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**Body mass index, abdominal adiposity, obesity and cardiovascular reactions to psychological stress in a large community sample.**

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Body mass index, abdominal adiposity, obesity and cardiovascular reactions to psychological stress in a large community sample.

Douglas Carroll, Anna C. Phillips, Geoff Der

## **Abstract**

**Objective:** Previous studies, although far from conclusive, imply a positive association between adiposity and the magnitude of cardiovascular reactions to acute psychological stress. The present analyses revisited this issue both cross-sectionally and prospectively in a large community sample, with greater power to detect relationships and more scope to control for possible confounders.

**Methods:** Blood pressure and heart rate were measured at rest and in response to a brief time-pressured mental arithmetic stress in 1647 adults. At the same session and five years later, height, weight, waist and hip circumference were measured and body mass index and waist-hip ratio computed. Obesity was defined as a body mass index  $\geq 30\text{kg/m}^2$ .

**Results:** Contrary to expectations, the most robust and consistent results to emerge from cross-sectional analyses were negative associations between all three measures of adiposity and heart rate reactivity; those with greater body mass and waist-hip ratios and those categorized as obese displayed smaller heart rate reactions to stress. In prospective analyses, high heart rate reactivity was associated with a reduced likelihood of becoming obese in the subsequent five years.

**Conclusions:** Our analyses suggest that it is low not high heart rate reactivity that is related to adiposity. Low heart rate reactivity, probably by reflecting generally blunted sympathetic nervous system reactions to challenge, may be a risk marker for developing obesity.

**SBP** = Systolic Blood Pressure, **DBP** = Diastolic Blood Pressure, **HR** = Heart Rate, **PASAT** = Paced Auditory Serial Addition Test.

It is now recognized that obesity in Western countries has reached epidemic proportions (1). Longitudinal cohort studies in the United States (2-4), the United Kingdom (5), and elsewhere (6, 7) testify to an inexorable rise in adiposity. The health consequences are unlikely to be trivial. Obesity, defined in terms of body mass index, has been consistently linked to all-cause and especially cardiovascular disease mortality (8-11). Excess adiposity is also associated with a range of cardiovascular and metabolic disease outcomes, such as type 2 diabetes (12, 13) and hypertension (14, 15), as well as overall cardiovascular disease morbidity (16, 17). Attention has also been paid to the health consequences of the location of fat deposition, and in particular to abdominal adiposity, measured as waist circumference or waist-hip ratio (18). Evidence now suggests that abdominal fat can predict cardiovascular and metabolic disease outcomes independently of generalized obesity, as well as providing additional risk information to that afforded by body mass index (14, 19-22).

Adiposity, and particularly abdominal adiposity, has been linked with psychological distress and it has been argued that an increased vulnerability to stress in the abdominally obese may be manifest as hyper-reactivity (23). Through their impact on the neuroendocrine system, stress exposures have been postulated to promote abdominal fat deposition (24). As a corollary, it has been hypothesized that obesity, and especially central adiposity, will be associated with exaggerated cardiovascular reactions to stress (25, 26). Exaggerated cardiovascular reactions to acute psychological stress have long been considered a risk factor for cardiovascular pathology (27). Several prospective studies have now shown with reasonable consistency that high reactivity confers a modest additional risk for a range of cardiovascular outcomes, including high blood pressure, carotid atherosclerosis, carotid intima thickness, and increased left ventricular mass (28-34). The question arises as to whether adiposity and exaggerated cardiovascular reactivity are positively related or whether they are independent risk factors for cardiovascular pathology.

