Major Depressive Disorder Is Associated With Attenuated Cardiovascular Reactivity and Impaired Recovery Among Those Free of Cardiovascular Disease

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Objective: To examine cardiovascular reactivity and recovery to laboratory stress among a naturalistic sample of individuals diagnosed with major depressive disorder (MDD) and healthy control participants. Prospective evidence suggests that MDD confers risk for cardiovascular disease equal to or greater than the risk associated with depressed mood. Enhanced cardiovascular reactivity has been proposed as a mechanism explaining increased risk, but data are inconsistent as to whether depressed individuals exhibit enhanced or attenuated reactivity. Further, few studies have examined appraisal and recovery differences. Design: Participants diagnosed with MDD (N = 25) and healthy control participants (N = 25) engaged in a cardiovascular reactivity protocol including 2 tasks, each followed by a brief recovery period. Main outcome measures: Blood pressure, heart rate, pre-ejection period, cardiac output and total peripheral resistance were assessed. Appraisals of tasks were assessed prior to each task. Results: Depressed participants exhibited significantly less systolic blood pressure, heart rate, and cardiac output reactivity during speech, less heart rate reactivity during mirror tracing, and less heart rate recovery after speech and mirror tracing than controls. Depressed participants appraised the tasks as more demanding, threatening, and stressful and reported being less able to cope than controls. Appraisals were related to heart rate reactivity, but appraisals did not mediate the relationship between depression group and reactivity. Conclusion: Impaired recovery rather than exaggerated cardiovascular reactivity may partially explain the increased prospective cardiovascular disease risk in depressed individuals.

Keywords: depression, cardiovascular reactivity, recovery, cognitive appraisals.

Prospective evidence suggests that depression confers risk for the development of cardiovascular disease (CVD; Kuper, Marmot & Hemingway, 2002; Wuslin & Singal, 2003) with an overall relative risk of 1.64 (Rugulies, 2002). Much of this research has focused on depressed mood, but a few studies have shown that diagnosed major depressive disorder (MDD) is associated with at least equal to if not higher risk than that of depressed mood (Aromaa et al., 1994; Ford et al., 1998; Pratt et al., 1996). In a recent meta-analysis of prospective studies involving initially physically healthy samples, MDD was associated with a relative risk of 2.69 for the development of CVD whereas depressed mood was associated with a relative risk of 1.49 (Rugulies, 2002). Similarly, depressed mood has been prospectively linked to the development of hypertension (Jonas & Lando, 2000) and stroke mortality (Gump, Matthews, Eberly, Chang, & MRFIT Research Group, 2005).

Several mechanisms have been proposed to explain the increased CVD risk associated with depression including poorer health behaviors, obesity, hypothalamic-pituitary-adrenocortical axis hyperactivity, elevated catecholamine levels, and hypercoagulability (for a review, see Joyn, Whellan, & O’Connor, 2003). One proposal receiving research attention within the framework of the reactivity hypothesis (Krantz & Manuck, 1984; K. A. Matthews et al., 1986) is that depression is associated with exaggerated cardiovascular responses to laboratory stressors, which serves as a marker of CVD risk. However, the idea that depression is related to exaggerated cardiovascular reactivity (i.e., larger changes from resting to task) has not received consistent support. Although some investigations report a positive association between depressed mood and reactivity (e.g., S. C. Matthews, Nelesen & Dimsdale, 2005; Thornton & Hallas, 1999), other studies report no association (Guinjoan, Bernabo, 16& Cardinali, 1995; Taylor et al., 2006) and still others report either a negative association overall (e.g., Carroll, Phillips, Hunt, & Der, 2007; Straneva-Meuse, Light, Allen, Golding, & Girdler, 2004; York et al., 2007), or among subgroups (e.g., Delehanty, Dimsdale, & Mills, 1991; Knight & McCallum, 1998). A recent meta-analysis reported small, nonsignificant effect sizes linking depression to blood pressure reactivity and a significant modest effect size for heart rate (HR) reactivity (Kibler & Ma, 2004). However, a recent report based on over 1,600 adults showed...
that depressed mood was negatively related to systolic blood pressure and HR reactivity (Carroll et al., 2007).

