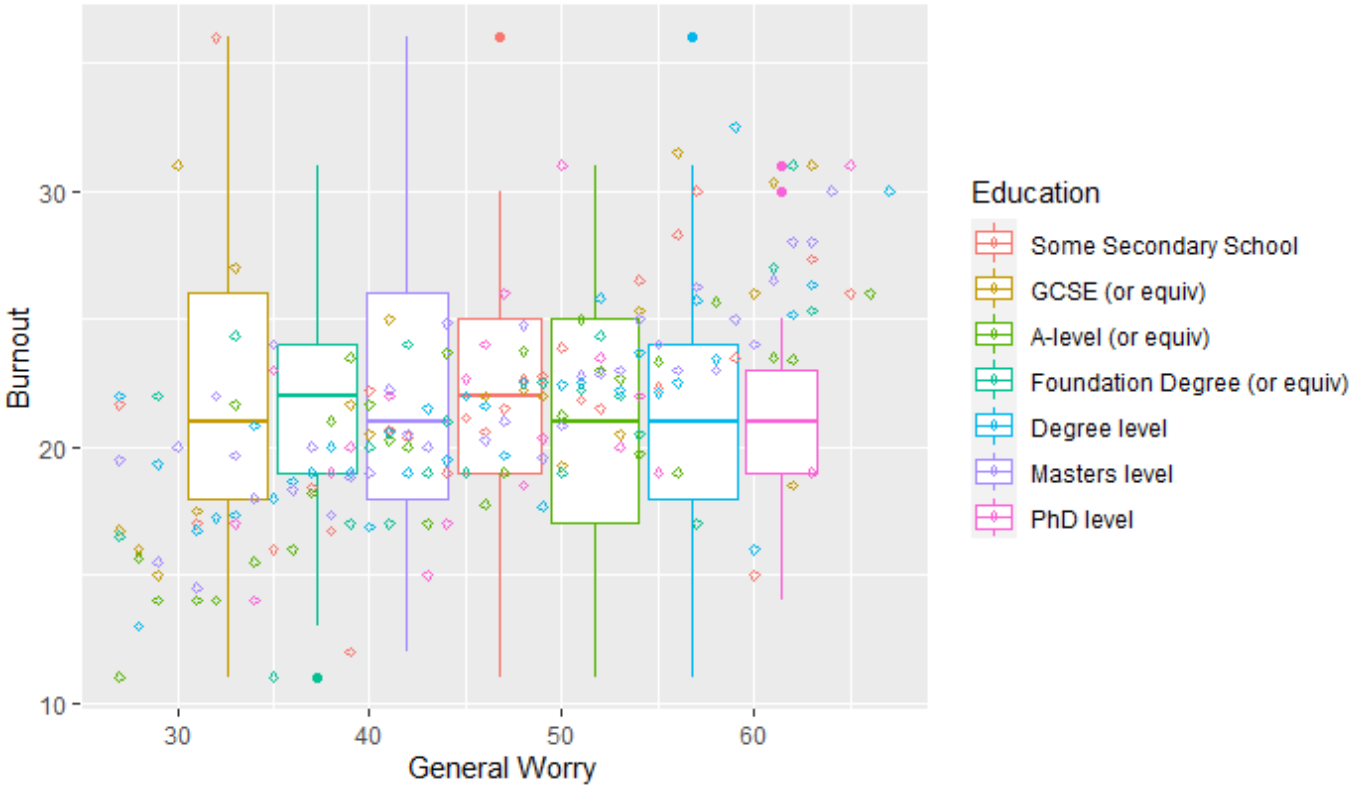


OSM Figure 5. Worry and Burnout by Nationality

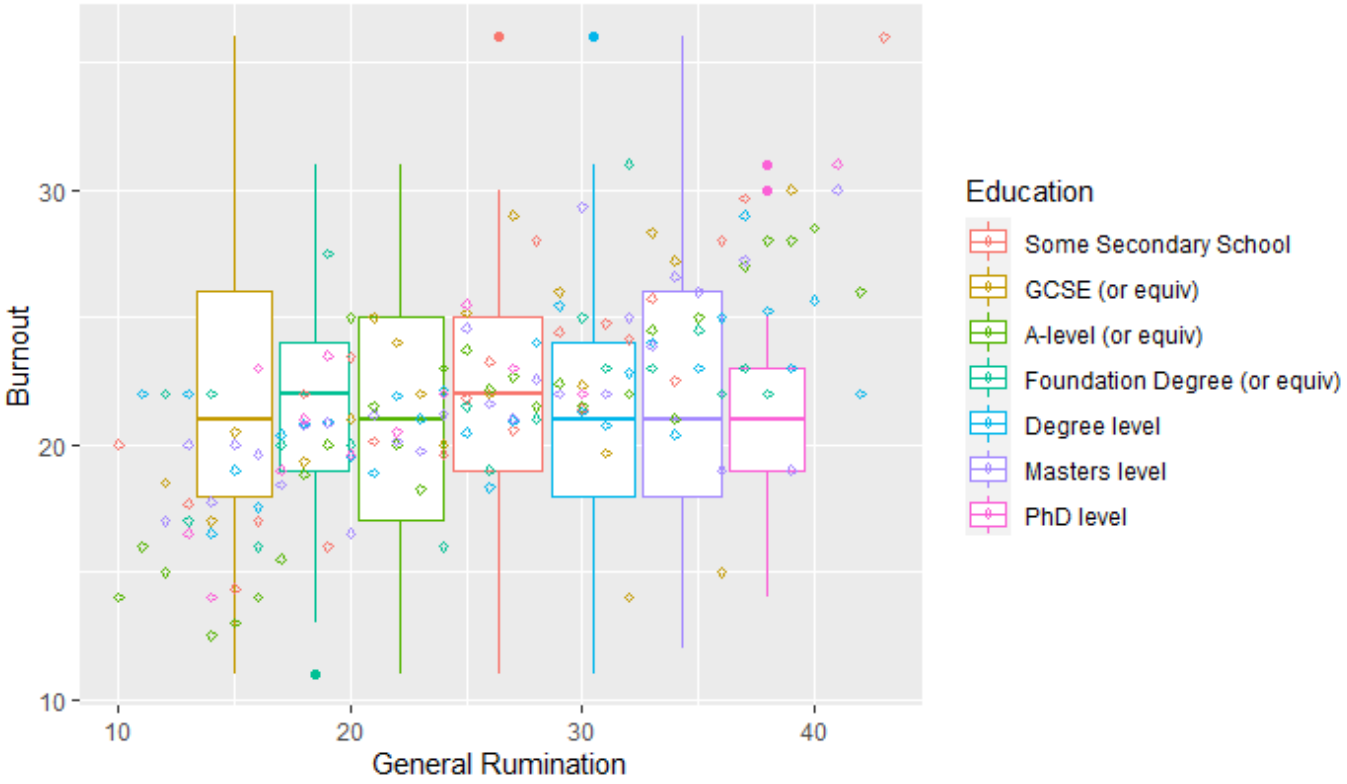
OSM Figure 6. Rumination and Burnout by Nationality



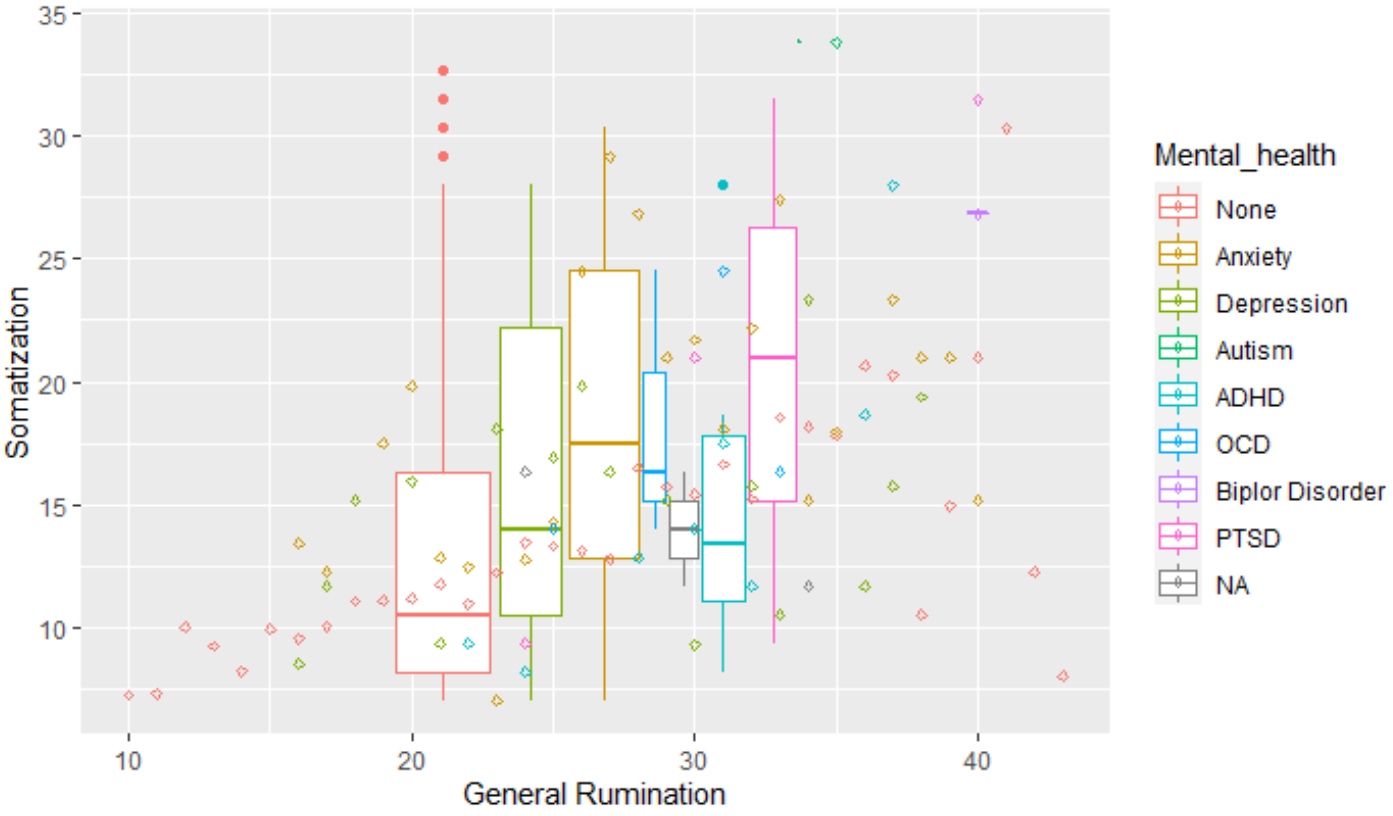
OSM Figure 7. Worry and Burnout by Educational Attainment