A handful of mainly small scale studies have attempted to address this issue. In an early study of 20 healthy young men, systemic vascular resistance levels during mental stress were found to be negatively correlated with body mass index, the higher the index the lower the resistance, but positively associated with waist-hip ratio, the higher the ratio the higher the resistance; no significant associations emerged for blood pressure or cardiac activity during stress (35). From a study of 95 adolescents, it was reported that the peak systolic blood pressure (SBP) reaction to mental stress was larger for participants in the upper tertile of waist-hip ratios; neither cardiac nor resistance reactions were associated with abdominal adiposity (36). Waist circumference has been reported to be positively associated with heart rate (HR) and diastolic blood pressure (DBP) reactivity in a sample of 22 older African American men, although the association with HR reactivity was no longer significant following adjustment for basal blood pressure and the association with DBP reactivity following adjustment for basal blood pressure and insulin level (26). In a contemporary study of 24 women with body mass indices  $\geq 28 \text{ kg/m}^2$ , those with abdominal obesity, i.e. high waist-hip ratios, had higher DBP and systemic resistance reactions, but lower HR reactions, to a speech task (25). Higher waist circumference has also been associated with higher SBP reactions to four stress tasks in a bi-racial sample of 211 adolescent boys and girls and with higher DBP reactions in the 104 boys; no associations emerged for HR (37). In the largest study to date, body mass index was not significantly related to cardiovascular reactivity in 225 middle-aged public servants, although waist-hip ratio was positively associated with diastolic reactivity; the greater the abdominal adiposity, the higher the reactivity (38). In addition, the upward drift in body mass index and waist-hip ratio over a 3-year follow-up period was not associated with the earlier measures of cardiovascular reactivity. Finally, the change in cardiovascular reactivity following a successful 12-week weight loss intervention did not differ between the intervention and control groups (39).

It is difficult to draw firm confident conclusions from the results of these studies. The most consistent result appears to be a positive association between systemic resistance reactivity, as reflected by DBP and/or total peripheral resistance, and abdominal

adiposity, although not all studies report this. In addition, with two exceptions (37, 38), the sample sizes in previous studies were small. Samples were also, for the most part, poorly representative of the general population. Finally, again with one exception (38), these analyses adjusted for few or no possible confounding variables, including baseline cardiovascular status. Accordingly, it would seem opportune to revisit this issue in a study with greater power to detect relationships and greater scope to control for possible confounders. The present analyses explored the association between cardiovascular reactivity and adiposity, both cross-sectionally and prospectively, in a large and diverse community sample. It was hypothesized that, at least cross-sectionally, greater abdominal adiposity would be associated larger DBP reactions to mental stress. With regard to the prospective outcomes, disturbances in cardiovascular reactivity may reflect autonomic dysfunction which in turn may contribute to the development of obesity and adiposity (38). Accordingly, we anticipated that, on balance, higher reactivity may be associated with a greater likelihood of becoming obese.

## **Method**

### ***Participants***

Data were collected as part of the West of Scotland Twenty-07 Study. Participants were all from Glasgow and surrounding areas in Scotland, and have been followed up at regular intervals since the baseline survey in 1987 (40). The Study's principle aim was to investigate the processes that generate and maintain socio-demographic differences in health (41). Participants were chosen randomly with probability proportional to the overall population of the same age within a zip code area (42). Thus, the sample was selected as a clustered random stratified sample; three narrow age cohorts (aged 15, 35, and 55 years at entry) were chosen so that age specific effects could be estimated with greater precision than an all age sample of the same size. The specific ages were chosen to reflect important stages of life and transitions: the transition from youth to adulthood and from education to employment in the case of the youngest cohort; from middle age to old age and from working to retirement for the oldest cohort. The middle cohort would



be experiencing changes in family structure, approaching their peak occupational attainment and the age at which differences begin to emerge in risk of premature death (41). The achieved sample sizes in 1987 were 1009, 985, and 1042 for the age 15, 35, and 55 cohorts, respectively. A comparison of these samples with equivalent samples drawn from the 1991 UK census revealed equivalence in terms of sex, occupational group, and home ownership (43). The sample was almost entirely Caucasian, reflecting the West-of-Scotland population from which it was drawn. The data reported here are from the third and fourth follow-ups. The mean (SD) temporal lag between follow-ups was 5.5 (1.00) years. At the third follow-up, cardiovascular reactions to an acute psychological stress task were measured (30, 44). Data were available for 1647 participants, following exclusion of physiologically impossible outlying blood pressure values and attrition, which was largely a result of relocation. The sample at this time point comprised 592 (36%) 24-year olds, 624 (38%) 44-year olds, and 431 (26%) 63-year olds. There were 890 (54%) women and 757 (46%) men, and 772 (47%) were from manual and 870 (53%) from non-manual occupation households. Household occupational group data were not available for five participants. Overall mean age at the third follow-up was 41.8 (SD = 15.44) years. The mean (SD) ages of the young, middle aged, and older cohorts were 23.7 (0.56) 44.1 (0.85), and 63.1 (0.67) years, respectively. Body mass index and waist-hip ratio data were available at both follow-ups. The attrition rate was 23%; data were available for 1272 participants at the later follow-up. This study was approved by the appropriate Ethics committees.

