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Depressive symptoms and attenuated physiological reactivity to laboratory stressors

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ABSTRACT

There is evidence that depressive symptoms are associated with attenuated physiological reactivity to active stressors. However, it is not known whether blunted reactivity in depressed individuals is stressor-specific. We examined cardiovascular and electrodermal reactivity in non-clinical participants with varying levels of depressive symptoms to different active and passive stressors. Depressive symptoms were inversely related to both blood pressure and skin conductance reactivity during a public speaking task and the viewing of the speech video. However, no effects were found during a cold pressor task. Together these findings suggest that depressive symptoms are related to attenuated sympathetic nervous system reactivity in response to self-relevant stressors.

1. Introduction

Depression has been discussed to constitute a robust psychosocial risk factor for cardiovascular diseases (CVD; Barth et al., 2004; Rugulies, 2002; Wulsin and Singal, 2003). In particular, individuals with depressive symptoms are at higher risk for developing coronary artery disease, myocardial infarction, and complications following heart surgery. It has been suggested that the relationship between depression and CVD is mediated by behavioral factors on the one hand (e.g., substance abuse, diminished physical activity), and dysregulations in various physiological systems on the other hand [including the endocrine, immune, and autonomic nervous systems (Joynt et al., 2003; Lett et al., 2004)]. With respect to autonomic nervous system (ANS) dysregulation there is evidence to suggest that depressive symptoms are related to higher sympathetic nervous system (SNS) activation (Joynt et al., 2003), attenuated vagal tone (Carney et al., 2001; Hughes and Stoney, 2001; Rottenberg, 2007; Schwerdtfeger and Friedrich-Mai, 2009; Udupa et al., 2007), attenuated baroreceptorreflex-sensitivity (Watkins and Grossman, 1999), and elevated blood pressure (e.g., Grewen et al., 2004; Hamer et al., 2007; Light et al., 1998). These various effects may impose elevated load on the cardiovascular system, thus fostering the development of CVD.

Of note, depressive symptoms have also been related to cardiovascular reactivity (CVR). According to the reactivity hypothesis, repeated stress-related increases in cardiovascular function are assumed to accelerate a wear and tear on the artery walls, leading to endothelial dysfunction and, ultimately, CVD (Chida and Steptoe, 2010; Harris and Matthews, 2004; Schwartz et al., 2003). Hence, it seems reasonable to assume that elevated CVR constitutes one path through which depression could affect cardiovascular health.

A considerable number of studies has been devoted to the relationship between depressive symptoms and CVR. Whereas some studies could observe elevated CVR to laboratory stressors in depressed individuals (e.g., Light et al., 1998; Matthews et al., 2005), other studies found that this effect was dependent on other psychological variables (e.g., aggression; Betensky and Contrada, 2010) or even failed to support this relationship (Taylor et al., 2006). Of note, a meta-analysis of 11 studies published until 2001 (Kibler and Ma, 2004) report positive, however, not reliable associations between depression and CVR, thus providing only limited support for the assumption that CVR links depression with adverse health outcomes.

On the contrary, an increasing number of recently published studies found evidence for attenuated – and not elevated – CVR with increasing depression scores (e.g., Carroll et al., 2007; Phillips, 2011; Salomon et al., 2009; York et al., 2007). For example, using a mental arithmetic task as a laboratory stressor Carroll et al. (2007) found in a population study that individuals with elevated depressive symptoms showed lower systolic blood pressure (SBP) and heart rate (HR) responses. Essentially the same finding was reported by Phillips (2011). Similarly, York et al. (2007) could observe that depressed individuals with coronary artery disease exhibited smaller increases in HR and SBP during a public speaking task than their counterparts with comparably low depression

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scores. Hence, it appears that depression might be associated with diminished CVR during certain aversive encounters.

However, the role of stressor type in studies on depression and CVR is not well understood. Generally, two types of laboratory stressors can be distinguished, namely active stressors (e.g., public speaking tasks, mental arithmetics) and passive stressors [e.g., the cold pressor task (CP) or mirror tracing; Hurwitz et al., 1993; Obrist, 1981]. Whereas active stressors are associated with a cardiovascular response pattern, which can be characterized by blood pressure increase, elevated cardiac contractility and cardiac output, as well as decreased peripheral resistance (indicating a beta-adrenergic response profile), the blood pressure increase to passive stressors is accompanied by attenuated cardiac output, and elevated peripheral resistance (indicating an alpha-adrenergic response profile; Brownley et al., 2000; Hurwitz et al., 1993). It is interesting to note here that attenuated CVR in depressed individuals was mainly found when beta-adrenergic stressors were applied (Carroll et al., 2007; Phillips, 2011; York et al., 2007), but there is little support for this finding for alpha-adrenergic stressors. In particular, Salomon et al. (2009) examined CVR to a public speaking task and a mirror tracing task in individuals diagnosed with major depressive disorder and healthy controls, thus allowing to contrast the effects of stressor type within the same study. In line with recent evidence, depressed individuals showed significantly lower SBP, HR, and cardiac output during the speech stressor, whereas the evidence for attenuated CVR was less clear for the mirror tracing task, which is an alpha-adrenergic stressor. Hence, blunted CVR in depressed individuals seems to depend on the type of stressor.