OSM Figure 8. Rumination and Burnout by Educational Attainment

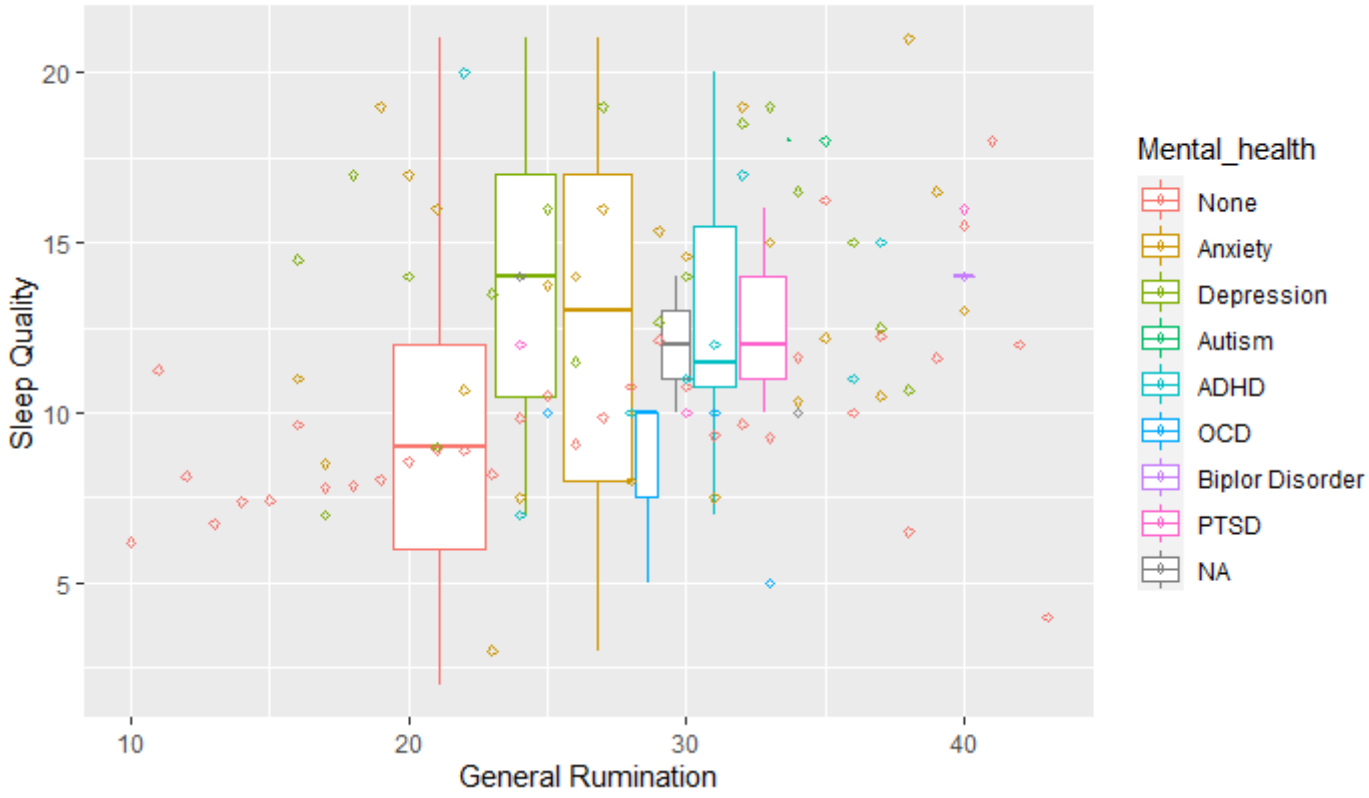


OSM Figure 9. Rumination and Somatization by Mental Health



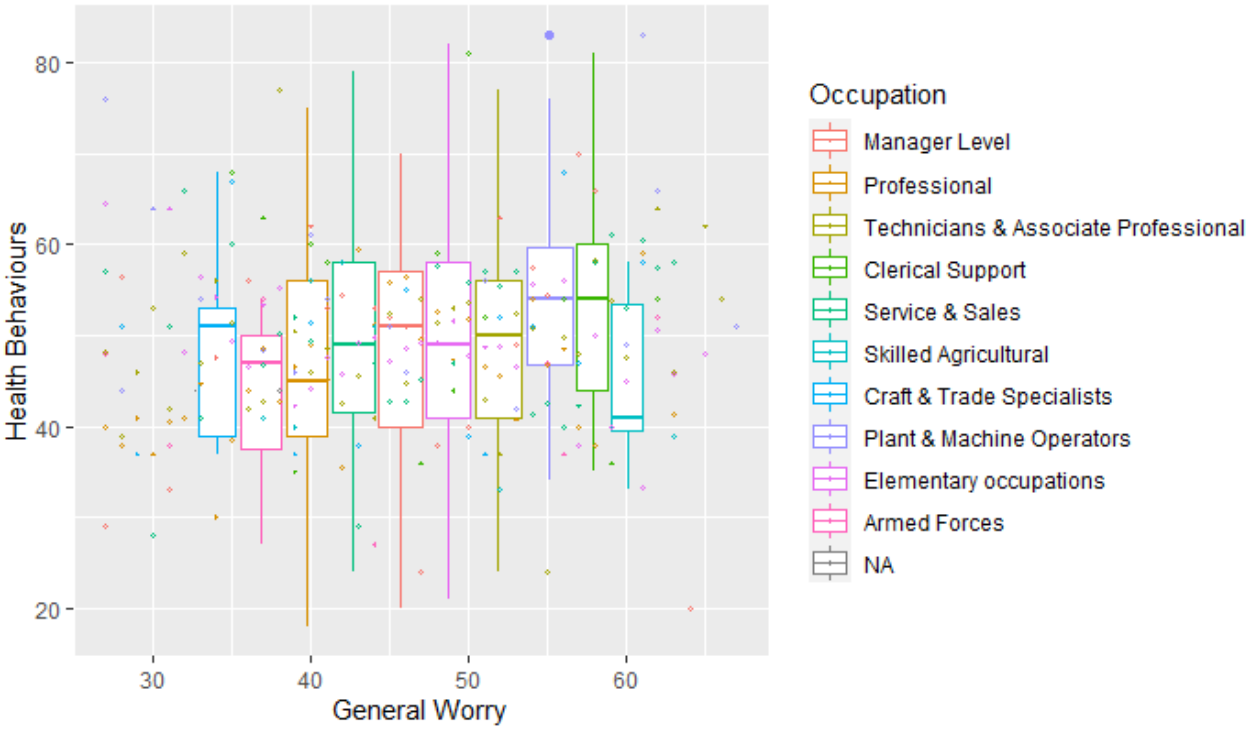
OSM Figure 10. Worry and Somatization by Mental Health

