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Symptoms of Depression and Cardiovascular Reactions to Acute Psychological Stress: Evidence from a Population Study

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Running Head: Depression and Cardiovascular Reactivity

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Abstract

Depression and exaggerated cardiovascular reactivity are considered risk factors for cardiovascular disease, possibly as a result of common antecedents, such as altered autonomic nervous system function. We examined the association between depressive symptomatology and cardiovascular reactions to psychological stress in 1608 adults (875 women) comprising three distinct age cohorts: 24-, 44-, and 63-year olds. Depression was assessed using the Hospital Anxiety and Depression Scale. Blood pressure and heart rate were measured at baseline and during the paced auditory serial arithmetic test. Depression scores were negatively associated with systolic blood pressure and heart rate reactions, after adjustment for likely confounders such as sex, cohort, occupational status, body mass index, stress task performance score, baseline cardiovascular activity, antidepressant and antihypertensive medication. The direction of association was opposite to that which would be expected if excessive reactivity were to mediate the association between depression and cardiovascular disease outcomes or if they shared common antecedents.

Key words: Blood pressure; depression; heart rate; psychological stress

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1. Introduction

Depression has been linked prospectively to mortality in general and death from cardiovascular disease in particular (for reviews, see Hemingway & Marmot, 1999; Wulsin, Vaillant, & Wells, 1999). However, the mechanisms underlying this association have yet to be established. Candidate mediators or confounders include: socio-economic position; ill-health and disability; and unhealthy behaviours, such as smoking, and generally poorer self-care (Wulsin, Vaillant, & Wells,1999). At a physiological level, increased platelet aggregation (Mikuni, Kayaga, Takahashi, & Meltzer, 1992) and exaggerated cardiovascular reactions to psychological stress exposure (Kibler & Ma, 2004) have been implicated. It is the latter which is the focus of the current analyses.

Depression has been associated with a variety of physiological adaptations that suggest altered autonomic function. For example, enhancement of cardiac sympathetic activity relative to vagal tone has been reported in those with depression and subclinical depressive symptoms (Carney, Rich, TeVelde, Saini, Clark, & Freedland, 1988; Light, Kothan, Dapani, & Allen, 1998), as have increased plasma noradrenalin concentrations in patients with major depression (Rudorfer, Ross, Linnoila, Sherer, & Potter, 1985). Thus, the hypothesis that such autonomic dysregulation in depression may also be manifest as exaggerated cardiovascular reactivity which in turn increases the risk of cardiovascular pathology is intuitively appealing. First, exaggerated cardiovascular reactions to acute psychological challenge have long been considered a risk factor for cardiovascular pathology (Lovallo & Gerin, 2003; Schwartz, Gerin, Davidson, Pickering, Brosschot, Thayer, Christenfeld, & Linden, 2003) and several prospective studies have now shown consistently that high reactivity confers a modest additional risk for a range of cardiovascular outcomes, such as high blood pressure, carotid atherosclerosis, carotid intima-thickness, and increased left ventricular mass (e.g. Allen, Matthews, & Sherman, 1997; Barnett, Spence, Manuck, & Jennings, 1997; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Kamarck, Everson, Kaplan, Manuck, Jennings, Salonen, & Salonen, 1997; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Treiber, Kamarck, Scneiderman, Sheffield, Kapuku,

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& Taylor, 2003). Second, there would appear to be at least some provisional evidence that symptoms of depression may be associated with heightened reactivity. A meta-analysis of 11 relevant studies found small to moderate effect sizes indicative of a positive relationship between depressive symptomatology and cardiovascular reactions to acute psychology stress (Kibler & Ma, 2004). Unfortunately, none of the aggregate effects were statistically significant at conventional levels. Previous studies generally tested fairly small samples and were often conducted on patients with established cardiovascular disease. Further, few of these studies adjusted for potential confounding variables such as demographic factors and medication status.