Critically, inconsistency in prior findings is likely to reflect heterogeneity between studies. Prior studies vary along critical dimensions, such as whether the sample is free from CVD, or is selected on the basis of a dysphoric mood versus a diagnosis of MDD. The presence of CVD may alter cardiovascular reactivity through peripheral physiological mechanisms unrelated to psychosocial factors (Dimsdale, Siegler, Mills, Delahanty, & Berry, 1990; Lovallo & Gerin, 2003), and depressed mood has been shown to alter a number of physiological responses (Allen, Trinder, & Brennen, 1999; Dawson, Schell, & Catania, 1977). To provide evidence that exaggerated reactivity contributes to the prospective risk that MDD confers, medically healthy depressed individuals should exhibit exaggerated reactivity relative to healthy controls. To our knowledge only two studies have carefully excluded participants who are positive for a history of cardiovascular disease. One showed no differences in cardiovascular reactivity between MDD participants and healthy controls during autonomic function tests (Guinjoan et al., 1995). The other showed evidence that those with MDD exhibited attenuated reactivity (Straneva-Meuse et al., 2004), although all depressed participants were taking one of two specific antidepressants, so these results may not generalize to a more naturalistic sample. Therefore, one goal of the present study was to examine cardiovascular reactivity to two laboratory stressors in a naturalistic sample of participants with diagnosed depression and healthy controls with no self-reported history of CVD.

Perceived task difficulty is an important construct in the cardiovascular reactivity literature, and one that may be useful for explaining depression-related differences in reactivity and between-study heterogeneity. Depression involves changes in cognition that are likely to alter perceptions of demanding situations. For example, dysphoria and rumination, two common features of depression, have been related to perceiving stressors as severe and unsolvable as well as reduced self-confidence, optimism and perceived control (Lyubomirsky, Tucker, Caldwell, & Berg, 1999). Further, those in a negative mood, compared to those in a positive mood, exhibit greater reactivity when presented with easy tasks but less reactivity when presented with difficult tasks (Gendolla & Brinkmann, 2005). Thus, a second goal of the present study was to examine differences between depressed individuals and healthy controls in cognitive appraisals of laboratory stressors and if appraisal differences partially explain group differences in reactivity.

A third goal of the present study was to examine whether depressed persons exhibit impaired recovery from laboratory stressors. Impaired cardiovascular recovery has been identified as a CVD risk factor that is independent of cardiovascular reactivity (Hocking Schuler & O’Brien, 1997; Steptoe & Marmot, 2006). Incomplete recovery after stress predicts hypertension risk (e.g., Hocking Schuler & O’Brien, 1997; Steptoe & Marmot, 2005; Stewart, Janicki, & Kamarck, 2006; Trieber et al., 2001) and has been related to carotid intima-media thickness (Steptoe, Donald, O’Donnell, Marmot, & Deanefield, 2006). Perseverative cognitive styles associated with negative affect, such as worry and rumination, have been linked to delayed recovery from laboratory stressors (Brosschot, Gerin, & Thayer, 2006; Glynn, Christenfeld, & Gerin, 2002); in turn, high positive emotionality has been linked to accelerated cardiovascular recovery (Tügade & Frederickson, 2004). This is important because MDD is a disorder that is often characterized by excessive worry and rumination (Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001) and low positive emotionality (Kasch, Rottenberg, Arnow, & Gotlib, 2002). Thus the hypothesis that depression impairs cardiovascular recovery from stress is compelling.

Unfortunately, few studies have examined cardiovascular recovery in depressed individuals. Two studies found that depressed mood was related to higher cardiovascular levels across phases (i.e., baseline, tasks, recovery) but did not find interactions between phase and depression group, suggesting recovery did not differ by group (Hamer, Tanaka, Okamura, Tsuda, & Steptoe, 2007; S. C. Matthews et al., 2005). One study of a sample aged 55 and older at risk for CVD reported no differences in level across phases or interactions between diagnosed depressed and control groups (Taylor et al., 2006). More important, these prior studies did not examine recovery independent of baseline and reactivity, a limitation that the present study attempts to remedy.

Hypotheses

H1. We predicted that significant differences in reactivity would emerge between MDD participants and controls. Because the literature suggests that enhanced or diminished reactivity is possible in MDD, we did not make directional predictions.

H2. Two common features of depression, dysphoria and rumination, are related to perceiving stressors as severe and reduced self-confidence. Therefore, we expect that MDD participants will exhibit higher levels of threat, demand, and stress appraisals and report less ability to cope with the task than control participants.