OSM Figure 11. Rumination and Sleep Quality by Mental Health

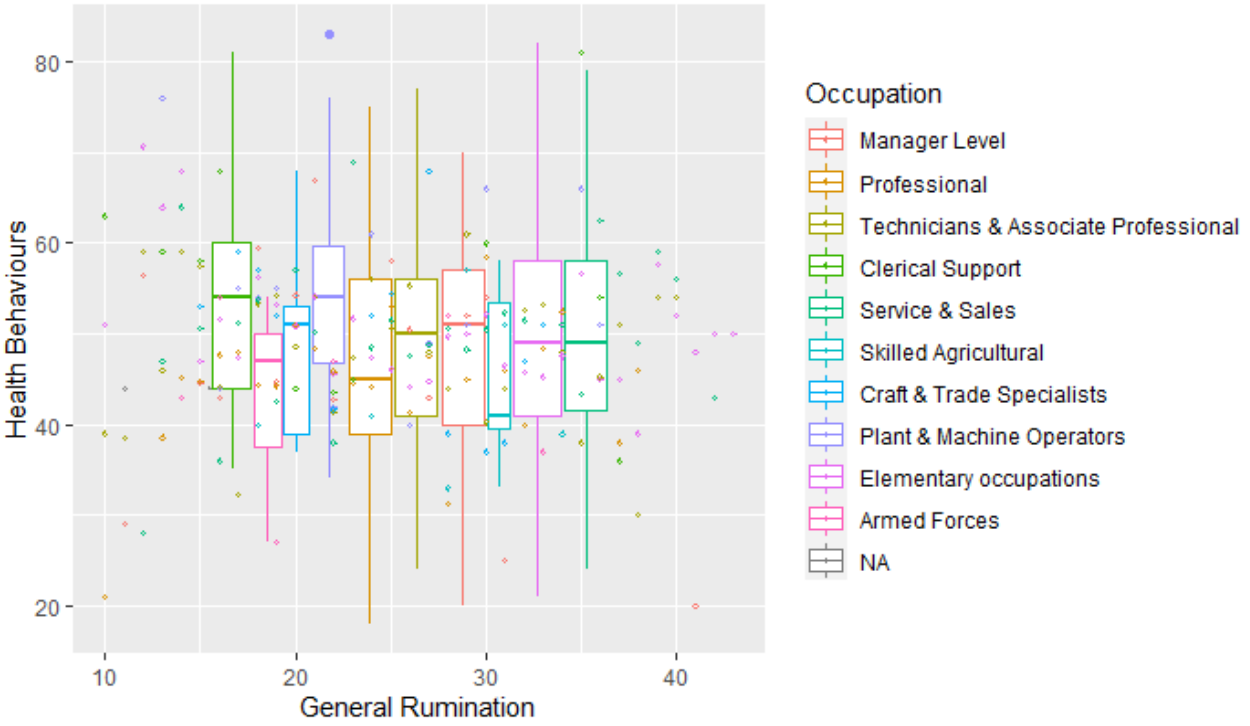


OSM Figure 12. Worry and Sleep Quality by Mental Health

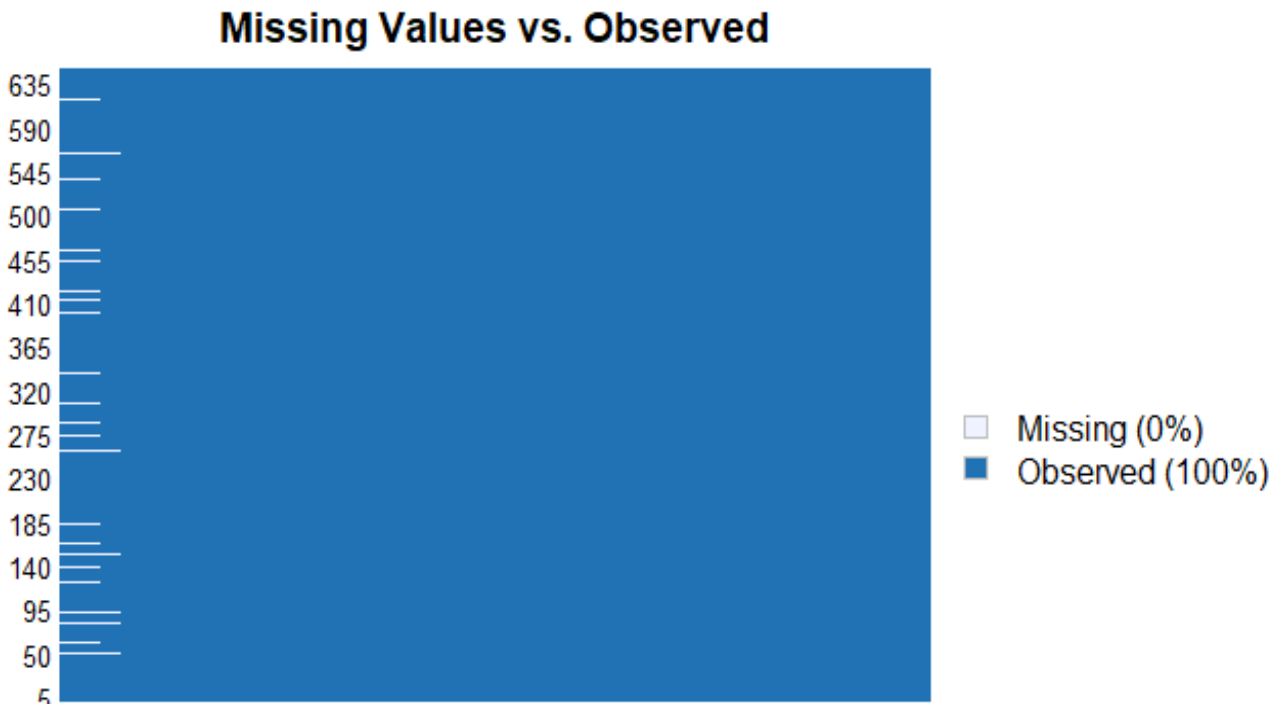
OSM Figure 13. Worry and Health behaviours by Occupation Group



OSM Figure 13. Rumination and Health behaviours by Occupation Group



Appendix 2.2.2 Missing Data Figure



Appendix 2.1 – Chapter 3 Supplementary Material A

Appendix 2.1.1 Search Terms

1 Rumination.mp.

2 (Ruminat* and (thought* or thinking)).mp.

3 (perseverative and (thought* or thinking or cognition*)).mp.

4 (Repetitive and (thought* or thinking)).mp.

5 (Intrusive and (thought* or thinking)).mp

6 worr*.mp.

7 (Stress* and (thought* or thinking)).mp

8 (Self referential and (thought* or thinking)).mp.

9 brooding.mp.

10 reflection.mp.

11 (obsessive and (thought* or thinking)).mp

12 unconscious stress*.mp.

13 implicit stress*.mp.

14 anticipat* stress*.mp.

15 cognitive intrusion*.mp.

16 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 11 or 12 or 13 or 14 or 15

17 intervention*.tw.

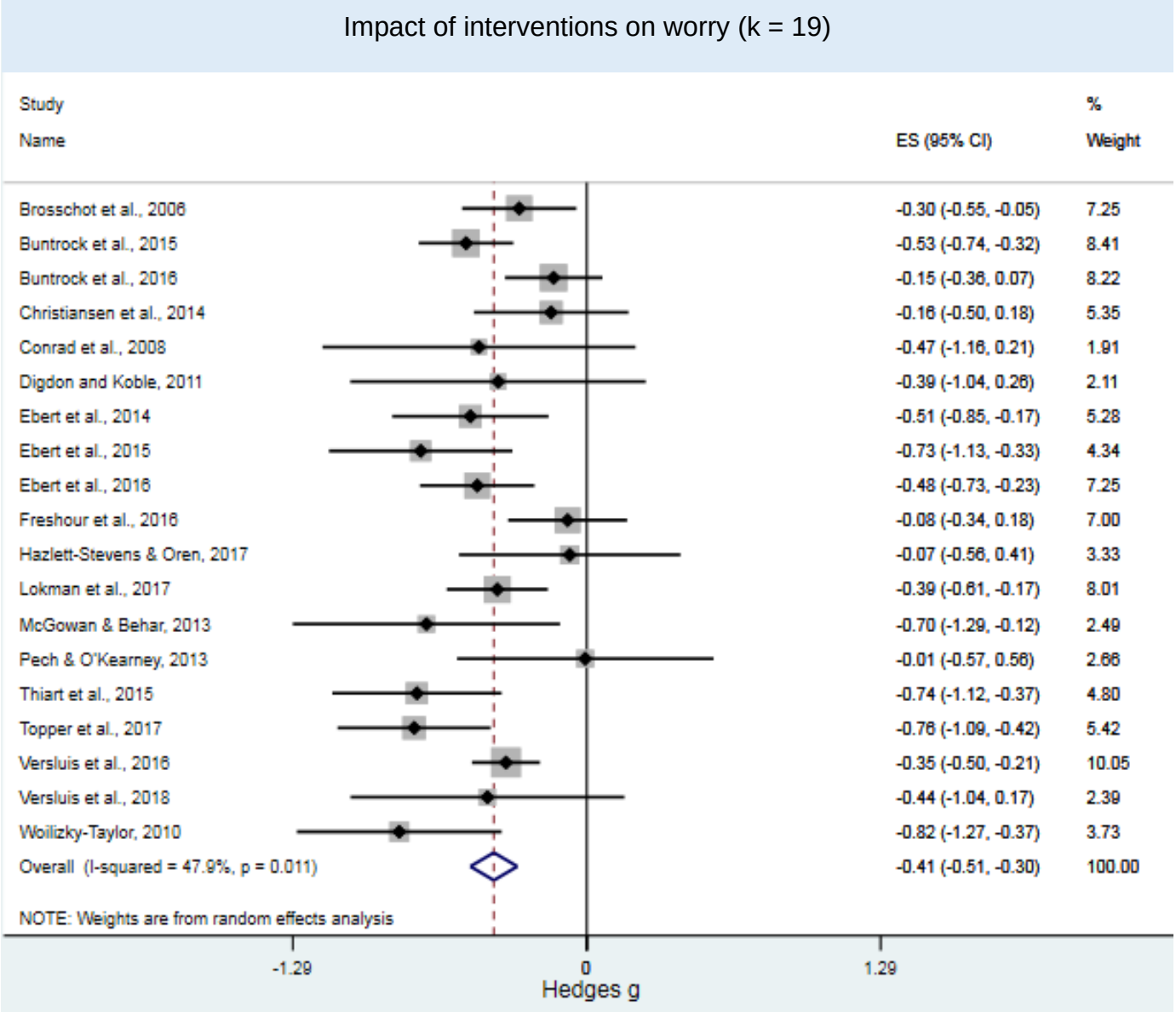
18 random*.tw.

19 17 or 18

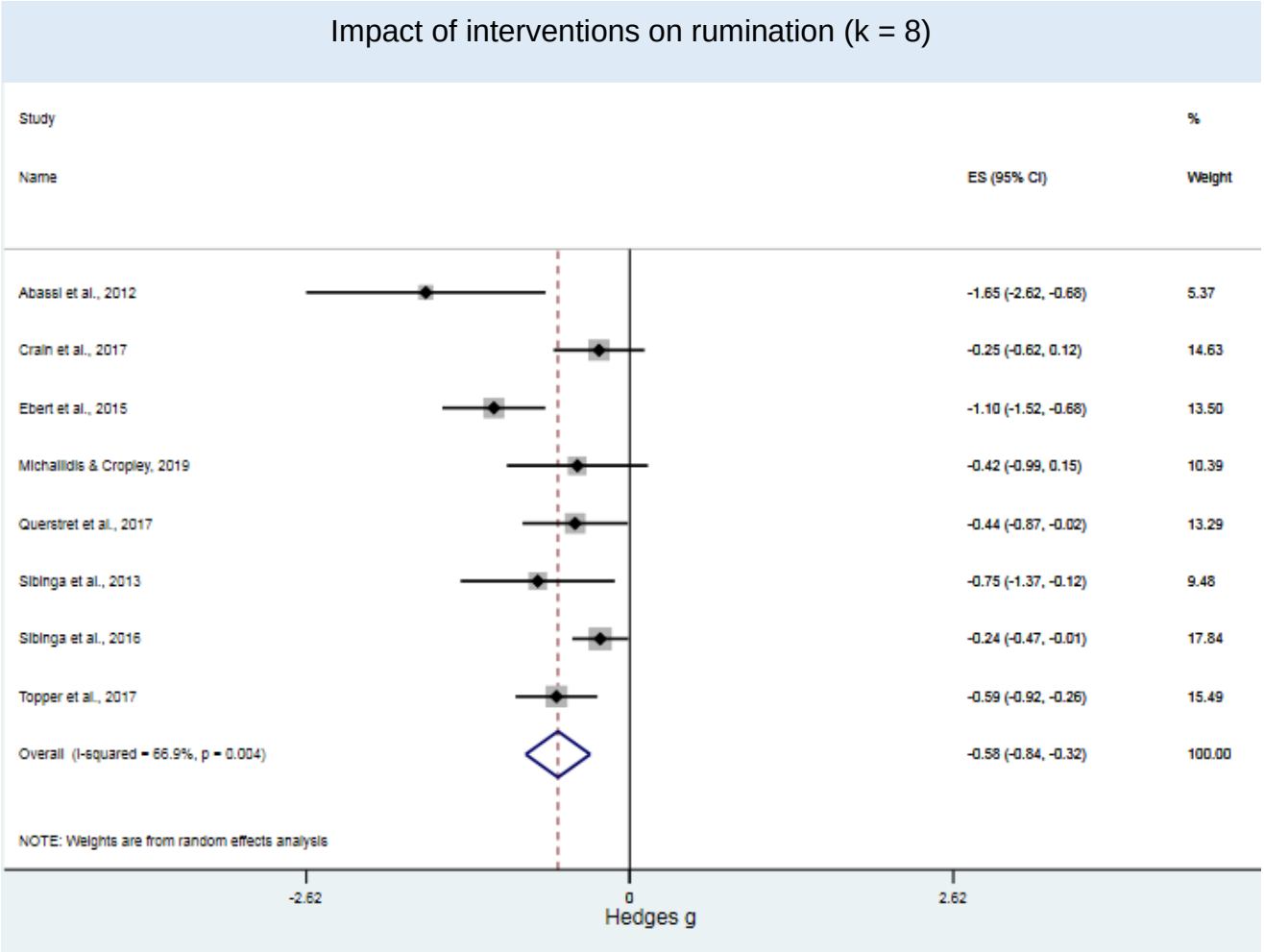
20 16 and 19

21 limit 20 to (English language and human)