This underscores the need to examine the association between depressive symptomatology and cardiovascular stress reactivity in a large representative sample. The present study, then, re-visited this association in a substantial and demographically diverse sample of participants. In addition, statistical adjustment for a range of possible confounders was possible. It was hypothesized that symptoms of depression would be positively correlated with the magnitude of cardiovascular reactions to stress.

2. Methods

2.1. Participants

Participants were all from Glasgow and the surrounding areas in Scotland. As part of the West of Scotland Twenty-07 Study, they have been followed up at regular intervals since the baseline survey in 1987 (Ford, Ecob, Hunt, Macintyre, & West, 1994). The full sample size at entry to the study was 3036. The data reported here are from the third follow-up when cardiovascular reactions to an acute psychological challenge were measured (Carroll, Harrison, Johnston, Ford, Hunt, Der, & West, 2000; Carroll, Ring, Hunt, Ford, & MacIntyre, 2003). Reactivity data were available for 1647 participants, with scores for depression and anxiety symptomatology recorded for 1608 of these respectively; this, then, constituted the effective study sample. Participants comprised three distinct age cohorts: 575 (36%) 24-year olds, 606 (38%) 44-year olds, and 427 (26%) 63-year olds, 875 (54%) were women and 733 (46%) men, and 746 (47%) were

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from manual and 851 (53%) non-manual occupation households. The mean age of the whole sample was 42.3 (SD = 15.48) years and mean body mass index was 25.7 (SD = 4.27) kg/m^2.

2.2. Apparatus and procedure

Participants were tested in a quiet room in their own homes by trained nurses. Demographic information was obtained by interview. Household occupational status, an accepted index of socio-economic position, was classified as manual or non-manual from the occupational status of the head of household, using the Registrar General's ("Classification of Occupations"1980) classification of occupations. For the youngest of the three cohorts, head of household was either the participant, if working and living independently, or the parent, if the participant was a student or lived with their parents. For the other two cohorts, head of household was either the participant or his/her spouse/partner, depending on which of the two held or had held the highest occupational status; the was usually the man. Height and weight were measured and body mass index computed.

Depression and anxiety were measured using the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983). The HADS is a well-recognised assessment tool that comprises 14 items, seven measuring depression and seven measuring anxiety. The depression subscale emphasises anhedonia and largely excludes somatic items. Items are scored on a 4-point scale, 0 to 3; the higher the score, the greater the depression and anxiety. The HADS has good concurrent validity (Bramley, Easton, Morley, & Snaith, 1988; Herrmann, 1997), performs well as a psychiatric screening device (Bjelland, Dahl, Haug, & Neckelmann, 2002; Herrmann, 1997), and boasts acceptable psychometric properties; for example, a Cronbach's α of .90 for the depression items and .93 for the anxiety items has been reported (Moorey, Greer, Watson, Gorman, Rowden, Tunmore, Robertson, & Bliss, 1991) and test-retest reliability coefficients as high as .85 for depression and .84 for anxiety have been found (Herrmann, 1997).

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At the end of the session, participants undertook an acute psychological challenge: the paced auditory serial addition test (PASAT), which has been shown in numerous studies to reliably perturb the cardiovascular system (Winzer, Ring, Carroll, Willemsen, Drayson, & Kendall, 1999) and to demonstrate good test-retest reliability (Willemsen, Ring, Carroll, Evans, Clow, & Hucklebridge, 1998). Participants were presented with a series of single digit numbers by audiotape and requested to add sequential number pairs while retaining the second of the pair in memory for addition to the next number presented, and so on throughout the series. Answers were given orally and, if participants faltered, they were instructed to recommence with the next number pair. The correctness of answers was recorded as a measure of performance. The first sequence of 30 numbers was presented at a rate of one every four seconds, and the second sequence of 30 at one every two seconds. The whole task took three minutes, two minutes for the slower sequence and one minute for the faster sequence. Only participants who registered a score on the PASAT were included in the analyses. Out of a possible score of 60, the median score was 45 (Inter-quartile range = 11).

Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were determined by an Omron (model 705CP) sphygmomanometer. This is one of the semi-automatic blood pressure measuring devices recommended by the European Society of Hypertension (O'Brien, Waeber, Parati, Staessen, & Myers, 2001). Following interview, (which took at least an hour), there was then a formal 5-minute period of relaxed sitting, at the end of which a resting baseline reading of SBP, DBP, and HR was taken. Task instructions were then given and the participant allowed a brief practice to ensure that they understood task requirements. Two further SBP, DBP, and HR readings were taken during the task, the first initiated 20 seconds into the task (during the slower sequence of numbers), and the second initiated 110 seconds later (at the same point during the fast sequence). For all readings, the nurses ensured that the participant's elbow and forearm rested comfortably on a table at heart level. The two task readings were averaged and the resting baseline value subsequently subtracted from the resultant average task value to yield reactivity measures for SBP, DBP, and HR for each participant.

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2.3. Statistical analyses

Given the sizeable co-variation between depression and anxiety scores, r (1600) = .60, p < .001, analyses focused primarily on depression. This high co-variation is a common finding; the mean correlation between depression and anxiety from 18 studies, with a combined N = 8160, was reported to be 0.63 (Herrmann, 1997). The main analyses testing the association between depression and cardiovascular reactivity applied multiple linear regression; reactivity was the dependent variable throughout. In the first models tested, various likely covariates of reactivity, cohort, sex, household occupational condition, body mass index, performance score on the PASAT, and baseline cardiovascular levels, were entered at step 1. Depression score was then entered at step 2. In the second set of models tested, the same variables were entered at step 1, but medication status (taking or not taking antidepressives or anxiolytics) was entered at step 2, with depression now entered at step 3. Analogous analyses were conducted for anxiety, but will only be reported summarily. Data on anxiety was lost for six participants and this is reflected in the slightly reduced degrees of freedom on occasion.

3. Results

3.1. Sociodemographics and depression

The mean depression score for the sample as a whole was 3.65 (SD = 2.86); the analogous summary statistic for anxiety was 7.19 (SD = 3.81). ANOVA yielded main effects for all three independent varables: the older two cohorts recorded higher depression scores than the youngest cohort, F (2,1591) = 14.61, p < .001, η^2 = .018; women displayed higher scores than men, F (1,1591) = 7.63, p = .006, η^2 = .005; those from manual occupational households had higher scores than those from non-manual households, F (1,1591) = 7.10, p = .008, η^2 = .004. The summary statistics are presented in Table 1.

[Insert Table 1 about here]

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Analyses of the anxiety data also revealed significant main effects for cohort, $F(2,1585) = 4.61$, $p = .007$, $\eta^2 = .006$, sex, $F(1,1585) = 41.05$, $p < .001$, $\eta^2 = .025$, and occupational status, $F(1,1585) = 4.30$, $p = .04$, $\eta^2 = .003$. As with depression, women and those in the manual occupational group reported more symptoms of anxiety than men and those in the non-manual. However, in the case of anxiety, the oldest cohort registered lower scores than the other two cohorts.