### ***Apparatus and procedure***

Participants were interviewed in a quiet room in their homes by trained nurses. At the third follow-up, household occupational group was classified as manual or non-manual from the occupation of the head of household, using the Registrar General's Classification of Occupations (45). Head of household was usually the man. Participants were classified as never, ex-, or current smokers by their response to a simple prompt. They then undertook an acute psychological stress task: the paced auditory serial addition test (PASAT), which has been shown in numerous studies to reliably perturb the

cardiovascular system (46, 47) and to demonstrate good test-retest reliability (48). The nurses were all trained in the PASAT protocol by the same trainer. They followed a written protocol during every testing session and noted any deviations from protocol, e.g., participant gave up on the test. Such deviations were rare and the data were excluded from the analyses. The task comprised a series of single digit numbers presented by audiotape and participants were 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. A brief practice was given to ensure that participants understood the requirements of the task. Only those who registered a score on the PASAT were included in the analyses. Out of a possible score of 60, the mean score was 40.9 (SD = 9.03).

SBP, DBP and HR were determined by an Omron (model 705CP) sphygmomanometer. This semi-automatic blood pressure measuring devices is recommended by the European Society of Hypertension (49). A larger blood pressure cuff was used for participants with larger arm circumferences in order to ensure accurate measurement. Following interview, (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, followed by the brief practice. 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 faster 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.

At both the third and fourth follow-ups, height, using the Leicester Height Measure stadiometer, and weight, using portable electronic scales (Soehnle, Nassau, Germany), were measured and body mass index computed. The standard criterion of  $\geq 30 \text{ kg/m}^2$  was used to identify obesity. Waist circumference was measured over light indoor clothes at the point midway between the iliac crest and inferior margin of the last rib. Hip circumference was measured at the widest point over the buttocks and below the iliac crest. Waist-hip ratio was then computed.

### ***Statistical analyses***

Initially, repeated measures analysis of variance (ANOVA) was used to compare baseline with task SBP, DBP, and HR levels. Further repeated measures ANOVAs, again using baseline and task values, were undertaken to compare variations between age cohorts, sexes, and household occupational groups. ANOVA and  $\chi^2$  were applied to examine the socio-demographic patterning of body mass index and obesity. Analysis of the body mass index, waist-hip ratio and reactivity was by hierarchical linear regression. The effects of obesity on cardiovascular reactivity were tested using analysis of covariance (ANCOVA). In ANOVA and ANCOVA, partial  $\eta^2$  was the measure of effect size. In both the regression analyses and ANCOVA of reactivity, baseline cardiovascular activity was always entered as a covariate. Change scores have long been recognised as an appropriate means of characterising reactivity (50). Further, in large scale studies (e.g., (51), baseline has been observed to co-vary negatively with reactivity and resting blood pressure is observed to be higher in obese individuals (14, 15). In the present data, SBP baseline was negatively correlated with SBP reactivity,  $r(1645) = -.19, p < .001$ , DBP baseline was negatively correlated with DBP reactivity,  $r(1645) = -.29, p < .001$ , and HR baseline was negatively correlated with HR reactivity,  $r(1645) = -.29, p < .001$ .

Subsequent regression and ANCOVA models were tested to determine whether any effects that emerged from the primary analyses withstood adjustment for potential

confounding variables. The possible confounders selected were age cohort, sex, occupational status, performance scores on the PASAT, whether or not participant were taking blood pressure lowering medication, and smoking status. All have been associated with reactivity and/or adiposity in previous published research (44, 47, 52-54). Slight variations in the degrees of freedom reflect occasional missing data for some variables. Finally, regression analyses were applied to determine whether cardiovascular reactivity predicted changes in body mass index, waist-hip ratio, and obesity between follow-ups. For the continuous variables, linear regression was applied entering the adiposity variable value at the earlier follow-up at step 1 and reactivity at step 2; the later follow-up values were the dependent variables. For the binary variable, logistic regression was applied, examining whether reactivity predicted whether or not participants had become obese in the interim, or had remained or become non-obese; those who had remained obese were excluded for the purposes of such analyses. As all the models tested were relatively simple, i.e. with few predictors relative to large sample size, the chances of over-fitting, with resulting poor predictive validity, are extremely low.