Appendix 2.1.2 Worry forest plot



Appendix 2.1.3 Rumination forest plot



Appendix 2.1.4 Sub-group analyses for PC and health outcomes

Table 2. Sub-group analyses between intervention types and PC and health outcome variables.

Intervention type◎	Outcome	Test Statistic		
		Hedges g	*Z*	*p*
Pain management	PC (*k* = 2)	-.807	1.06	.290
	Worry (*k* = 0)	-	-	-
	Rumination (*k* = 1)	-1.65	3.34	.001**
	HO (*k* = 2)	0.213	0.87	.382
	HB (*k* = 0)	-	-	-
	PHO (*k* = 2)	0.283	0.87	0.382
	Sleep (*k* = 0)	-	-	-
PC action plans	PC (*k* = 9)	-0.396	4.89	.001***
	Worry (*k* = 5)	-0.360	5.86	.001***
	Rumination (*k* = 0)	-	-	-
	HO (*k* = 9)	0.422	3.41	.001**
	HB (*k* = 4)	0.635	3.59	.001***
	PHO (*k* = 6)	0.203	2.01	.044*
	Sleep (*k* = 4)	0.440	3.84	.001**
Stress management	PC (*k* = 4)	-0.264	2.78	.005**
	Worry (*k* = 3)	-0.242	1.74	.081
	Rumination (*k* = 0)	-	-	-
	HO (*k* = 4)	0.190	3.56	.001**
	HB (*k* = 3)	0.184	2.31	.021*
	PHO (*k* = 2)	0.165	1.46	.145
	Sleep (*k* = 2)	0.163	1.78	.075
Mindfulness/relaxation	PC (*k* = 7)	-0.382	3.94	.001***
	Worry (*k* = 3)	-0.462	1.89	.059
	Rumination (*k* = 4)	-0.310	3.59	.001***
	HO (*k* = 7)	0.246	4.33	.001***
	HB (*k* = 3)	0.217	2.34	.019*
	PHO (*k* = 5)	0.252	3.02	.003**
	Sleep (*k* = 3)	0.214	2.60	.009**
Psychological detachment	PC (*k* = 2)	-0.673	3.15	.002**
	Worry (*k* = 2)	-0.552	4.90	.001***
	Rumination (*k* = 1)	-1.100	5.16	.001***
	HO (*k* = 2)	0.617	2.81	.005**
	HB (*k* = 2)	0.623	2.73	.006**
	PHO (*k* = 1)	0.429	4.47	.001***
	Sleep (*k* = 2)	0.623	2.73	.006**
CBT/ACT	PC (*k* = 10)	-0.450	5.39	.001***
	Worry (*k* = 6)	-0.432	4.09	.001***
	Rumination (*k* = 1)	-0.594	3.51	.001***
	HO (*k* = 10)	0.216	6.31	.001***
	HB (*k* = 7)	0.202	4.16	.001***
	PHO (*k* = 4)	0.245	3.29	.001**

	Sleep (*k* = 5)	0.201	3.02	.003**
Expressive writing	PC (*k* = 2)	-0.361	1.91	.056
	Worry (*k* = 0)	-	-	-
	Rumination (*k* = 1)	-0.424	4.37	.145
	HO (*k* = 2)	0.416	2.20	.028*
	HB (*k* = 1)	0.390	1.34	.179
	PHO (*k* = 1)	0.435	1.74	.082
	Sleep (*k* = 1)	0.390	1.34	.179

Note: **p* =.05; ***p*<.05; ****p*<.001; ◎ = the categorical predictors for these analyses are set as 1 (type present) and 0 (type not present);CBT/ACT = cognitive behavioural/acceptance and commitment style therapies; PC = perseverative cognition; HO = health outcomes (overall); HB = health behaviours; PHO = physical health outcomes; *Hedges g* statistic = effect size estimate; *Z* statistic = the distribution under the null hypothesis that can be approximated by a normal distribution (accompanied by a significance test, p).

Appendix 2.1.5 Associations between intervention types and study outcomes

Table 3. Associations between intervention types and study outcome effect sizes.

Intervention type◎	Outcome	Test Statistic			Heterogeneity
		B	*SE*	*p*	I^2
Pain management (k = 2)	PC (*k* = 36)	.084	.22	.71	58.61
	Worry (*k* = 19)	-	-	-	-
	Rumination (*k* = 8)	-1.14	.56	.09	60.98
	HO (*k* = 36)	-.145	.15	.35	45.58
	HB (*k* = 21)	-	-	-	-
	PHO (*k* = 21)	-.125	.12	.32	18.12
	Sleep (*k* = 17)	-	-	-	-
PC action plans (k = 9)	PC (*k* = 36)	-.004	.12	.97	60.81
	Worry (*k* = 19)	.029	.13	.83	50.38
	Rumination (*k* = 8)	-	-	-	-
	HO (*k* = 36)	.123	.09	.19	48.50
	HB (*k* = 21)	.366	.14	.02**	39.12
	PHO (*k* = 21)	-.047	.11	.66	26.78
	Sleep (*k* = 17)	.188	.15	.26	31.60
Stress management (k = 4)	PC (*k* = 36)	.171	.15	.25	59.27
	Worry (*k* = 19)	.199	.15	.19	44.29
	Rumination (*k* = 8)	-	-	-	-
	HO (*k* = 36)	-.135	.11	.19	48.44
	HB (*k* = 21)	-.156	.15	.32	52.05
	PHO (*k* = 21)	-.082	.12	.51	28.17
	Sleep (*k* = 17)	-.144	.14	.31	34.30
Mindfulness/relaxation (k = 7)	PC (*k* = 36)	.027	.13	.84	60.86
	Worry (*k* = 19)	-.066	.19	.74	50.28
	Rumination (*k* = 8)	.456	.22	.09	44.42
	HO (*k* = 36)	-.034	.10	.73	49.59
	HB (*k* = 21)	-.132	.17	.45	53.56
	PHO (*k* = 21)	.012	.11	.86	27.90
	Sleep (*k* = 17)	-.094	.13	.47	37.14
Psychological detachment (k = 2)	PC (*k* = 36)	-.263	.19	.18	58.63
	Worry (*k* = 19)	-.188	.17	.29	47.01
	Rumination (*k* = 8)	-.652	.33	.09	47.92
	HO (*k* = 36)	.304	.18	.01**	36.63
	HB (*k* = 21)	.332	.16	.05*	42.19
	PHO (*k* = 21)	.225	.13	.09	9.83
	Sleep (*k* = 17)	.346	.11	.01***	3.21
CBT/ACT (k = 10)	PC (*k* = 36)	-.051	.11	.63	58.35
	Worry (*k* = 19)	-.031	.11	.79	49.97
	Rumination (*k* = 8)	-.0002	.44	.99	70.81
	HO (*k* = 36)	-.071	.08	.38	48.78
	HB (*k* = 21)	-.195	.11	.09	47.57

	PHO (*k* = 21)	.029	.11	.78	28.05
	Sleep (*k* = 17)	-.134	.10	.19	31.73
Expressive writing (*k* = 2)	PC (*k* = 36)	-.056	.26	.83	60.98
	Worry (*k* = 19)	-	-	-	-
	Rumination (*k* = 8)	.19	.50	.71	71.57
	HO (*k* = 36)	.14	.23	.53	49.1
	HB (*k* = 21)	.075	.37	.84	54.06
	PHO (*k* = 21)	.208	.27	.45	26.32
	Sleep (*k* = 17)	.108	.33	.74	38.08

Note: **p* =.05; ***p*<.05; ****p*<.001; ◎ = the categorical predictors for these analyses are set as 1 (type present) and 0 (type not present);CBT/ACT = cognitive behavioural/acceptance and commitment style therapies; PC = perseverative cognition; HO = health outcomes (overall); HB = health behaviours; PHO = physical health outcomes; *B* statistic = standardized beta (accompanied by standard error, *S.E* and significance test, p); I^2 statistic = percentage of residual variation due to heterogeneity.