3.2. Cardiovascular reactions to acute psychological stress

These data are summarized in Table 2. Two-way (baseline × task) repeated measures ANOVAs indicated that the PASAT successfully perturbed cardiovascular activity: for SBP, $F(1,1607) = 1564.08$, $p < .001$, $\eta^2 = .493$, for DBP, $F(1,1607) = 1048.71$, $p < .001$, $\eta^2 = .395$, and for HR, $F(1,1607) = 1108.77$, $p < .001$, $\eta^2 = .408$. ANCOVAs, with PASAT score and baseline cardiovascular levels as covariates, were applied to the reactivity scores. There were main effects of cohort, $F(2,1589) = 42.47$, $p < .001$, $\eta^2 = .051$, and sex, $F(1,1589) = 52.91$, $p < .001$, $\eta^2 = .032$, on SBP reactivity; reactivity increased with age and men exhibited larger reactions than women. Analogous main effects for cohort, $F(2,1589) = 19.47$, $p < .001$, $\eta^2 = .024$, and sex, $F(1,1589) = 14.75$, $p < .001$, $\eta^2 = .009$, were apparent for DBP reactivity. In addition, there was also an effect of occupational status, $F(1,1589) = 4.00$, $p = .04$, $\eta^2 = .003$; those from non-manual occupational households exhibited larger DBP reactions. For HR, there were main effects for cohort, $F(2,1589) = 18.92$, $p < .001$, $\eta^2 = .023$, and occupational status, $F(1,1589) = 5.87$, $p = .01$, $\eta^2 = .004$; HR reactivity declined with age and was greater in those from non-manual households. The summary data are presented in Table 3. In all these analyses, baseline cardiovascular levels exerted significant effects on the dependent variables: $F(1,1589) = 115.55$ to 194.95, $p < .001$, $\eta^2 = .080$ to $.109$. In the case of SBP and HR reactivity, PASAT score also had an influence: $F(1,1589) = 30.19$, $p < .001$, $\eta^2 = .019$ and $F(1,1589) = 57.27$, $p < .001$, $\eta^2 = .035$, respectively.

[Insert Tables 2 and 3 about here]

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3.3. Depression and cardiovascular reactivity

The bivariate associations between depression and anxiety, and cardiovascular reactivity are displayed in Table 4. Negative associations were observed between depression and anxiety, and SBP and HR reactivity: the higher the depression or anxiety score, the lower the reactivity. Subsequent hierarchical linear regression analyses, adjusting for cohort, sex, household occupational status, body mass index, performance score on the PASAT, and baseline cardiovascular levels, confirmed a negative association between SBP reactivity and HADS depression scores. A similar negative association emerged for HR reactivity and depression. The relevant statistics for these models are summarized in Table 5. In these multivariate analyses, HADS anxiety scores predicted only HR reactivity; again the relationship was a negative one, $\beta = -.04$, $t = 2.02$, contribution to $R^2 = .002$, $p = .04$. Only 71 (4%) and 46 (3%) of the sample reported taking reported taking antidepressives and anxiolytics. The negative associations between depressive symptomatology and cardiovascular reactivity were not attenuated following subsequent additional adjustment for whether or not participant were taking antidepressive medication, $\beta = -.06$, $t = 2.43$, contribution to $R^2 = .003$, $p = .02$ for SBP reactivity, and $\beta = -.05$, $t = 2.02$, contribution to $R^2 = .002$, $p = .04$ for HR reactivity, respectively: nor, in the case of SBP reactivity, following further adjustment for the cohort × sex and cohort × occupational status interactions, $\beta = -.06$, $t = 2.47$, contribution to $R^2 = .003$, $p = .01$. However, with additional adjustment for whether or not participants reported taking anxiolytics, the association between HADS anxiety score and HR reactivity became marginally significant, $\beta = -.05$, $t = 1.93$, contribution to $R^2 = .002$, $p = .05$. One hundred and forty four (9%) of the sample reported taking antihypertensive medication. The associations between depression and reactivity were also not attenuated with further adjustment for antihypertensive use; for SBP reactivity, $\beta = -.06$, $t = 2.53$, contribution to $R^2 = .004$, $p = .01$ and for HR reactivity, $\beta = -.05$, $t = 2.00$, contribution to $R^2 = .002$, $p = .04$.

[Insert Table 4 and 5 about here]

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Finally, a cut-off of ≥ 8 on the depression subscale of the HADS was employed as an indicator of possible pathology (Zigmond & Snaith, 1983). ANOVA revealed that those with probable pathology (N = 180) had marginally lower SBP reactivity, mean = 10.1, SD = 11.75 mmHg than without *prima facie* evidence of pathology, mean = 11.7, SD = 11.71 mmHg, $F(1,1606) = 3.01$, $p = .08$, $\eta^2 = .002$, and significantly lower HR reactivity, mean = 6.3, SD = 9.09 versus mean = 8.4, SD = 9.89 bpm, $F(1,1606) = 7.45$, $p = .006$, $\eta^2 = .005$.