H3. For those in a negative mood, appraisals are related to greater reactivity for easy tasks and less reactivity for difficult tasks. Therefore, we expect that cognitive appraisals will mediate the relationship between group (MDD and control) and reactivity.

Method

The study was conducted from January 2005 to May 2006 and was approved by the Social & Behavioral Sciences Institutional Review Board at the University of South Florida.

Participants and Clinical Assessment

Participants were 25 unipolar depressed persons and 25 healthy nonpsychiatric controls. Groups were matched on average age, self-reported ethnicity, gender, and income (see Table 1).

General selection criteria. Participants responded to local ads or flyers in the immediate campus area, including the waiting areas at the university counseling services centers. Ads targeted either depressed persons suffering currently from common depressive symptoms or healthy controls who had no history of psychiatric illness. Diagnostic evaluations were based on the Diagnostic and Statistical Manual of Mental Disorders (4th ed. [DSM–IV]; American Psychiatric Association, 1994) criteria and were determined by an initial telephone screening and the Structured Clinical Interview for DSM–IV Axis I (SCID–I; First, Gibbon, Spitzer, & Williams, 1995). All participants met the following inclusion and exclusion criteria: English fluency, aged 18 to 60; no reported history of brain injury; no lifetime history of primary psychotic ideation, no lifetime diagnoses of bipolar disorder, no behavioral
indications of impaired mental status, and no reported abuse of any psychoactive substances, including alcohol, within the past 6 months.

Exclusion of CVD was based on self-report by participants during initial screening and during the SCID–I interview. On screening, participants were asked whether they had a history of CVD, such as hypertension or heart attacks. During the SCID–I interview, participants’ medical history was extensively probed for past and present medical problems, including diagnoses, prescribed medications, and hospitalizations. Participants were excluded for a self-reported history of medication-dependent diabetes, heart disease, hypertension, mood episodes secondary to general medical conditions or medical conditions specific to the central nervous system, or current use of medications known to have significant effects on cardiovascular function (e.g., tricyclic antidepressants, antipsychotic agents, antihistamines, beta-blockers). Six participants reported use of oral contraceptives; all were in the control group. One control participant reported use of barbiturate medication for the treatment of a seizure condition. Nine depressed participants reported use of psychotropic medication.

Depressed and healthy control diagnostic criteria. The SCID–I interview (First et al., 1995) was conducted by clinical doctoral students to confirm that the depressed group met current diagnostic criteria for current MDD. Consistent with high levels of anxiety comorbidity reported elsewhere (Kessler, Chiu, Demler, & Walters, 2005), 17 MDD participants also met diagnostic criteria for at least one anxiety disorder. Fifteen participants met criteria for melancholic MDD and 4 met criteria for atypical MDD. The average duration of the current depressive episode was 24.3 months (SD = 36.6). Six MDD participants reported one episode of major depression, 3 reported two episodes, and 16 reported three or more episodes. Five MDD participants reported history of attempted suicide and 6 reported history of psychiatric hospitalization. The average age of onset was 18.8 years (SD = 9.6). Healthy control participants met the medical exclusion criteria and did not have any SCID–I assessed lifetime diagnoses of Axis I disorders.

Severity of depression and anxiety. The Beck Depression Inventory–II (BDI–II; Beck, Steer & Brown, 1996) and the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) were used to measure depression symptom severity and anxiety symptom severity, respectively. Both are 21 item self-report measures that have demonstrated validity and reliability (Beck et al., 1988; Beck et al., 1996).

Assessment of Cognitive Appraisals

We assessed pretask cognitive appraisals of demand, threat, stress, and ability to cope based on prior research suggesting that pretask appraisals are related to cardiovascular responses during laboratory reactivity tasks (Tomaka, Blascovich, Kelsey, & Leitten, 1993; Tomaka, Blascovich, Kibler, & Ernst, 1997). We generated four appraisal items: How demanding do you expect the upcoming task to be, how threatening (or intimidating) do you expect the upcoming task to be, how stressful do you expect the upcoming task to be, and how able are you to cope with the upcoming task. Responses ranged from (1) not at all to (5) very much on a 5-point Likert-type scale. We formed a challenge-threat ratio by dividing coping appraisal score by demand appraisal score. Items and scoring were virtually identical to those used in prior research (Tomaka et al., 1993, 1997).