Appendix 2.1.6 Association between effect sizes and study characteristics

Outcome	Predictor©	Test Statistic	
		B	*S.E.*
PC	Age	-.003	.003
(*k* = 36)	Sleep disturbance	-.26	.09
	GAD participants	-.21	.18
	% of participants female	-.0002	.002
	Adult's vs children	-.005	.13
	Measure time-point	-.0008	.001
	Number of sessions	-.15	.01
	ITT analyses	-.19	.02
	Mode of delivery		
	Health-care professional	-.39*	.18
	Self-administered	-.02	.10
	Self-administered with support	.08	.11
	Trained facilitator	.10	.12
	Intervention setting		
	Medical	.06	.12
	Educational	-.04	.12
	Academic	-.01	.10
	Hosted online vs In person	-.06	.10
	Active vs non-active control	.05	.10
	Individual vs group delivery	-.002	.11
	Student sample	-.14	.12
	Attrition	.002	.003
Worry	Age	.002	.004
(*k* = 19)	Sleep disturbance	-.23	.16
	GAD participants	-.14	.11
	% of participants female	-.01	.06
	Adult's vs children	.10	.17
	Measure time-point	.0007	.002
	Number of sessions	-.0002	.15
	ITT analyses	-.09	.11
	Mode of delivery		
	Health-care professional	—	—
	Self-administered	-.02	.10
	Self-administered with support	.15	.12
	Trained facilitator	.02	.16
	Intervention setting		
	Medical	.27	.18
	Educational	-.09	.13
	Academic	-.02	.12
	Hosted online vs In person	-.14	.12
	Active vs non-active control	-.16	.11
	Individual vs group delivery	.07	.12
	Student sample	-.35*	.14
	Attrition	.07	.12

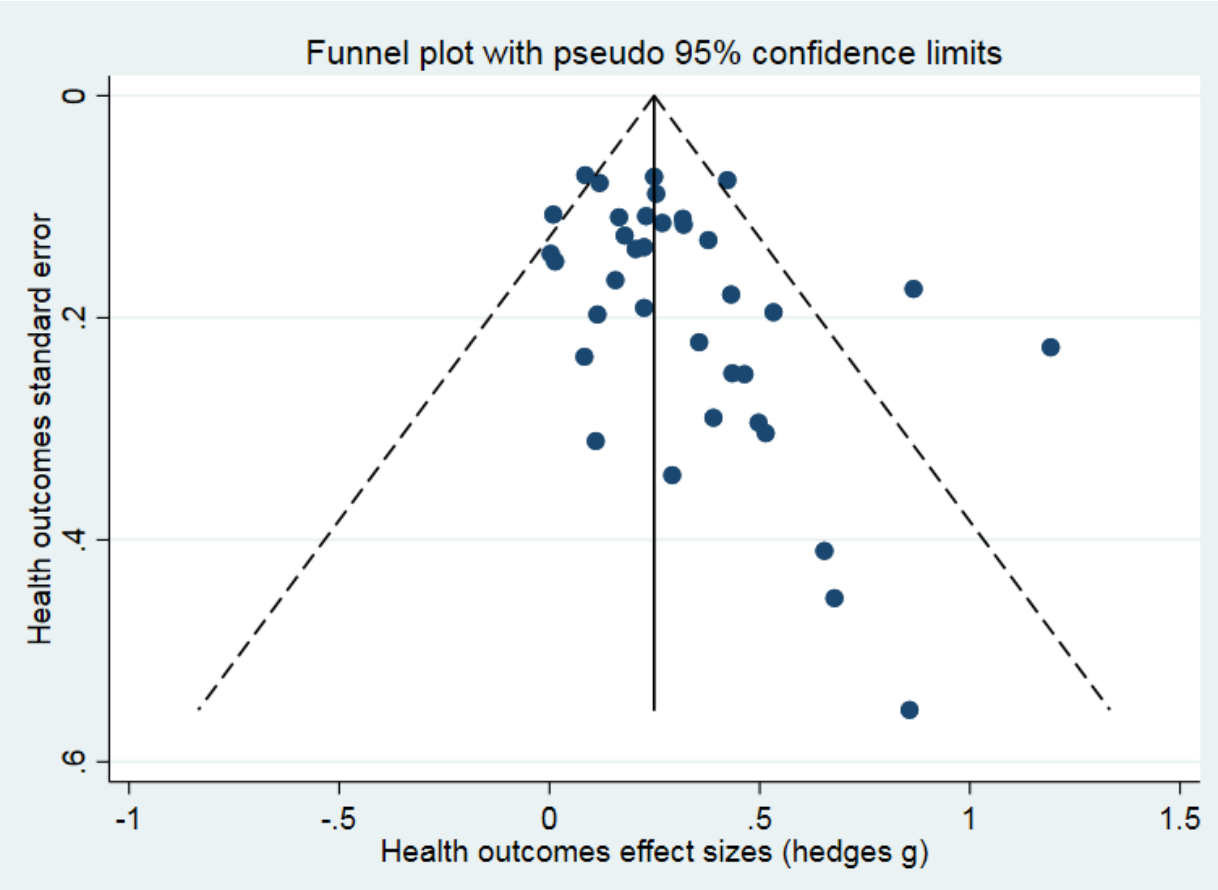
Rumination	Age	.003	.01
(*k* = 8)	Sleep disturbance	-.12	.02
	GAD participants	—	—
	% of participants female	-.0009	.01
	Adult's vs children	-.18	.30
	Measure time-point	.006	.006
	Number of sessions	.05	.04
	ITT analyses	-.30	.33
	Mode of delivery		
	Health-care professional	-1.14†	.56
	Self-administered	-.32	.33
	Self-administered with support	.17	.46
	Trained facilitator	.38	.26
	Intervention setting		
	Medical	-1.14†	.56
	Educational	-.10	.32
	Academic	.34	.28
	Hosted online vs In person	-.23	.29
	Active vs non-active control	.26	.30
	Individual vs group delivery	-.08	.31
	Student sample	.18	.30
	Attrition	.009	.001
HO (*k* = 36)	Age	-.002	.002
	Sleep disturbance	-.14	.17
	GAD participants	-.12	.09
	% of participants female	-.002	.002
	Adult's vs children	-.02	.10
	Measure time-point	-.0005	.0003
	Number of sessions	-.0007	.01
	ITT analyses	-.19	.08
	Mode of delivery		
	Health-care professional	.12	.15
	Self-administered	.18*	.07
	Self-administered with support	-.14†	.08
	Trained facilitator	-.11	.08
	Intervention setting		
	Medical	-.08	.08
	Educational	.14	.08
	Academic	-.05	.07
	Hosted online vs In person	.001	.07
	Active vs non-active control	-.02	.07
	Individual vs group delivery	-.16	.07
	Student sample	.07	.09
	Attrition	-.002	.002
HB (*k* = 21)	Age	-.004	.004
	Sleep disturbance	-.13	.11
	GAD participants	-.11	.12
	% of participants female	-.005	.004
	Adult's vs children	.10	.25
	Measure time-point	-.001**	.0003
	Number of sessions	.02	.02
	ITT analyses	.05	.12

	Mode of delivery		
	Health-care professional	—	—
	Self-administered	.26*	.09
	Self-administered with support	-.17	.12
	Trained facilitator	-.15	.14
	Intervention setting		
	Medical	-.20	.18
	Educational	.22	.15
	Academic	-.02	.13
	Hosted online vs In person	.10	.12
	Active vs non-active control	-.08	.12
	Individual vs group delivery	-.25*	.12
	Student sample	-.19	.13
	Attrition	.002	.004
PHO (*k* = 21)	Age	-.002	.003
	Sleep disturbance	.009	.006
	GAD participants	.002	.003
	% participants female	-.001	.001
	Adult's vs children	-.15	.11
	Measure time-point	.0003	.005
	Number of sessions	-.002	.001
	ITT analyses	-.02	.001
	Mode of delivery		
	Health-care professional	.16	.13
	Self-administered	.09	.09
	Self-administered with support	-.14	.09
	Trained facilitator	-.05	.10
	Intervention setting		
	Medical	.01	.10
	Educational	.19**	.07
	Academic	-.17*	.08
	Hosted online vs In person	-.13	.09
	Active vs non-active control	.04	.09
	Individual vs group delivery	.002	.09
	Student sample	-.01	.12
	Attrition	-.004	.002

Note: *p < .05; **p < .01; *** p <.001; † = p>.05-.09; — = dropped due to collinearity issues; ◎= the categorical predictors for these analyses are set as 1 (feature present) and 0 (feature not present); PC = perseverative cognition; HO: health outcomes (health behaviours and physical health outcomes combined); HB: health behaviours; PHO: physical health outcomes, Clin vs non-clin: whether participants derived of a clinical or . background; M time-point: point in time at which measures were taken; N sessions: number of sessions participants were exposed too; ITT analyses: whether the results influenced intention-to-treat analysis.

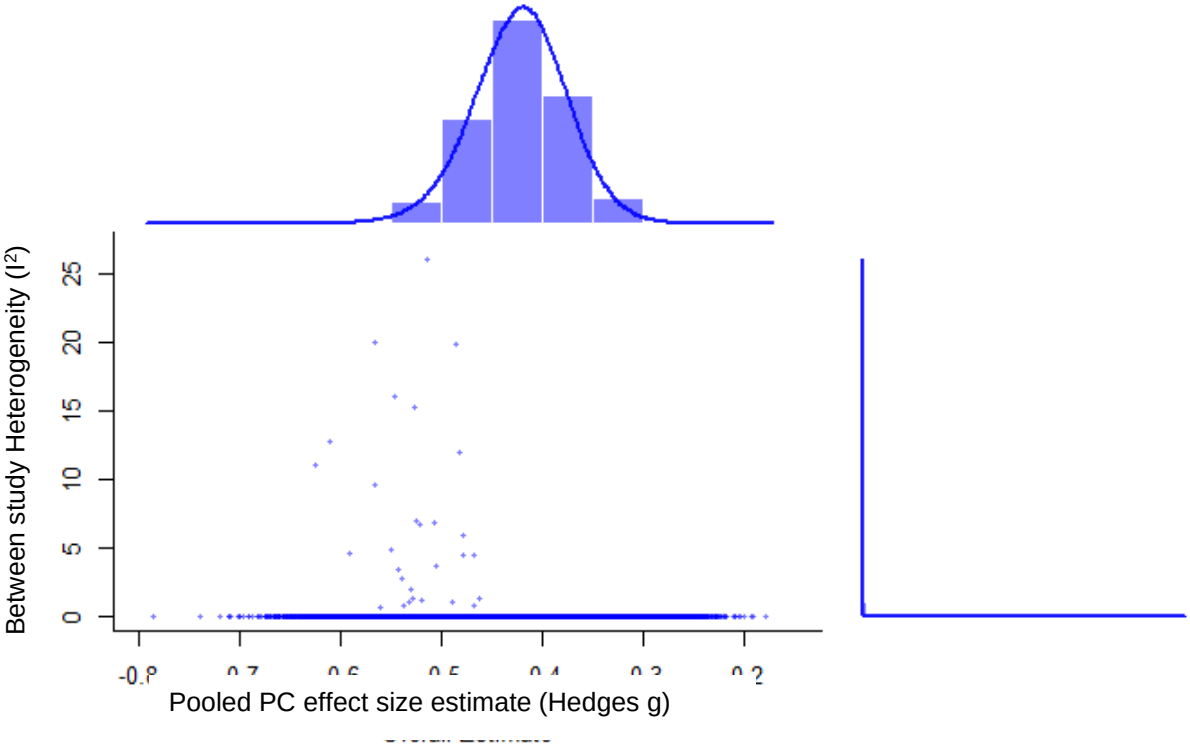
Appendix 1.1.7 Funnel plot for health outcomes

Funnel plot for study outcomes (health outcomes).