Importantly, the finding of blunted CVR to active, beta-adrenergic stressors in depressed individuals is entirely consistent with the phenomenon of a motivational deficit in depression. Depressed individuals show a deficit in approach-related behavior. For example, McFarland and Klein (2009) recently found that depressed individuals exhibited attenuated emotional reactivity to anticipated monetary rewards, but did not differ from non-depressed when they anticipated non-reward or punishment. In line with this evidence, Brinkmann and Gendolla (2008) could observe that depressive symptoms among otherwise healthy participants were associated with attenuated SBP reactivity in response to a difficult stress task but not in response to an easy task. The authors argued that individuals mobilize resources as long as success is possible and worthwhile. In the case of depression, negative mood functions as information for high task demand, resulting in effort deterioration and, correspondingly, lower SBP reactivity. Taken together, depressed individuals show an appetitive deficit in laboratory tasks and, consequently, may not invest much effort during active tasks, resulting in lower CVR.

1.1. Aim of the study

The aim of this study was to examine CVR in non-clinical individuals with varying depression scores to different laboratory stressors. In order to investigate the specificity of the findings with respect to stressor type in more detail, participants were faced with both active and passive aversive encounters. We implemented three different stressors. There was an active beta-adrenergic stressor (public speaking task), in which participants were instructed to prepare and deliver a speech within a social-evaluative context (similar to the public speaking task used by Salomon et al., 2009), and two different alpha-adrenergic passive stressors (a CP task and a video viewing task). We decided to implement two different passive stressors for the following reasons: first, previous studies largely neglected passive stressors to provoke CVR in non-clinical individuals with depressive symptoms. Hence, there is a need for research applying different passive stressors to gain a broader view of blunted CVR in mildly depressed individuals. Second, passive stressors usually are of little self-relevance, whereas active stressors are much more relevant to the self (e.g., via the evaluation of personal performance). Hence, when contrasting the role of stressor type to examine CVR there is a risk of confounding stressor type with self-relevance. Of note, personal relevance and negative self-views are a central feature of many theories of depression (e.g., Beck et al., 1979; Wisco, 2009). Thus, self-relevance could be more crucial for CVR as related to depressive symptoms than stressor type.

Taken together, we were interested to examine CVR to two passive tasks which differed with respect to self-relevance. The CP was chosen as a physically challenging task with little self-relevance. In order to contrast the CP with a passive self-relevant stressor, we additionally implemented a video viewing task in which participants were asked to watch the videotape of them presenting the speech. Thus, the video viewing task mirrored the speech task, but this time it was a passive task, requiring no effort allocation. In line with previous evidence we expected that depressive symptoms would be associated with blunted CVR to the active task (i.e., less effort allocation and approach behavior in depressives when active task performance is required), but not during the passive non self-relevant task (e.g., Salomon et al., 2009). Moreover, if self-relevance is the key to diminished CVR in depressive individuals, we would predict that depressive symptoms will also be related with attenuated reactivity to the viewing of the speech video, but not to the CP.

Importantly, besides the well-studied cardiovascular system we also opted for recording electrodermal reactivity (EDR). Of note, electrodermal hyporeactivity in depressed individuals has been reported in a number of previous studies (e.g., Dawson et al., 1977; Donat and McCullough, 1983; Greenfield et al., 1963; Iacono et al., 1983; Lader and Wing, 1969; McCarron, 1973; Noble and Lader, 1971; Thorell, 2009; Zuckerman et al., 1968). Similar to blood pressure, electrodermal activity is predominately influenced by sympathetic nerve fibers (e.g., Boucsein, 1992). Hence, these findings might be interpreted in terms of a more generalized sympathetic nervous system dysfunction in individuals with depressive symptoms.

Finally, we aimed to exploratively analyze physiological recovery. Of note, recent research suggests that especially cardiovascular recovery may be more important for physical health than peak reactivity to a challenge (Brosschot et al., 2006; Steptoe and Marmot, 2005). With respect to depression, the so-called perseverative cognition hypothesis (e.g., Brosschot, 2010; Brosschot et al., 2006) suggests that ruminating thoughts and worry may lead to sustained physiological activation, ultimately imposing risk for disease. However, recent research on diminished CVR in depression largely neglected recovery (e.g., Carroll et al., 2007; Phillips, 2011; York et al., 2007) or found no consistent association (Salomon et al., 2009), thus necessitating further research.

2. Methods

2.1. Participants

Fifty-five volunteers (34 females) participated in the study. They had a mean age of 22.95 years ($SD = 3.83$) and a mean BMI of 22.55 ($SD = 3.79$). Twenty-nine percent of the sample were smokers and 69% reported regular physical activity. All participants were free of cardioactive and antidepressive medications. They were not allowed to consume caffeine or cigarettes 2 h prior to the experiment. Participants were recruited through advertisements at the university campus and oral communication. They received course credit for participating.

2.2. Stress tasks

Three stress tasks were implemented to examine psychological and physiological reactivity. Throughout the stressor phases, the experimenter accompanied the participant in the room to provide ratings of affect and task performance. This procedure was implemented to enhance social-evaluative cues during task perfor-