Cardiovascular Measures

An Accutorr Plus blood pressure monitor (Datascope Corp., Mahwah, NJ) collected systolic (SBP) and diastolic (DBP) blood pressure according to published guidelines (Shapiro et al., 1990). HR was measured via electrocardiogram (ECG), using Cleartrace LT disposable Ag/AgCl electrodes (Conmed Andover Medical, Haverhill, MA), placed in a modified Lead II configuration on the chest. ECG was amplified using a Biopac MP150 system with an ECG100 amplifier (Biopac Instruments Inc., Goleta, CA). Impedance cardiography was collected according to the Sherwood et al. (1990) using four mylar-band electrodes fully encircling the neck and torso with the Biopac EBI100C monitor. ECG and impedance (Z0) signals were digitized, acquired, and stored using a PC and Biopac AcqKnowledge software.

Cardiovascular reactivity tasks. Two cardiovascular reactivity tasks were administered. A 2-min mirror tracing task required participants to trace the image of a star as quickly and accurately as possible while only seeing a mirror image of their hand and the star. The mirror tracing device (Stoelting Co., Chicago, IL) provided auditory feedback when the metal stylus lost contact with the star. A speech task required participants to prepare a speech on a specific topic (i.e., defending themselves against a traffic ticket), and to deliver the speech. The preparation and delivery phases of the speech task were each 2 min. To further increase evaluation apprehension during the speech task, an experimental observer was present in the room and silently took notes on the participant’s behavior. Tasks were followed by 2-min recovery periods, during which participants were instructed to sit quietly.

Procedure

Participants were first assessed for height and weight with a fixed steel tape and a beam scale. Waist circumference was mea-
sured in cm at the level of the umbilicus. Next, the experimenter attached the cardiovascular equipment. Participants were seated comfortably in a small recording room. The experimenter noted the presence of an unobtrusive, ceiling-mounted camera and informed the participants that they would be monitored throughout the protocol. Participants then viewed a neutral travelogue film for a 10-min acclimation and baseline assessment. The speech and mirror tracing tasks then were administered in counterbalanced order, separated by a 10-min rest period. After the second task, participants completed the BDI–II and the BAI, sensors were removed, and participants were paid, debriefed, and thanked.

Data Recording and Processing

BP recordings were taken during the 6th, 8th, and 10th minute of the 10-min rest period and ECG and impedance cardiography was recorded continuously during the last 5 min. BP was obtained during each minute of the speech preparation, speech, and mirror tracing tasks. During the recovery phases, one BP reading was recorded during the second minute. Impedance and ECG data were collected continuously during the task and recovery phases.

HR and the impedance-derived measures of pre-ejection period (PEP) and cardiac output (CO) were obtained using MindWare IMP 2.56 software (MindWare Technologies, Ltd., Gahanna, OH). The ECG and dZ/dt signals were ensemble-averaged over 60-s epochs. The data were screened for artifact by visual inspection. Mean arterial pressure (MAP) was calculated as (SBP + (2 * DBP))/3. Total peripheral resistance (TPR) was estimated using the formula TPR = (MAP(CO) * 80 in dyne-s/cm²).

Baseline, task, and recovery values for each measure were computed by averaging the available values for each phase, except for recovery BP, which included only one assessment. Reactivity scores were calculated as the arithmetic difference between task and baseline averages. Recovery scores were calculated as the arithmetic difference between recovery and baseline values, such that smaller values indicate greater recovery.

Data Analyses

We first examined baseline levels of SBP, DBP, and HR for significant group differences using analysis of covariance (ANCOVA) controlling for gender. To examine group differences in reactivity, we conducted ANCOVAs with group (control, MDD) as the between-subjects factor and the speech preparation, speech delivery, and mirror tracing task change scores as dependent measures, controlling for gender.

To examine group differences in appraisals, we conducted independent samples t tests with pretask appraisal scores as the dependent measures. To test for mediation, we followed steps outlined by Baron and Kenny (1986). We first identified which reactivity analyses and which appraisals t tests indicated a significant group difference. Next, we examined if appraisals were related to reactivity measures for the corresponding task. In cases in which all three conditions for mediation were met, we conducted hierarchical regression analyses regressing gender, group, and appraisal scores onto reactivity measures and examined if group continued to predict reactivity while controlling for appraisals.