Appendix 1.1.7 GOSH plot

Graphical Display of Heterogeneity (GOSH) plot with PC effect sizes as a function of between-study heterogeneity across all studies (*k* = 36).



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Appendix 2.2 – Chapter 3 Supplementary Material B: Supplementary Results Section: Additional exploratory analyses and robustness checks

A range of additional analyses were conducted to: (a) check data met the statistical assumptions associated with regression such as multivariate normality, low multicollinearity, lack of auto-correlation and homoscedasticity; (b) identify potential confounds that may have affected the conclusions and consider the results when the behavioural and physical health outcomes were combined as an *overall* health index; (c) assess the possible impact of two studies for which we had concerns regarding the measures of behaviour; assess the robustness of the findings when focused only on studies (d) measuring PC *immediately* post-intervention and then health at a *later* point in time and (e) measured sleep; (f) check for small-study bias; (g) assess, when an alternative study arm was available (i.e., two treatment arms/different control types), if our approach to arm selection significantly altered study effect sizes for both PC and health; h) control for the possibility that baseline between group differences influenced effect sizes; i) detect if clinical heterogeneity influenced effect sizes.

A. Statistical assumptions

Visual inspection (i.e. radial & QQ plots) and formal tests (i.e. Cook's distance, DFBETAS) were conducted to ensure data met the statistical assumptions associated with regression such as multivariate normality, low multicollinearity, lack of auto-correlation and homoscedasticity. To identify potential patterns of effect sizes and heterogeneity in our data Graphic Display of Heterogeneity (GOSH) plots (Olkin, Dahabreh, and Trikalinos 2012) were computed. This function fits the same random effects meta-analysis model to all possible subsets of included studies meaning not only K^{-1} models are fitted, but all 2^{k-1} possible study combinations. Further, as an extra safeguard against detecting false-positives the Hartung-Knapp-Sidik-Jonkman (HKSJ, see Hartung & Knapp, 2001a) method was used to calculate effect sizes across all primary analyses when between study heterogeneity was statistically significant (in addition to Hedges' *g*).

Throughout, all appropriate statistical assumptions and graphical checks were met across these tests and no assumptions were found to be violated. The GOSH plot analysis revealed that although heterogeneity was high, the calculated effect sizes for PC represent a consistent distribution across all possible random sub-sets of the studies in this review with no significant sub-clusters present in the data. Furthermore, when the HKSJ method was used to calculate effect sizes due to significant heterogeneity within the analyses the effects from all primary analyses (using Hedges' g) were upheld (a summary is available from the lead author upon request).

B. Confounding assessments

To identify potential confounds that may have affected the conclusions, chi-square analyses and Pearson's correlations were conducted to examine whether pairs of significant moderators co-occurred. When significant moderators co-occurred, they were entered simultaneously as predictors in multivariate meta-regressions to determine whether or not any predictor explained significant unique variance in effect size outcomes. For clarity, and to understand the context in which these tests were run, all analyses that aimed to identify potential confounds between study variables are reported in the appropriate 'objective' sub-section below.

Objective 1b:

Study characteristics associated with greater effect sizes for PC.

In the main report, one study characteristic was associated with larger effect sizes for PC: studies testing interventions delivered by healthcare professionals generated larger effect sizes than studies testing interventions not delivered by healthcare professionals, $B = 0.39$, $S.E. = 0.18$, $CI = -0.77 - -.009$, $p = .045$. As no other moderator significantly predicted PC, no further analyses were conducted.

Objective 2a

Can interventions targeting PC also impact health outcomes?

In the main report, we report the effect of the interventions targeting PC on health behaviour and physical health outcomes separately. Here, and wherever the term *overall* is used, we

report the effect of these interventions on a combined outcome (health behaviours + physical health outcomes – health *overall*).

The interventions produced, on average, small, but significant and heterogeneous I^2 = 48.1%; $Q(35) = 67.45$ $p < .001$, effect sizes for health outcomes overall $g = 0.28$, 95% CI = 0.21 to 0.34 ($k = 36$).

Objective 2b

Study characteristics associated with larger effect sizes for Health Overall.

As above, we repeated the analyses that were conducted separately for health behaviour and physical health outcomes and reported in the main text such that we test the association between study characteristics and for health overall.

These analyses revealed that all intervention types had a significant positive effect on health overall with the exception of pain management strategies. The effect sizes in studies testing psychological detachment style interventions were larger than in studies testing different types of interventions, for health overall, $B = 0.30$, *S.E.* = 0.18, *CI* = 0.07 − 0.54, $p = .014$. For health overall, interventions were significantly more effective at yielding larger effect sizes in studies where content was self-administered $B = 0.18$, *S.E.* = 0.07, *CI* = -0.04 − 0.31, $p = .013$, as opposed to those in which content was delivered by a health-care professional, $B = 0.12$, *S.E.* = 0.15, *CI* = -0.18 − 0.42, $p = .415$, or a trained facilitator, B = -0.11, *S.E.* = 0.08, *CI* = -0.28 − 0.06, $p = .196$.

Study characteristics associated with larger effect sizes for Health Behaviours.

Further to the main report of: Effect sizes were significantly larger when interventions were self-administered $B = 0.26$, *S.E.* = 0.09, *CI* = 0.07 − 0.45, $p = .01$, delivered at an individual level rather than group-level, $B = -0.25$, *S.E.* = 0.11, *CI* = -0.49 − 0.006, $p = .045$, and when health behaviours were assessed closer to the conclusion of an intervention $B = -0.001$, *S.E.* = .0003, *CI* = -.002 − -.0003, $p = .01$ ($k = 21$). Given these moderators co-occurred, we ran further analyses to test for confounding. Accordingly, self-administered interventions tended to be delivered to individuals, χ^2 (1) = 11.08, $p < .001$, and self-administered interventions tended to have shorter follow-ups, $r = -.60$, $p < .001$. In subsequent multivariate meta-

regressions to account for these potential confounds, self-administered interventions marginally predicted health behaviour effect sizes when controlling for group/individual delivery format, *B* = 0.20, *S.E.* = 0.10, *CI* = -0.02 − 0.43, *p* = .05, but not after controlling for time-point, *B* = 0.16, *S.E.* = 0.13, *CI* = -0.12 − 0.42, *p* = .237. Neither group/individual delivery format, *B* = 0.002, *S.E.* = 0.09, *CI* = -0.18 − 0.19, *p* = .979 or measure time point, *B* = -0.0006, *S.E.* = 0.0005, *CI* = -0.0018 − 0.0005, *p* = .24, explained unique variance in health behaviour effect sizes, thus suggesting some evidence of confounding.

Study characteristics associated with larger effect sizes for Physical Health Outcomes

Further to the main report of: while no particular intervention type was related to significantly larger effect sizes for physical health outcomes, interventions were at their most effective when delivered in educational, *B* = 0.19, *S.E.* = 0.07, *CI* = 0.48 − 0.32, p = .01, and academic settings, *B* = -0.17, S.E. = 0.08, *CI* = -0.35 − 0.06, *p* = .043, as opposed to delivered in medical settings, *B* = 0.009, *S.E*. = 0.10, *CI* = -0.19 − 0.21, *p* = .919. We did not, however, conduct further tests to detect confounding as it was not theoretically possible for a study to be conducted in more than one setting and because no other moderators co-occurred.

Objective 3:

Are larger effect sizes for PC associated with larger, but positive, effect sizes for health overall?

There was a non-significant trend regarding the association between PC effect sizes health outcomes overall *effect* sizes, *B* = -0.21, *S.E.* = 0.11, *CI* = -0.43 − 0.02, *p* = .067 (*k* = 36, see Table 1). However, following the removal of two studies identified as multivariate influential cases (Magnan et al., 2014 & Thiart et al., 2015), medium-sized effects for PC, *g* = .41, were associated with small, but positive, *g* = .25, effect sizes for health overall, *B* = -0.25, *S.E.* = 0.09, *CI* = -0.44 − -0.07, *p* = .008 (*k* = 34). This effect was upheld in subsequent permutation tests with 10,000 random computations, *B* = -0.36, *S.E.* = 0.21, *CI* = -0.78 − 0.05, *p* = .038. Larger effect sizes for worry, *B* = -0.46, *S.E.* = 0.21, *CI* = -0.92 − 0.09, *p* = .054 (*k* = 14), and rumination, *B* = -0.71, *S.E.* = 0.27, *CI* = -1.58 − 0.15, *p* = .062 (*k* = 5), specifically, were

marginally associated with larger effects for health behaviours, with a g = .41 for worry corresponding with a g = .27 in health behaviours, and a g = .56 in rumination corresponding with a g = .38 in health behaviours.

Effect sizes for worry, B = -0.38, *S.E.* = 0.22, *CI* = -0.83 − 0.08, p = .091 (k = 19), and rumination (k = 8), B = -0.43, *S.E.* = 0.21, *CI* = -0.94 − 0.07, p = .081, were not significantly associated with effect sizes for health *overall*.

C. Sensitivity analyses for two studies using proxy measures for health behaviours.

Given two of the included studies interested in health behaviour (Christiansen et al., 2014; Aardoom et al., 2016) used measures (AUDIT & EDE-Q, respectively) incorporating items relevant to both health behaviours *and* determinants of health behaviours within a single index (i.e., proxy measures, while all other related studies only included behavioural items), we removed these two studies in an additional sensitivity analysis to ensure this feature did not influence any of the conclusions.

The findings reported in the main manuscript were upheld. The interventions, on average, led to a small-to-medium, and heterogeneous I^2 = 48.8%; Q(33) = 64.39 p < .001, effect sizes for health behaviours, g = 0.29, 95% *CI* 0.22 to 0.36 (k = 34) and effects for PC were only marginally associated with positive effect sizes for health behaviours, B = -0.20, *S.E.* = 0.10, *CI* = -0.40 − 0.007, p = .058. Thus, suggesting the inclusion of these two studies had no meaningful impact on the study objectives relating to health behaviours.