## Results

### *Cardiovascular reactivity*

The stress task increased cardiovascular activity: for SBP,  $F(1,1646) = 1562.32, p < .001, \eta^2_p = .487$ , for DBP,  $F(1,1646) = 1066.62, p < .001, \eta^2_p = .393$ , and for HR,  $F(1,1646) = 1132.96, p < .001, \eta^2_p = .408$ . As can be seen from Table 1, HR reactivity declined with age,  $F(2,1644) = 21.11, p < .001, \eta^2_p = .025$ ; with the youngest cohort exhibiting higher reactivity than the middle cohort who, in turn, showed higher reactivity than the eldest cohort ( $p < .05$  in each case). Heart rate reactivity was also greater in men,  $F(1,1645) = 5.23, p = .02, \eta^2_p = .003$ , and in participants from non-manual occupational status households,  $F(1,1640) = 21.08, p < .001, \eta^2_p = .013$ . SBP reactivity varied significantly among the age cohorts,  $F(2,1644) = 6.81, p = .001, \eta^2_p = .008$ , with the youngest cohort having significantly lower reactivity than the other two cohorts ( $p < .05$  in both cases). Women had smaller SBP reactions than men,  $F(1,1645) = 16.61, p < .001, \eta^2_p = .010$ . DBP reactivity did not vary significantly with age cohort, sex, or occupational class. The

statistics reported above relate to significant group by baseline/task condition interactions. However, for the sake of illustration, we report baseline and reactivity in Table 1.

[Insert Table 1 about here]

### ***Body mass index, abdominal adiposity, and obesity***

At the earlier follow-up the younger cohort had lower body mass indices than the other two cohorts,  $F(2,1641) = 30.76, p < .001, \eta^2_p = .036$ . Body mass index did not differ significantly between the sexes or between occupational class groups. Younger participants,  $F(2,1641) = 29.83, p < .001, \eta^2_p = .035$ , women,  $F(1,1642) = 1277.78, p < .001, \eta^2_p = .438$ , and those from non-manual occupational households,  $F(1,1637) = 7.35, p < .001, \eta^2_p = .004$ , had smaller waist-hip ratios. Two hundred and twenty five (14%) of the participants met the criterion for obesity. Proportionally less of the younger cohort were identified as obese,  $\chi^2(2) = 12.45, p = .002$ . The frequency of obesity did not vary significantly by sex or occupational class. The summary statistics are presented in Table 2.

Body mass index increased across the five years between follow-ups,  $F(1,1272) = 342.12, p < .001, \eta^2_p = .212$ , as did waist-hip ratio,  $F(1,1295) = 58.69, p < .001, \eta^2_p = .042$ . The respective mean (SD) values were 25.6 (4.12) kg/m<sup>2</sup> and 26.9 (4.43) kg/m<sup>2</sup> and 0.86 (0.08) and 0.87 (0.08). At the later follow-up, a higher proportion, 261 (21%), of participants met the criterion for obesity,  $\chi^2(2) = 504.84, p < .001$ . The variations in body mass index, waist-hip ratio and obesity among demographic groups at the third follow-up were largely preserved at the fourth follow-up. Body mass index increased over time, with the youngest cohort showing the greatest increase (1.90 kg/m<sup>2</sup>) and the oldest cohort registering a slight decrease (-0.43 kg/m<sup>2</sup>),  $F(2,1270) = 32.31, p < .001, \eta^2_p = .048$ . This increase in body mass index did not vary with sex, but those from non-manual occupational class households (1.39 kg/m<sup>2</sup>) showed a slightly greater upward drift than those from manual occupational households (1.05 kg/m<sup>2</sup>),  $F(1,1266) = 6.58, p = .01, \eta^2_p$

= .005. The youngest cohort showed the greatest increase in waist-hip ratio over time (0.02) and the oldest cohort showed the least (0.01),  $F(2,1293) = 5.34, p = .005, \eta^2_p = .008$ . The change in waist-hip ratio was not associated with occupational class, but was greater for women (0.02) than for men (0.01),  $F(1,1294) = 5.86, p = .02, \eta^2_p = .005$ .

