Psychological and cumulative cardiovascular effects of repeated angry rumination and visuospatial suppression

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ABSTRACT

Brooding rumination is associated with depressed mood, increased negative affect, prolonged anger and inhibited cardiovascular (CV) recovery. Distraction from rumination on a stressful interpersonal encounter is associated with faster CV recovery and decreased negative affect. Studies have suggested that a concurrent visuospatial (VS) task inhibits the maintenance of imagery associated with the perseveration of intrusive negative memories. 120 healthy participants were recruited for the study. As an analogue of repeated angry rumination, the authors explored the effects of repeated visual recall of a provocative confederate and the subsequent impact of two visuospatial (VS) distraction tasks on negative affect, blood pressure (BP) and heart rate (HR). Repeated recall of the provocation generated repeatedly elevated HR with a cumulative trend that may have CV disease risk implications for chronic ruminators. VS distraction did not aid recovery compared with the Control task.

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1. Introduction

1.1. Psychological and CV effects of anger

Anger experience and expression is a widely researched domain spanning cognitive, emotional and physiological features. Whilst some contradictory findings exist in the psychophysiology literature regarding the effects of suppression of anger and to a lesser degree, anger expression (e.g. Hogan & Linden, 2004, Suls et al., 1995), many researchers agree that aggressive expression or suppression of anger is detrimental to one's CV health (see for example Davidson et al., 1999; Siegman et al., 2000; Siegman & Snow, 1997; and Friedman & Booth–Keewley, 1987).

The anger–hypertension model posits that stressors tend to generate acute levels of BP (reactivity) and that over time this results in elevation of the tonic BP level (attenuated recovery) leading to hypertension and incidence of atherosclerosis (Manuck et al., 1990). Linden et al. (1997) and Linden and Long (1987) contend that persistent BP elevation (poor recovery) is as clinically important as physiological reactivity, but that the former has received far less empirical attention. Delayed HR recovery and elevated resting HR have also been implicated in CV disease risk (e.g. Jae et al., 2008) with chronic psychological stress (e.g. Lucini et al., 2005) and rumination (Linden et al., 1997) posited as mechanisms for increased autonomic activity and decreased parasympathetic activity. In contrast to the extensive literature linking anger and elevated BP with hypertension, fewer studies have focussed on anger and HR. Earle et al. (1999) found that the largest physiological reaction to harassment for females was for HR, not BP and Brosschot and Thayer (2003) reported that negative rumination was linked to prolonged attenuation of HR, post-provocation.

1.2. The role of heart rate in cardiovascular disease risk

An increasing body of literature in leading journals has linked resting HR (RHR) with coronary artery disease (CAD) risk. Dyer et al. (1980) claimed that data from three large US epidemiologic studies demonstrated increased RHR was a predictive factor for CAD and early death. Diaz et al. (2005) reported that the Dyer et al. findings were supported by the Framingham Heart Study data and by their own data from 24,913 patients tracked over 14 years. Diaz et al. found that increased HR and high RHR were independent predictors for increased all-cause and CV mortality.

Furthermore, Borer (2008), Palatini (2005) and Fox et al. (2007) claim there is extensive, compelling evidence that elevated RHR is independently and strongly linked to CAD and CV mortality and has direct detrimental effects on the progression of coronary atherosclerosis, on the occurrence of myocardial ischemia and ventricular arrhythmias, and on left ventricular function. The authors concluded that future CV guidance documents need to address the role of HR and HR modulation.
In addition, low HR variability (HRV) has been repeatedly associated with CV mortality (e.g., Tsuji et al., 1994, 1996; Huikuri et al., 1999). Sajadieh et al. (2004) found in a healthy middle-aged sample that low HRV and increased HR were both significantly associated with subclinical CV inflammations which have been posited in numerous studies as independent markers of subclinical and clinical atherosclerosis (e.g., Rizzo et al., 2009).

1.3. Effects of anger rumination

Rumination tends to increase anger beyond initial provocation levels (Rusting & Nolen-Hoeksema, 1998; Tice & Baumeister, 1993) and prolong anger as well as slow BP recovery (Glynn et al., 2002). Rusting et al. posited that self-justified angry rumination enables repeated rehearsal and maintenance of angry thoughts, inhibiting disengagement. Nolen-Hoeksema (2000) posited rumination as repeatedly dwelling on a stressor; one may ruminate for an extended period or repeatedly. In this study, repeated rumination refers to independent rumination events on the same stressor.

1.4. Repeated rumination and recurrent stressors

A few laboratory studies exist in which participants were subjected to a recurrent stressor, with mixed results. Kelsey et al. (2004) found that HR reactivity was attenuated but vascular reactivity was not attenuated by repeated stress exposure whereas Kelsey et al. (1999) found that prior exposure to stress-inducing maths tasks attenuated CV reactivity. Kirschbaum et al. (1995) found that repeated exposure to the same stressors over 5 days tended to repeatedly elevate physiological stress responses and Glynn et al. (2002) showed that recalling a previous laboratory stressor one week later generated significant BP elevations. This current study appears to be the first to explore the psychophysiological effects of repeatedly recalling stress-related memories.