4. Discussion

The mean HADS depression and anxiety scores for participants in the present study are broadly similar to those reported by others in large non-clinical adult samples (Crawford Crawford, Henry, Crombie, & Taylor, 2001). Depression scores were higher in women, participants from manual occupational households, and those in the middle and oldest cohorts. Variations in depression and depressive symptomatology with sex (e.g. Maier, Gansicke, Gater, Rezaki, Tiemens, & Florezano Urzua, 1999; Piccinelli & Wilkinson, 2000; Weissman, Bland, Joyce, Newman, Wells, & Wittchen, 1997) and socioeconomic status (e.g., Bruce, Takeuchi, & Leaf, 1991; Dohrenwend et al., 1992; Stansfeld, Head, Fuhrer, Wardle, & Cattell, 2003) are well documented. However, previous data on age and depression are inconsistent. Although studies have observed an increase in depression in the elderly (Kessler, Foster, Webster, & House, 1992; Mirosky & Reynolds, 2000), others report a negative relationship between age and depression and symptoms of depression (Charles, Reynolds, & Gatz, 2001; Scheiman, Van Gundy, & Taylor, 2002; Turner & Noh, 1988). It has been argued the former can be attributed to age variations in chronic health and that normally functioning older adults are at no greater risk for depression than younger adults (Roberts, Kaplan, Shema, & Strawbridge, 1997). It is worth noting here that age effect in the present study was abolished when self-reported disability status was taken into account (analysis not reported). Anxiety levels varied similarly with sex and occupational status: again results not without precedent (e.g. Fryers, Melzer, & Jenkins, 2003; Reich, 1986). In the case of HADS

anxiety, levels declined with age; this has also been reported by others (e.g. Weissman & Merikangas, 1986).

As indicated in earlier reports from this study (Carroll, Phillips, Ring, Der, & Hunt, 2005; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Phillips, Carroll, Hunt, & Der, 2006; Phillips, Carroll, Ring, Sweeting, & West, 2005), the acute stress was successful in perturbing cardiovascular activity. In addition, in line with the outcomes of previous analyses of these data, socio-demographic factors influenced the magnitude of reactions to stress (Carroll, Harrison, Johnston, Hunt, Der, & West, 2000). Blood pressure reactivity increased with age whereas HR reactivity decreased. Men tended to be more reactive than women and those from manual occupational households were less reactive that those from non-manual households. This latter result runs contrary to the notion that those from lower socioeconomic strata, who are at increased risk of cardiovascular disease (Hein, Suadicani, & Gyntelberg, 1992; Marmot, Shipley, & Rose, 1984; Rosengreen, Wedel, & Wilhelmsen, 1988), will exhibit more detrimental cardiovascular adaptations to stress exposure (Strike & Steptoe, 2004). It very much in line, however, with results from an earlier analysis of cardiovascular reactivity in a substantial occupational cohort; the magnitude of SBP reactions to an acute mental stress task was positively associated with occupational status among public servants (Carroll, Davey Smith, Sheffield, Shipley, & Marmot, 1997).

The major aim of the present analyses was to examine the relationship between symptoms of depression and cardiovascular reactions to acute psychological stress. HADS depression scores were negatively associated with both SBP and HR reactivity. Those with higher symptom levels that exhibit reduced reactivity of parameters presumed to reflect sympathetic nervous system activation. That the associations appeared for SBP and HR, but not DBP, reactivity implicates cardiac rather than vascular activation. The present associations, although statistically significant, were small in size. Nevertheless, they withstood adjustment for a range of possible confounders. For example, one very parsimonious explanation for the direction of relationship observed in the present study is

that participants registering high HADS depression scores were relatively de-motivated and simply engaged less with the stress task. This would be reflected in a poorer performance score. However, although both depression, $r(1606) = -.11$, $p < .001$, and anxiety, $r(1605) = -.09$, $p < .001$, scores correlated negatively with PASAT performance, adjustment for performance did not attenuate the association between depression and cardiovascular reactivity. Further, in analyses in which those with very low (<15) PASAT scores were eliminated, the associations between depression and reactivity remained. In contrast, the negative association between HADS anxiety scores and HR reactivity was reduced to marginal statistical significance following adjustment for potential confounders, including anxiolytic medication.