To examine group differences in recovery, we conducted ANCOVAs on the recovery change scores while controlling for gender and corresponding reactivity change score. For the speech task, reactivity covariates were computed from averaged preparation and delivery values minus baseline averages. We examined if appraisals mediated the effect of group on recovery using the procedures outlined above. Slight variations in degrees of freedom reflect missing data for some cardiovascular variables due to inadequate signals from movement artifact.

Before testing the hypotheses, we first conducted preliminary analyses to examine the potential role of medication and anxiety comorbidity. Preliminary analyses of medications using only participants not taking psychiatric medication and the overall pattern of findings was very similar to those reported below. Similarly, preliminary analyses of anxiety were conducted using only participants without an anxiety disorder and again the overall pattern of findings was remarkably similar to those reported below. In light of these preliminary results, these variables were not considered further.

Results

Demographic and Physical Variables

Table 1 presents the demographic, physical and clinical characteristics of the sample. As reported elsewhere (Rottenberg, Clift, Bolden, & Salomon, 2007), the two groups did not differ on age, income, body mass index, or waist circumference. Depressed participants reported more depressive symptoms and more anxiety symptoms than healthy control participants.

Baseline Levels

A significant difference in resting HR between depressed participants and healthy controls emerged, F(1, 47) = 17.31, p < .001, η² = .269. Depressed participants exhibited a higher resting HR on average than control participants. As displayed in Table 2, no significant group differences in baseline levels of SBP or DBP were found.

Reactivity

For speech preparation, significant effects of group were found for SBP, F(1, 47) = 4.37, p < .05, η² = .085; HR, F(1, 46) = 4.64, p < .05, η² = .092; and CO, F(1, 46) = 10.69, p < .01, η² = .189 reactivity. Depressed participants exhibited less SBP, HR, DBP, and CO than control participants.

Table 2

<table>
<thead>
<tr>
<th>Measure</th>
<th>MDD (M) (SEM)</th>
<th>Control (M) (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>115.84 (1.96)</td>
<td>113.93 (1.96)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>70.94 (1.45)</td>
<td>69.12 (1.45)</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>80.63 (2.10)</td>
<td>68.21 (2.10)</td>
</tr>
</tbody>
</table>

Note. Numbers reported are covariate adjusted Means (M) and standard error of the mean (SEM). bpm = beats per minute.

*Significant effect of group, p < .05, using ANCOVA controlling for gender.
and CO reactivity than healthy controls. No other significant differences in speech preparation reactivity were found.

For speech delivery reactivity, significant effects of group were found for HR, $F(1, 47) = 12.37, p < .01, \eta^2 = .208$; and CO, $F(1, 47) = 4.80, p < .05, \eta^2 = .093$ reactivity. Depressed participants exhibited less HR and CO reactivity. For mirror tracing reactivity, a significant effect of group was found for HR reactivity, $F(1, 46) = 6.15, p < .05, \eta^2 = .118$. No other significant differences in mirror tracing reactivity were found. Figure 1 presents means for reactivity by group.

*Figure 1.* Mean reactivity and recovery change scores by group and phase. (Note: Significant effect of group indicated by * $p < .05$ or ** $p < .01$ using ANCOVA. Bars reflect ± 1 standard error of the mean. Means for speech preparation, speech, and mirror are adjusted for gender. Means for recovery phases are adjusted for gender and task reactivity. $\Delta$ = change in; bpm = beats per minute; L/min = liters per minute; dyne-sec/cm$^5$ refers to peripheral resistance units.)
Table 3 reports the means and standard deviations for task appraisal items and raw challenge-threat ratios. Depressed participants exhibited less HR recovery after the tasks compared to healthy controls. No significant effects were found for other cardiovascular recovery variables. The means for recovery are presented in Figure 1. Also, pretask appraisals were not related to HR recovery for either the speech or mirror tracing tasks (ps > .36), controlling for gender and reactivity. Therefore, appraisals could not mediate the effect of group on recovery.