1.5. Resentment

Provocation-related resentment is largely unstudied and yet Harburg et al. (2003, p.595) suggested that the interaction of suppressed anger with high BP (HR was not measured) reported in their 17-year study “may be partially generated by chronic resentment...” resulting from suppressive responses to ongoing severe injustice. Resentment is viewed as a complex domain that has biological (Cassel, 1976; Kahn et al., 1987) and morbidity implications (Kiecolt-Glaser et al., 1987). Whilst Harburg et al. (p.595) did not directly measure resentment, the authors stated that the “private, chronic, iterative re-arousal of anger/hostility due to the perception of chronic social attack is the sociopsychobiological toxic process we term chronic resentment”.

Rumination styles include worry (Brosschot & Thayer, 2003), and depressive or anger cognitions (Rusting & Nolen-Hoeksema, 1998). Anger, however, tends to be very short-lived (Kassinove et al., 1997). Lasting resentment (Mullet et al., 2005) is a more durable form of angry affect that involves holding an extended grudge (Ashton et al., 1998) and is associated with refusal to forgive or forget the offense (Worthington & Wade, 1999). For example, Mauger et al. (1992) operationalized resentment in the Forgiveness of Others scale as: I have grudges which I have held onto for months or years.

1.6. Distraction from rumination: psychological and CV effects

Cognitive distraction is reported as inhibiting rumination and accelerating CV recovery post-provocation (Schwartz et al., 2000). Gerin et al. (2006) found that anger-based memory recall significantly elevated BP and HR whereas distraction lowered BP but not HR. However, Neumann et al. (2004) found distraction generated lower anger and rumination scores and accelerated HR recovery but not BP recovery. Gerin et al. (2006), however, generally found, mean differences between distraction and rumination conditions in studies to be small.

1.7. Visuospatial suppression as a distraction task

Cognitive distraction has also been investigated via visuospatial (VS) suppression of the vividness of images related to stressful emotion-laden memories (e.g., Andrade et al., 1997; Kavanagh et al., 2001; Kemps & Tiggemann, 2007), and food cravings (e.g., McClelland et al., 2006). The working memory (WM) model (Baddeley & Hitch, 1974), posits that verbal and auditory information is coded and maintained separately in WM via the VS sketchpad and the phonological loop respectively. Both are viewed as slave components to the central executive attentional system. In separate studies, VS suppression tasks have overloaded the limited capacity of the VS sketchpad, disrupting the maintenance of recalled stressful images or craved-food images, resulting in lowering of emotions and food cravings, respectively. These effects suggest VS suppression tools are highly suitable for distraction from rumination tasks, post-provocation. Two VS suppression tools were utilised in the current study for this purpose. To date, past VS research has not measured CV correlates of emotional arousal associated with stressful memory recall, and any associated acceleration of CV recovery achieved through suppression of stressful memories utilising concurrent VS suppression tasks.

1.8. Laboratory social stressors and non-social stressors

In this study, we incorporated a social laboratory stressor—an insulting, critical evaluation—analogue to similar real-life stressors. Strong criticism has been voiced regarding the premise that non-social behavioural challenges increase CV reactivity and play a role in essential hypertension (e.g., Pickering & Gerin, 1990). Schwartz et al. (2003) and Linden et al. (1998) concurred with Turner et al. (1994) that stressful interpersonal tasks are more representative, reliable and generalizable as predictors of real-life ambulatory BP compared with traditional laboratory stressors and offer better explanatory power for studying disease pathways (Linden et al., 1997). The latter authors suggest that interpersonal stressors tend to elicit anger, which mediates sustained BP elevation, whereas math tests and similar tend to elicit initial, short-lived stress with unrealistically rapid recovery.

In summary, interpersonal stressors tend to generate high levels of anger and CV reactivity with attenuated recovery, (implicated in CV disease risk), particularly when angry rumination is present. Furthermore, VS suppression tasks have successfully disrupted stressful memories, however, the CV effects have not been explored, nor have the CV and emotional effects of repeated recall of an anger or resentment inducing provocation. Hence in this study we explored the CV and emotional effects of repeated recall of a stressful provocation, whilst assessing the impact of VS distraction tasks known to suppress emotional memory recall.

It was hypothesised that repeated visual recall of a provoking confederate would produce repeatedly elevated BP and HR relative to Baseline. It was also predicted that BP and HR would be lower during both the two VS distraction tasks compared with the Control task and that there would be no CV differences between the VS tasks.

2. Materials and methods

2.1. Participants

Participants comprised 115 volunteer healthy students and staff from Monash University. Unusable physiology data reduced the sample number to between 97 and 102, depending on the stage of the
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