The direction of the relationship between depressive symptomatology and cardiovascular reactivity was contrary to expectations based on the reactivity hypothesis and previous research. However, few studies have directly or even indirectly addressed this particular issue. Those that have generally suffered from low power ($N < 100$), which can increase the likelihood of committing type 1 as well as type 11 errors (Oakes, 1987). In addition, few previous studies adjusted for or were able to adjust for potential confounders. Importantly, closer inspection reveals that most of these small scale previous studies yielded statistically null results; indeed, as indicated, the aggregate effect sizes calculated from 11 previous studies of depression and SBP, DBP, and HR reactivity were non-significant (Kibler & Ma, 2004). It would appear that a hypothesis can be so intrinsically compelling that it transcends the need for a strong and consistent evidential base. The two studies which provide at least indicative evidence of an association between depression and reactivity were conducted on coronary artery disease patients (Sheffield, Krittayaphong, Cascio, Light, Golden, Finkel, Glekas, Koch, & Sheps, 1998; Thornton & Hallas, 1999). However, in one of these studies (Sheffield, Krittayaphong, Cascio, Light, Golden, Finkel, Glekas, Koch, & Sheps, 1998), those in the low depression half of the sample of 41 patients were more likely to be taking beta-blockers, which can attenuate cardiac reactions to acute psychological stress exposures (Winzer, Ring, Carroll, Willemsen, Drayson, & Kendall, 1999). In the present study, we additionally adjusted

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for antihypertensive medication status. In the other study (Thorton & Hallas, 1999), positive relationships between HADS depression and anxiety and cardiovascular reactivity emerged for the 30 myocardial infarction patients tested with self-initiated reactivity during ambulatory recording but not during laboratory testing with standard stress tasks. It is also possible that the associates of risk vary between the largely healthy community samples and groups of patients with confirmed disease (Bennett & Carroll, 1990). It is worth noting that high levels of depressive and related symptomatology are particularly prevalent in those with established coronary heart disease (Lane, Carroll, Ring, Beevers, & Lip, 2002).

The present study suffers from a number of limitations. First, it was cross-sectional and, accordingly, causality is impossible to determine. Since affective disposition was assessed prior to stress testing, it is difficult to see how it might be influenced by reactivity rather than *vice versa*. Nevertheless, some unanalyzed confounding variable might drive the relationship observed. However, as indicated, the association between depression and SBP and HR reactivity survived adjustment for the most obvious candidates. Second, as also indicated, the effect sizes were small. This, though, was our *a priori* expectation based on previous research and reinforces the value of large samples were examining some of the more subtle correlates of cardiovascular reactivity. Third, although performance on the stress task seems a reasonable proxy for task engagement, in hindsight it would have been useful to have self-report measures of task impact. Fourth, it is difficult to discern from the present data whether these associations concern contemporary dysphoria or more chronic depressive disposition. However, the relationships between depression and reactivity were also evident in analyses comparing those above and below the usual HADS cut-off for likely caseness. This suggests that the present associations cannot be wholly attributed to variations in transient dysphoria. Finally, only blood pressure and HR were measured. Although, it would have been useful to have a more comprehensive assessment of haemodynamics of the sort afforded by impedance cardiography, the large sample and the decision to test participants in their

homes precluded this. In this context, it is worth noting that most of the previous studies have similarly restricted their focus to blood pressure and heart rate.

This is the first population study we know of to examine the relationship between symptoms of depression and cardiovascular reactions to acute psychological stress. Depressive symptomatology was related to SBP and HR reactivity, and relationships were still evident following adjustment for possible confounders such as sex, age, occupational status, resting cardiovascular activity, task performance, and antidepressant and antihypertensive medication. However, the direction of these associations was opposite to that which would be expected if depression and excessive reactivity shared common antecedents or if excessive reactivity were to mediate the association between depression and cardiovascular disease outcomes. In conclusion, it would appear that as putative risk factors for cardiovascular disease, high levels of depressive symptomatology and exaggerated cardiovascular reactions to stress may operate independently of one another.