D. Accounting for the potential impact of reverse causality between PC and health.

To minimize the potential impact of reverse causality between PC and health (i.e. intervention content first influencing health before being captured within measures of PC), studies measuring PC *immediately* post-intervention and then health at a *later* point in time, were subject to additional tests. This sub-set of studies (k = 18, 50%) were subject to a separate meta-regression examining if effect sizes for PC were positively, and significantly, associated with effect sizes for health outcomes (overall), to control for this possibility. As an

extra precaution, PC effect sizes for these 18 studies were also directly compared via a Welches *t*-test to the remainder of studies which simultaneously measured PC *and* health either immediately post-intervention (*k* = 15, 41%), or within follow-up measures (*k* = 3, 9%), to detect if they significantly differed depending on the point in time in which they were collected post-intervention. Note, we did not run this separately for health behaviours and physical health outcomes due to power concerns.

The sub-group meta-regression comprising studies measuring PC *immediately* post-intervention, and health outcomes (overall) later (*k* = 18, 50%), revealed effect sizes for PC significantly predicted more positive health effect sizes, *B* = -0.36, *S.E.* = 0.15, *CI* = -0.67 − -0.04, *p* = .031, denoting lower levels of PC in the intervention condition versus the control. Furthermore, a Welches two-sample *t*-test comparing this sub-set of studies to those which measured PC *and* health at the same point in time (*k* = 18), indicated that PC effect sizes did not significantly differ between the two sub-sets of studies as a function of time, *t* (36) = -.31, *p* = .371. Deviations in PC that occurred following the delivery of an intervention package are thus unlikely to have been driven by effects for health outcomes and do not differ across the period of time in which all post-intervention measures were collected.

E. Analyses relating to Sleep

Additional analyses were conducted for the most common health outcome (sleep, *k* = 17). The interventions produced, on average, small-medium and non-heterogeneous I^2 = 8.1%; *Q*(16) = 4.49 *p* = .997, effect sizes for sleep, *g* = 0.30, 95% CI = 0.11 to 0.49 (*k* = 17). Effect for PC, *B* = -0.21, *S.E*. = 0.11, *CI* = -0.52 − 0.04*, p* = .022, and worry specifically, *B* = -0.76, *S.E*. = 0.28, *CI* = -1.41 − -0.11*, p* = .027, but not rumination, *B* = -0.62, *S.E*. = 0.34, *CI* = -.1.72 − -0.47*, p* = .167, were positively associated with parameters of sleep (i.e., total-sleep-time/sleep-onset-latency) (see, Table 1). In addition, the effect sizes in studies testing psychological detachment style interventions were larger than in studies testing different types of interventions for sleep, *B* = 0.35, *S.E.* = 0.11, *CI* = .109 − .583, *p* < .001.

Studies which included a measure of sleep, versus those which did not, were entered as an additional moderator to assess if larger effect sizes were associated with this intervention

feature. However, there was no evidence to suggest studies which measured sleep yielder larger effect sizes on behaviour when compared to all other studies, *B* = -0.10, *S.E*. = 0.09, *CI* = -.30 − .10*, p* = .309 (*k* = 36). Furthermore, psychological detachment interventions generated significantly larger effect sizes for studies testing this type of intervention within measures of sleep versus those studies testing other types of intervention, *B* = 0.35, *S.E.* = 0.11, *CI* = .109 − .583, *p* < .001.

F. Testing for small study bias

Small-study bias, whereby larger effect sizes tend to be reported within smaller sample sizes, was examined using Egger's test. Duval and Tweedie's (2000) trim and fill analysis was conducted to estimate the impact of publication bias on PC and health outcome effect sizes.

Egger's regression coefficient was non-significant for PC (*p* = .087) but was significant for health outcomes (overall) (*p* = .022) suggesting small study bias for the latter. Thus, Duval and Tweedie's (2000) trim and fill analysis imputed nine additional effect sizes for the effect of the interventions on health outcomes (overall), generating an overall effect size of *g* = .22 (95%*CI* = 0.13 − 0.30). Consequently, the effect of the interventions on PC and health outcomes remained significant after controlling for small-study bias.

G. Potential impact of studies with multiple study arms.

We took extra steps to control for potential selection-bias when an alternative study arm was available (e.g., Topper et al., 2017; internet vs. group-based therapy). There were six studies whereby more than one study arm was available to choose from as the 'treatment' arm. For the 5 intervention arms, we first prioritized the intervention arm authors hypothesized to produce greatest effect sizes in PC (this was the case for 4/5 of studies). In one case when this was not reported, as we were interested in the most effective methods at influencing PC, we chose the arm which yielded the largest effect size in PC. Only 1 study required us to make a choice between comparator arms. In this one instance (Versluis et al., 2018), we followed the conservative approach of selecting the attention-placebo control, as it is well known that effect sizes of interventions compared with no-treatment control groups are

greater than effect sizes of interventions compared to attention-placebo control groups (Lipsey & Wilson, 1993).

To control for the potential inflation of effect-sizes we ran a further sensitivity analysis; first, via a series of meta-regressions with the feature of 'more than one intervention arm present' set as the predictor and study effect sizes for PC, health behaviours and physical health outcomes as the DV. Importantly, effect sizes for PC, *B* = 0.18, *S.E*. = 0.12, *CI* = -0.05 − 0.42, *p* = .124, health behaviours, *B* = - 0.10, *S.E*. = 0.09, *CI* = -0.21 − -0.82, *p* = .309, and physical health outcomes, *B* = - 0.07, *S.E*. = 0.16, *CI* = -0.31 − -0.71, *p* = .317, were unrelated to the number of intervention arms a study employed. Second, we conducted sensitivity analyses in which these studies that included more than one 'treatment arm' were removed from the meta-analyses. The impact on the conclusions was negligible for PC: *g* = - 0.42, 95% *CI* = -0.51 to -0.21; health behaviours: *g* = 0.32, 95% *CI* 0.24 - 0.49, and physical health outcomes: *g* = 0.22, 95% *CI* = 0.22 - 0.31. Crucially, these analyses shows our handling of intervention arms did not bias this reviews conclusions.

H. Assessing potential impact of baseline differences between study arms

To control for the possibility that baseline differences between study conditions influenced effect sizes, we carried out two further tests. Seven studies reported significant baseline differences in PC and two studies reported significant baseline differences in health outcomes. First, a univariate meta-regression, with reported vs. non-reported baseline differences set as the predictor and effect sizes for PC, health behaviours and physical health outcomes, respectively set as the DV, was carried out. Study effect sizes for PC, *B* = 0.11, *S.E*. = 0.23, *CI* = -0.12 − 0.32, *p* = .271, health behaviours, *B* = - 0.12, *S.E*. = 0.14, *CI* = -0.52 − 0.24, *p* = .159, and physical health outcomes, *B* = -0.09, *S.E.* = 0.19, *CI* = -0.49 − 0.19, *p* = .347, were unrelated to the presence of baseline differences among studies. Second, we conducted sensitivity analyses in which these studies that reported baseline differences on specific measures were removed from the meta-analyses. The impact on the conclusions was minimal (PC: *g* = - 0.41, 95% *CI* = -0.54 to -0.25; health behaviours: *g* =

0.30, 95% *CI* 0.19 - 0.40; physical health outcomes: *g* = 0.22, 95% *CI* = 0.12 - 0.29). Therefore, we can be fairly confident that any degree of baseline between-condition difference did not meaningfully impact any of our analyses which rest upon this assumption.

I. Examining the potential impact of clinical differences in participant characteristics.

To control for the possibility that clinical baseline heterogeneity between studies which either contained GAD participants (Conrad et al., 2008 & Freshour et al., 2016; *N* = 2) or pertained participants with sleep disturbance (Sandlund et al., 2018; Pech & O'Kearney, 2013; Jansson-Frojmark et al., 2012; Harvey et al., 2017; N = 4) affected the conclusions, we carried out three further analyses.

First, two sets of univariate meta-regressions (i.e., one for each sample type), with sample type (GAD sample: yes/no; sample with sleep disturbances: yes/no) set as the predictor and effect sizes for PC, health behaviours and physical health outcomes, respectively set as the DV, was carried out. Study effect sizes for PC (*B: -.26, S.E = .09, p = .204),* health behaviours *(B: -.13, S.E = .11, p = .112*) and physical health outcomes (*B: .002, S.E = .003, p = .62*) were not significantly impacted by GAD samples, and the same was true for those studies containing participants with sleep disturbances for PC: (*B: -.21, S.E = .18, p = .174),* health behaviours *(B: -.13, S.E = .11, p = .403),* and physical health outcomes *(B: .009, S.E = .003, p = .405*).

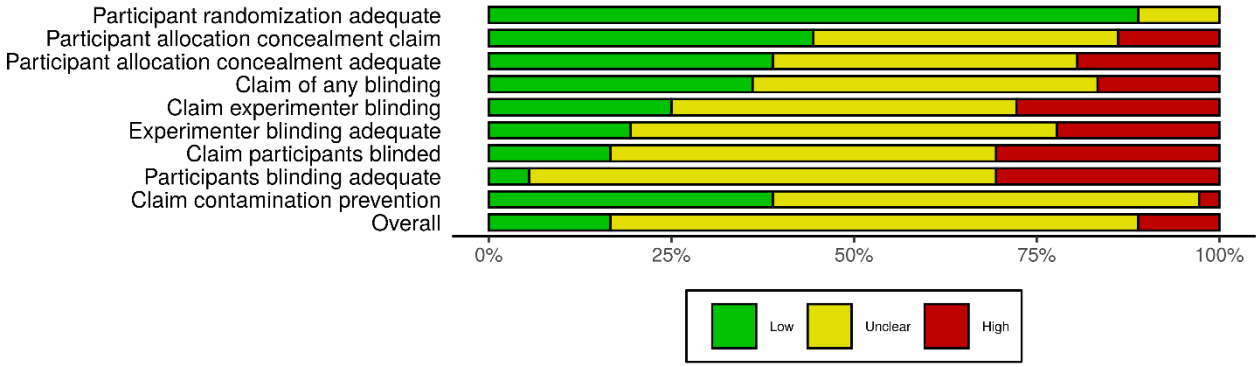
Second, we conducted two sensitivity analyses in which the studies that had GAD participants, or those with 'clinical' sleep disturbances, were removed from the respective meta-analyses. The impact on the conclusions was minimal (PC: *g* = - 0.41, 95% *CI* = -0.57 to -0.29; health behaviours: *g* = 0.29, 95% *CI* 0.17 - 0.39; physical health outcomes: *g* = 0.21, 95% *CI* = 0.15 - 0.31) when removing the 2 studies including GAD participants, and similar effects were found when (separately) removing the 4 studies comprising participants with sleep disturbances (PC: *g* = - 0.40, 95% *CI* = -0.54 to -0.33; health behaviours: *g* = 0.30, 95% *CI* 0.20 - 0.38; physical health outcomes: *g* = 0.23, 95% *CI* = 0.13 - 0.30).