Discussion

We sought to examine differences in sympathetically mediated cardiovascular reactivity and recovery between individuals diagnosed with MDD and healthy controls. The present study is unique in that we compared a group with diagnosed MDD to a virtually symptom-free group in a community-based sample. Further, in contrast to most prior studies, our participants were screened for the absence of CVD, allowing us to examine depression and reactivity in relatively healthy sample. Our results indicate that depressed persons exhibit less SBP, HR, and CO reactivity than controls. These findings are consistent with a growing number of studies showing attenuated reactivity as a function of depressed mood (e.g., Carroll et al., 2007; York et al., 2007) or of MDD (Straneva-Meuse et al., 2004). They are also consistent with our previous analysis of respiratory sinus arrhythmia (RSA) that demonstrated that depressed participants exhibited less RSA reactivity and impaired RSA recovery compared to controls (Rotenberg et al., 2007). However, these findings are inconsistent with the hypothesis that depression confers risk for CVD through exaggerated cardiovascular reactivity.

Given the finding that depression confers risk for CVD, and the now replicated finding that depression can attenuate reactivity in some contexts, it is tempting to speculate that attenuated reactivity is a pathway that explains depressed persons’ elevated risk for CVD. McEwen (1998) proposed that multiple patterns of stress responsivity reflect allostatic load, and thus, disease risk. One suggested pattern is nonresponsiveness in one physiological stress system that may indicate compensatory responses in other physiological stress systems (McEwen, 1998). Allostatic load is an attractive idea in the context of MDD, as individuals with this condition suffer from chronic depressed mood and a host of other physical and psychological symptoms. The attenuated reactivity associated with depression may reflect the nonresponse pattern of allostatic load, one that indicates an overactivation of the hypothalamic-pituitary-adrenocortical axis or other compensatory responses. For example, depression has been associated with chronic activation of the hypothalamic-pituitary-adrenocortical axis (Joynt et al., 2003). Thus, one may tentatively speculate that the attenuated cardiovascular reactivity seen among depressed individuals serves as a marker for CVD risk through compensatory mechanisms. The tenability of this hypothesis remains to be tested.

Mood may serve as a source of information when appraising task demand. When faced with a difficult task, those in a negative mood may perceive the demands of a task as too high for their abilities and may not mobilize effort resulting in attenuated cardiovascular reactivity for difficult tasks (Gendolla & Brinkmann, 2005). Accordingly, depressed participants appraised the upcoming tasks as more demanding and stressful, perceived that they were less able to cope, and perceived the task as less challenging.
than healthy controls. Further, greater perceived demand and threat, and lesser perceived coping ability and challenge appraisal were related to less HR reactivity. These findings resonate with findings from nonclinical samples indicating that challenge appraisals are associated with enhanced cardiac reactivity (Tomaka et al., 1993, 1997). More important, these appraisals did not mediate the relationship between depression and reactivity. Thus, the attenuated reactivity seen in MDD may be due to physiological factors or to psychological factors other than appraisals.

We also examined recovery from the tasks and found that depressed individuals exhibited less HR recovery compared to controls. Although depressed participants exhibited less reactivity and a higher resting HR, both of which would require less of a decrease in HR for recovery, they continued to exhibit elevated HR during the recovery period. Impaired recovery has been identified as a CVD risk factor that is independent of reactivity (Hocking Schuler & O’Brien, 1997; Steptoe & Marmot, 2006). Specifically, impaired HR recovery has been related to lack of physical fitness (Hocking Schuler & O’Brien, 1997) a positive family history of hypertension (Trieber et al., 2001), and has been found to predict increases in resting blood pressure (Steptoe & Marmot, 2005) and HR (Stewart et al., 2006). Further, inadequate recovery is also commensurate with the idea of allostatic load, as a prolonged response pattern (McEwen, 1998). To our knowledge, we are the first to report impaired cardiovascular recovery among medically healthy diagnosed depressed individuals. Our recovery findings suggest that depression may confer CVD risk through impaired recovery, independent of reactivity. As others have suggested (e.g., Brosschot et al., 2006) impaired recovery may be more pernicious for the course of disease given that these cardiovascular elevations persist longer than acute stress reactivity. Further, blunted reactivity followed by impaired recovery has been linked to chronic stress (K. A. Matthews, Gump, & Owens, 2001), suggesting that MDD serves as a chronic stressor or is associated with ongoing background stress. This suggests that impaired recovery is an epiphenomenon of blunted reactivity that may, as a specific response pattern, indicate risk. Perhaps those who are chronically stressed lack the psychological or physiological means to mount a large reactivity response, but perseverative processes then cause the response to persist after the stressor itself has ended. Although compelling, further research is needed to determine whether impaired recovery and blunted recovery are independent or reflect a coupled response pattern.