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Table 1. Mean (SD) HADS depression and anxiety scores by cohort, sex, and occupational status.

		Depression	Anxiety
Age Cohort:	Youngest	3.1 (2.60)	7.3 (3.78)
	Middle	3.9 (3.00)	7.4 (3.88)
	Eldest	4.0 (2.94)	6.7 (3.71)
Sex:	Male	3.5 (2.71)	6.5 (3.56)
	Female	3.8 (2.98)	7.7 (3.93)
Occupational Group:	Manual	3.9 (2.96)	7.3 (3.98)
	Non-manual	3. 5 (2.77)	7.1 (3.65)

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Table 2. Mean (SD) SBP, DBP, and HR, baseline and reactivity values for the sample as a whole.

SBP		DBP		HR	
Baseline	*Reactivity*	*Baseline*	*Reactivity*	*Baseline*	*Reactivity*
129.1 (20.50)	11.6 (11.72)	78.9 (11.60)	7.0 (8.61)	66.7 (10.80)	8.2 (9.82)

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Table 3. Mean (SD) SBP, DBP, and HR reactions by cohort, sex, and occupational status

		SBP		DBP		HR	
		Baseline	*Reactivity*	*Baseline*	*Reactivity*	*Baseline*	*Reactivity*
Age Cohort:	Youngest	120.0 (14.99)	10.2 (10.05)	73.4 (10.10)	6.8 (8.98)	67.6 (11.06)	10.0 (10.60)
	Middle	127.2 (18.13)	12.2 (11.41)	80.7 (11.17)	7.1 (8.06)	66.6 (11.08)	7.8 (10.04)
	Eldest	144.4 (21.66)	12.4 (13.92)	83.8 (11.16)	7.0 (8.91)	65.7 (9.97)	6.0 (7.73)
Sex:	Male	134.9 (18.27)	12.9 (11.72)	81.4 (11.12)	7.1 (8.40)	64.7 (10.43)	8.8 (9.83)
	Female	124.3 (21.05)	10.4 (11.61)	76.8 (11.61)	6.8 (8.79)	68.4 (10.83)	7.6 (9.80)
Occupational Group:	Manual	130.5 (21.40)	11.3 (12.13)	79.4 (11.93)	6.6 (9.02)	66.9 (11.23)	7.0 (9.56)
	Non-manual	127.9 (19.65)	11.7 (11.35)	78.5 (11.31)	7.3 (8.23)	66.5 (10.43)	9.2 (9.94)

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Table 4: Bivariate correlations between depression and anxiety, and cardiovascular reactivity

	Depression	**Anxiety**
SBP Reactivity	-.053*	-.049*
DBP Reactivity	-.016	-.005
HR Reactivity	-.105**	-.068**

*$p < .05$, ** $p < .01$

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Table 5. Summary of hierarchical regression outcomes indicating a negative association between depression and SBP and HR reactivity.

	β	t	*p*	ΔR^2
SBP Reactivity				
Step 1:				
Sex	-.18	7.08	<.001	
Cohort	.25	9.30	<.001	
BMI	-.03	1.19	.23	
Occupational Group	-.01	0.20	.84	
Baseline SBP	-.33	11.45	<.001	
PASAT score	.13	5.49	<.001	.115
Step 2:				
Depression	-.07	2.70	.007	.004
HR Reactivity				
Step 1:				
Sex	.00	0.16	.87	
Cohort	-.12	5.10	<.001	
BMI	-.14	6.01	<.001	
Occupational Group	-.06	2.73	.006	
Baseline HR	-.28	12.18	<.001	
PASAT score	.17	7.23	<.001	.172
Step 2:				
Depression	-.05	2.17	.03	.002

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