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Recreating cardiovascular responses with rumination: The effects of a delay between harassment and its recall

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Abstract

Cardiovascular responses occur not only in the immediate presence of stressors, but also while later thinking about those experiences. Evidence suggests that these delayed responses, such as those produced by ruminating about prior angering experiences, may play an important role in the development of cardiovascular disease. We examine whether physiological consequences of rumination depend on the delay between a stressor and its recall, and whether the magnitude of physiological responses decreases with repetition. Twenty-two participants experienced a three-minute harassment stressor, and later spent 3 min vividly recalling the task. Half the subjects returned for the first time after a week, and half returned after half an hour, and then also after a week. Blood pressure and heart rate were monitored during a baseline period, and during each session's stressor or rumination period. Results indicated that rumination was sufficient to elevate blood pressure (systolic and diastolic) above baseline, that the delay made no difference to the magnitude of the elevation, but that the second rumination seemed to be associated with a smaller response than the first. Response to the stressor was not associated with rumination responses, but the first rumination response was significantly correlated with the second. The effects of stress may be experienced long after the actual stressor is passed, and people who experience large delayed responses may not be the same as those with high initial responses. The "hot" affective portion of rumination may not be diminished by the passage of time, but by prior recreation of the experience.

Keywords: Harassment; Recall; Cardiovascular responses; Rumination; Blood pressure; Recovery; Anger

Recent work examining the role of cardiovascular responses to stress in CVD morbidity and mortality has started to incorporate a broader characterization of the cardiovascular stress response than previously used. It is becoming increasingly recognized that it is valuable to examine cardiovascular states not only during stressors, but also during recovery from stressors (Christenfeld et al., 2000; Gerin et al., 1994; Gregg et al., 1999; Haynes et al., 1991; Hocking-Schuler and O'Brien, 1997; Linden et al., 1997; Mezzacappa et al., 2001). Post-stress cardiovascular recovery profiles appear to differ depending on family history of hypertension and CHD (Borghi et al., 1986; Gerin and Pickering, 1995; Hocking-Schuler and O'Brien, 1997; Sheffield, Davy Smith, Carroll, Shipley and Marmot, 1997) and also seem to depend on factors such as race (Anderson et al., 1989; Jackson et al., 1999; Mills and Berry, 1999) and certain personality traits including Type A and anger expression style (Faber and Burns, 1996; Fang and Myers, 2001; Jamieson and Lavoie, 1987; Lai and Linden, 1992).

The concept of recovery from a stressor need not be narrowly defined as the lingering physiological activation immediately following the stressor. Even after a return to baseline cardiovascular levels, the mental recreation of a stressful or anger-provoking event hours, days or even years later can produce a physiological response. Research examining the physiological components of emotion provides some evidence that these later recalls are associated with physiological activation. Many of these studies use the recall of a prior, emotionally-laden experience as an emotion-induction tool to examine the physiological responses associated with particular emotions, and these studies demonstrate that the act of recalling an emotional experience is indeed associated with increases in blood pressure, heart rate and

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galvanic skin response (de Jong-Meyer et al., 1993; Ekman et al.,1983; Schwartz et al., 1981; Stemmler, 1989). Anger recall appears to be a particularly potent elicitor of concomitant increases in blood pressure. Angry ruminations are consistently associated with physiological responses and appear to impede cardiovascular recovery following provocation (Brosschot and Thayer, 1998; Jannsen et al., 2001; Lavoie et al., 2001; Neumann et al., 2004; Prkachin et al., 2001; Suchday et al., 2004).

To date, one study using the cardiovascular reactivity paradigm has examined the cardiovascular response to both a stressor and to the later recall of that same stressor. Glynn et al. (2002) exposed participants to either emotional or non-emotional stressors, and then, after a short delay, asked them to recall the experience as vividly as possible. Only the emotional tasks were associated with increases in blood pressure during recall. The size of the response during recall did not depend on whether the initial emotional stressor produced a high or low cardiovascular reactivity response.