Depression was not related to BP recovery, although many studies point to delayed BP recovery as an indicator of risk (Hocking Schuler & O’Brien, 1997; Steptoe & Marmot, 2005, 2006; Stewart et al., 2006; Trieber et al., 2001). However, our recovery period was relatively short, lasting only 2 min. HR may recover more quickly than BP as HR is under both sympathetic and parasympathetic control and parasympathetic control has near instantaneous effects on HR (Levy, Yang, & Wallick, 1993). Further, vagal rebound occurs quickly after cessation of a stressor, suggesting the predominance of parasympathetic mechanisms (Mezzacappa, Kelsey, Katkin, & Sloan, 2001; Rottenberg et al., 2007). Thus, the recovery period may have been too short to allow for variability in recovery of other cardiovascular responses.

The present study is not without limitations. The sample size was relatively small, partially a function of the challenge of recruiting participants who met diagnostic criteria for MDD and were in a current depressive episode, a condition that affects 10% of adults in any given year (Kessler et al., 1994). Also, it should be borne in mind that the absence of CVD was established via self-report methods only. Although this practice is commensurate with prior work (e.g., Light et al., 1998), we cannot rule out the possibility that our study may have included participants with undiagnosed or misreported CVD. Mitigating this concern, the negative predictive validity of self-reported CVDs is generally quite high; with commonly reported estimates ranging from 89% to 99% (Martin, Leff, Calonge, Garrett, & Nelson, 2000; St. Sauver et al., 2005). Given that this sample contained many younger participants, the self-reported absence of CVD is likely to accurately reflect not being diagnosable.

The naturalistic design of the study precluded a conclusive analysis of medication or comorbid anxiety disorder as moderators of the reported findings. A fair proportion of the MDD participants were taking one or more medications and small cell sizes precluded analysis of individual medications. Similarly, a proportion of the MDD participants had comorbid anxiety disorders, which were again heterogeneous in the type and number. We conducted parallel analyses focusing only on unmedicated participants and on nonanxious participants. Many of the effects remained statistically significant despite the reduced power (data not reported). Further, the pattern of findings was the same; MDD participants exhibited higher resting HR, attenuated reactivity, and impaired recovery relative to healthy controls. Thus, despite the limitations of a naturalistic design, there were no indications that the reported effects were driven by medication or the presence of comorbid anxiety.

As noted earlier, few studies have examined reactivity among individuals with diagnosed MDD. Our recruitment strategy excluded participants with minor or subclinical depression as evidenced by the range of BDI scores among our healthy and depressed groups. This prevented examination of reactivity and recovery across a range of depressed mood. Future research would benefit from explicit comparison of psychiatrically healthy, mildly depressed, and MDD diagnosed participants. Also, a longer posttask period would allow for a more detailed analysis of recovery. Finally, we did not assess participants’ reports of rumination or positive affect during recovery, which could prove useful for explaining impaired recovery in the MDD group.

MDD may confer prospective risk for CVD through impaired recovery from stress. If prolonged poststress activation is the result of psychological processes associated with depression, treatments aimed at reducing rumination and increasing positive affect may ameliorate this risk. The blunted reactivity exhibited by depressed individuals may also indicate risk if the nonresponse pattern represents allostatic load that signals compensatory responses in other physiological systems. It may also indicate that depressed participants do not always exhibit exaggerated responses to stress if the stressor is perceived as overly demanding and threatening and that impaired recovery may be a more consistent marker of risk.

References


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**Call for Nominations: Health Psychology**

Division 38 (Health Psychology) is currently accepting nominations for the editorship of *Health Psychology* for the years 2011-2016. Robert M. Kaplan is the incumbent Editor.

Candidates should be members of Division 38 and of APA, and should be available to start receiving manuscripts in 2010 to prepare issues to be published in 2011. Division 38 encourages participation by members of underrepresented groups and would welcome such nominees. Self-nominations are also encouraged.

Kevin D. McCaul, Ph.D., has been appointed as Chair for this search.

To nominate candidates, prepare a statement of two pages or less in support of each candidate, and provide a current CV. Submit all materials electronically to: apadiv38@verizon.net.

The deadline for receipt of nominations is April 15, 2009.