Therefore, we can be fairly confident that any degree of baseline between-condition difference did not meaningfully impact any of our conclusions.

Third, to assess if these samples had any impact on objective 3 (i.e., the association between PC and health) we re-analysed with the 6 studies (2 GAD studies & 4 sleep studies) removed; along with any influential cases relevant to either outcome removed to be consistent with the main report. The impact of removing these 6 studies (and one influential case, Magnan et al., 2014) on the findings was minimal. Medium-sized effects for PC, g = -.39, were still associated with a small, but positive, g = .24, effect for health behaviours, B = -0.22, *S.E.* = 0.13, *CI* = -0.47 − -0.11, p = .028. A similar trend was present with physical health outcomes when compared to our original analysis. Effect sizes for PC were still unrelated to effect sizes for physical health B = -0.15, *S.E.* = 0.21, *CI* = -0.58 − 0.17, p = .328, when removing these 6 studies (and an influential case, Digdon & Koble, 2011), B = -0.16, *S.E*. = 0.08, *CI* = -0.56 − 0.19, p = .292. As such, combined, these three sets of analyses show that we can be fairly certain that while sample characteristics are important to consider, they had very little bearing on the findings of this particular meta-analysis.

Appendix 2.3 Chapter 3 Supplementary Material C: Traffic Light Plot

Appendix 2.4 Chapter 3 Supplementary Material D: Traffic Light Plot



Appendix 3.1 – Chapter 5 Supplementary Material

Appendix 3.1. Moderation Analysis for Physical Activity

	Dependent variable:					
	Physical Activity Frequency (1)			Physical Activity Duration (2)		
	β	*p*	R^2	β	*p*	R^2
Step 1: Intention	.373	<.001***	.139	.243	<.001***	.059
Step 2: Worry	.005	.906	.012	-.013	.740	.059
Step 3: Intention*Worry	-.250,	.121	.143	-.178	.291	.061
Step 1: Intention	.373	<.001***	.139	.243	<.001***	.059
Step 2: Rumination	.060	.119	.019	.002	.960	.059
Step 3: Intention*Rumination	-.016	.930	.143	.107	.563	.059
Step 1: PBC	.338	<.001***	.114	.184	<.001***	.034
Step 2: Worry	.01	.820,	.114	-.013	.746	.034
Step 3: PBC*Worry	-.449	.011*	.123	-.321	.097	.039
Step 1: PBC	.338	<.001***	.114	.184	<.001***	.034
Step 2: Rumination	.071	.066	.119	.007	.854	.01
Step 3: PBC*Rumination	-.075	.701	.120	.035	.864	.034

Note: ***p* < .0125, ***p* < .01 ****p* < .001; PBC = Perceived Behavioural Control

Appendix 3.2 Moderation Analysis for Sedentary Activity & Unhealthy Snacking

	Dependent variable:					
	Sedentary Activity (1)			Unhealthy Snacking (2)		
	β	*p*	R^2	β	*p*	R^2
Step 1: Intention	.109	.008**	.012	-.071	083	.005
Step 2: Worry	.147	.232	.013	-.261	.030	.011
Step 3: Intention*Worry	-.059	.766	.015	-.273	.093	.011
Step 1: Intention	.109	.008**	.012	-.071	.083	.005
Step 2: Rumination	.154	.305	.012	-.216	.134	.006
Step 3: Intention*Rumination	-.065	.757	.012	-.273	.093	.011
Step 1: PBC	.207	.008**	.003	-.165	<.001***	.027
Step 2: Worry	.196	.131	.006	-.047	.771	.027
Step 3: PBC*Worry	-.277	.236	.007	.079	.672	.028
Step 1: PBC	.207	.008**	.003	-.165	<.001***	.027
Step 2: Rumination	.046	.766	.003	.019	.907	.026
Step 3: PBC*Rumination	.010	965	.003	-.026	.907	.027

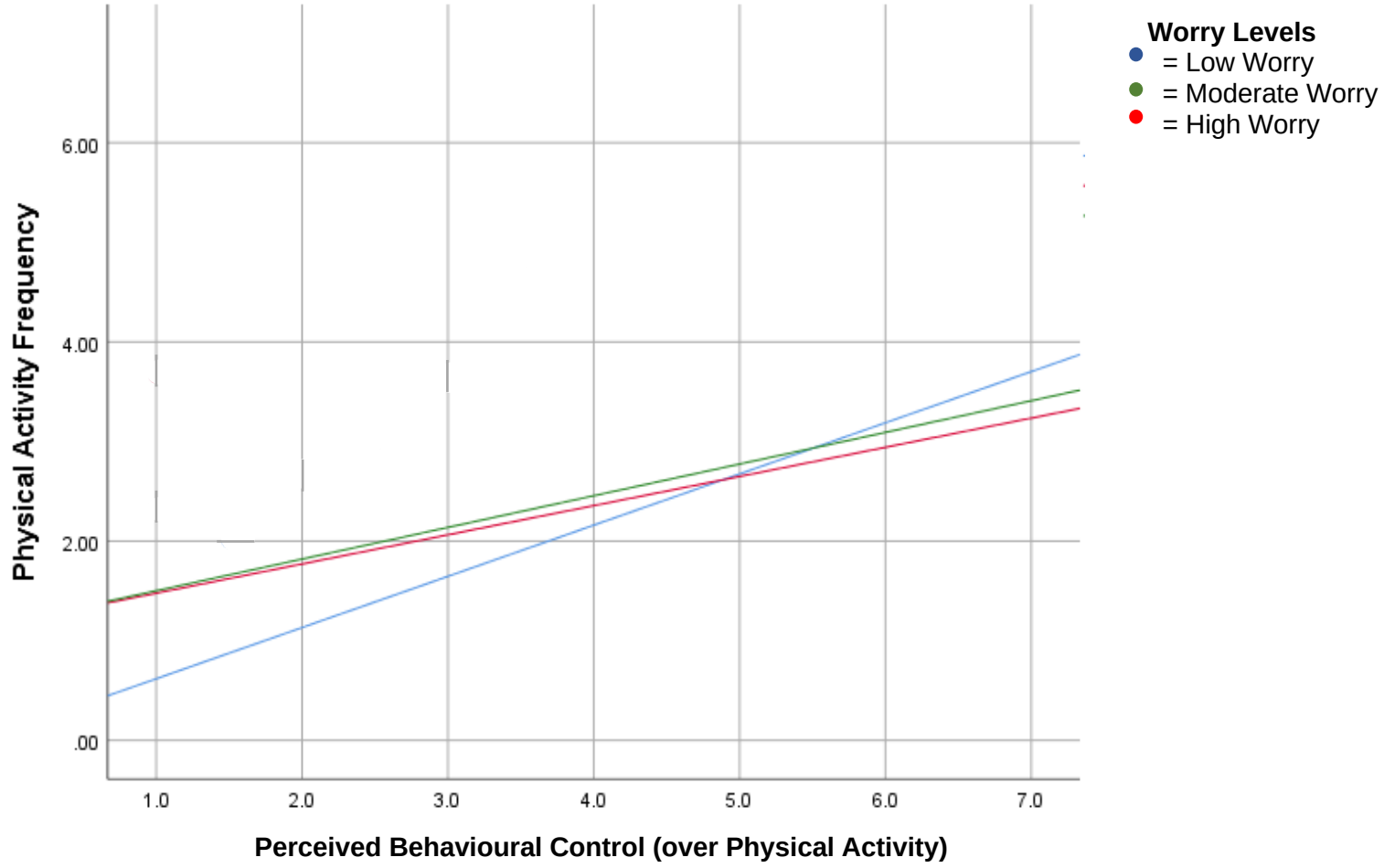
Note: ***p* < .0125, ***p* < .01 ****p* < .001; PBC = Perceived Behavioural Control

Appendix 3.3 Moderation Analysis for Sleep Behaviours

	Dependent variable:								
	Total Sleep Time (1)			Sleep Quality (2)			Sleep Onset Latency (3)		
	β	*p*	R^2	β	*p*	R^2	β	*p*	R^2
Step 1: Intention	.383	<.001***	.147	.263	<.001***	.069	-.060	.144	.004
Step 2: Worry	-.041	.285	.148	-.250	<.001***	.131	.053	.196	.006
Step 3: Intention*Worry	.128	.455	.149	.203	.239	.133	-.226	.221	.009
Step 1: Intention	.383	<.001***	.147	.263	.001***	.069	-.060	.144	.004
Step 2: Rumination	.086	.024	.154	-.204	.001***	.110	.076	.064	.009
Step 3: Intention*Rumination	-.055	.772	.154	.201	.301	.112	-.282	.169	.013
Step 1: PBC	.529	<.001***	.280	.498	<.001***	.248	-.115	.005**	.013
Step 2: Worry									
Step 3: PBC*Worry	.031	.805	.280	.133	.290	.287	-.170	.248	.017
Step 1: PBC	.529	<.001***	.280	.498	<.001***	.248	-.115	.005**	.013
Step 2: Rumination	-.043	.220	.281	-.160	<.001***	.273	.067	.107	.018
Step 3: PBC*Rumination	.036	.809	.281	.212	.155	.276	-.173	.316	.019

Note: ***p* < .0125, ***p* < .01 ****p* < .001; PBC = Perceived Behavioural Control

Appendix 3.4 Simple Slopes Analysis for PBC, Physical Activity (Frequency) and Worry



Note: For worry levels, High corresponds to +1SD above the mean, Moderate to the mean, and Low to -1SD below the mean.