The Glynn et al. (2002) study indicates that the emotional nature of the stressor is one important parameter determining whether later recall of a stressful event produces a physiological response. Another parameter that may be important is the delay between the initial stressor and the recall of, or rumination about, that stressor. The relation between the delay and the response at recall is important because it may shed light on the viability of rumination as a pathway for the development of CVD. If the passing of an hour is enough to extinguish the associated physiological response upon reflection, then rumination-associated cardiovascular responses would seem less important in the development of CVD. If, however, even after a considerable delay, it is still possible, simply by thinking about the prior angering event, to produce a significant cardiovascular response, then the role of angry rumination as a risk factor for CVD would seem more plausible.

Foster and Webster (2001) examined the relation between physiological responses to an emotion-recall task, and the age of the emotional memory recalled. Specifically, participants were instructed to identify an incident that had made them angry in the past (the average age of the memory was 2.1 years) and their heart rate and galvanic skin responses were recorded during the directed recall of this memory. The results indicated that older memories were associated with larger galvanic skin responses, but not heart rate responses. The authors suggested that the physiological response to the recall of an emotional memory increases with time. However, due to the correlational nature of the study, it is possible that the older memories were of more traumatic events, or that people who chose older events were people who generally show greater responses. This work does show, however, that memories from quite far back are still capable of eliciting some physiological response, although the precise effects of delay, and the cardiovascular concomitants of rumination, are not fully elucidated.

The purpose of the present study is to explore, experimentally, the relation between the delay to recall of an anger-provoking stressor and the cardiovascular response to that recall. Participants were brought into the laboratory and exposed to a harassment stressor. For the second visit, half of the subjects returned 30 min later, and half 1 week later for the recall task. In addition, those that recalled the task at 30 min also recalled a second time at 1 week. This design allows the assessment of the effect of a delay on the physiological response at recall. It also allows us to explore the relation between the size of the initial cardiovascular response to the stressor and the size of the response at recall, as well as the effect of repeated recall on cardiovascular responses.

1. Method

1.1. Overview

On the first visit to the laboratory, all participants completed a mental arithmetic task with harassment while their blood pressure and heart rate were monitored. On the second visit to the laboratory, participants were asked to recall as vividly as possible the arithmetic task they had experienced on the previous visit. Half of the subjects (those in the Immediate condition) returned to the laboratory for the recall task 30 min after the first session. The other half (Delayed condition) returned exactly 1 week later. Those in the Immediate condition also returned 1 week later, and performed the recall task a second time.

1.2. Subjects

Participants included 9 males and 13 females at a large university. The mean age was 20.1 years and none of the participants reported having conditions or taking medications that might affect the dependent variables of interest. Participants received course credit in exchange for participation.

1.3. Physiological monitoring

Systolic blood pressure, diastolic blood pressure, and heart rate were collected using an Ohmeda Finapres 2300 blood pressure monitor. This instrument takes beat-to-beat pressures in a non-invasive manner, using the Penaz method. The participant wears an inflatable finger cuff on the third finger of the non-dominant hand. The Finapres has been demonstrated to be a useful alternative to intra-arterial blood pressure measurement in laboratory testing and clinical practice (Gorback et al., 1999; Imholz et al., 1990; Wieling et al., 1991). In addition, it has been shown to track intra-arterial readings extremely well, even during sudden changes of blood pressure, making it a good candidate for use during reactivity testing (Parati et al., 1989).

1.4. Procedure for the each study session

At each visit participants were told they would perform a simple task while their blood pressure and heart rate were monitored. The cuff of the Finapres monitor was attached to the middle finger of the non-dominant hand and a six-minute baseline was taken. Participants were instructed to sit quietly and relax during this period, and the experimenter left the room. Following the baseline period, the participants were given instructions for one of the two tasks, either the mental arithmetic