[Insert Table 2 about here]

### ***Body mass index, abdominal adiposity, obesity and resting cardiovascular activity***

Body mass index was positively associated with resting SBP,  $r(1642) = .28, p < .001$ , and DBP,  $r(1642) = .32, p < .001$ , but not with resting HR. Waist-hip ratio was also positively associated with resting SBP,  $r(1642) = .29, p < .001$ , and DBP,  $r(1642) = .26, p < .001$ . In contrast, there was a small but statistically significant negative association between waist-hip ratio and resting HR,  $r(1642) = -.06, p = .01$ . Participants identified as obese had higher resting SBP,  $F(1,1642) = 53.71, p < .001, \eta^2_p = .032$ , DBP,  $F(1,1642) = 62.17, p < .001, \eta^2_p = .036$ , and HR,  $F(1,1642) = 6.28, p = .01, \eta^2_p = .004$ . The respective mean (SD) resting values for the three parameters for obese and non-obese participants were: 138.2 (19.35) and 127.6 (20.28) mmHg for SBP; 84.4 (10.67) and 78.0 (11.50) mmHg for DBP; 68.4 (10.22) and 66.4 (10.89) bpm for HR. These outcomes reinforce the necessity of controlling for resting baseline when analysing these reactivity data.

### ***Body mass index, abdominal adiposity, obesity, and cardiovascular reactivity: cross-sectional analyses***

In hierarchical regression models in which resting baseline was entered at step 1 and body mass index at step 2, body mass index was positively associated with DBP reactivity,  $\beta = .06, t = 2.37, p = .02, \Delta R^2 = .003$ , but negatively associated with HR reactivity,  $\beta = -.17, t = 7.22, p < .001, \Delta R^2 = .028$ . Body mass index and SBP reactivity were not significantly related. Waist-hip ratio was also negatively associated with HR reactivity,  $\beta = -.11, t = 4.54, p < .001, \Delta R^2 = .011$ , but positively related to both SBP,  $\beta = .14, t = 5.53, p < .001, \Delta R^2 = .018$ , and DBP,  $\beta = .10, t = 4.13, p < .001, \Delta R^2 = .009$ ,

reactivity. ANCOVA, again adjusting for resting baseline, comparing obese and non-obese revealed that obese participants had smaller SBP,  $F(1,1641) = 6.44, p = .01, \eta^2_p = .004$ , and HR,  $F(1,1641) = 21.31, p < .001, \eta^2_p = .013$ , reactions to the stress task. There were no effects of obesity on DBP reactivity. The respective mean (SD) values for the SBP, DBP, and HR reactivity for obese and non-obese participants were: 8.7 (12.47) and 11.9 (11.63) mmHg; 6.0 (9.27) and 7.1 (8.52) mmHg; 5.0 (8.53) and 8.6 (9.88) bpm.

### ***Analyses controlling for possible confounders***

In the present study, age cohort, sex, and occupational class all influenced reactivity, as well as indices of adiposity. Further, the performance score on the stress task was positively associated with both SBP,  $r(1645) = .12, p < .001$ , and HR reactivity,  $r(1642) = .32, p < .001$ . Although not associated with waist-hip ratio, poorer performance was associated with a higher body mass index,  $r(1642) = -.07, p = .007$ . Obese participants tended to perform more poorly on the stress task than their non-obese counterparts,  $F(1,1642) = 3.31, p = .07, \eta^2_p = .002$ ; the respective mean (SD) performance scores were 42.7 (9.58) and 43.9 (9.14). In addition, participants who were taking blood pressure lowering medication had greater body mass indices (mean = 27.2, SD = 4.86 versus mean = 25.5, SD = 4.16),  $F(1,1640) = 20.02, p < .001, \eta^2_p = .012$ , and greater waist-hip ratios, (mean = 0.88, SD = 0.08 versus mean = 0.86, SD = 0.09),  $F(1,1640) = 14.50, p < .001, \eta^2_p = .009$ . Obese participants,  $N = 33$  (15%), were also more likely to be taking anti-hypertensive medication than their non-obese counterparts,  $N = 108$  (8%),  $\chi^2(1) = 12.68, p = .001$ . Further, those on medication exhibited lower HR reactivity,  $F(1,1643) = 5.58, p = .02, \eta^2_p = .003$ ; the respective mean (SD) reactivity values were 6.3 (8.82) and 8.3 (9.87). Current smokers had significantly lower body mass indices than ex- or never smokers,  $F(2,1640) = 25.35, p < .001, \eta^2_p = .030$ , and proportionally fewer of them were obese,  $\chi^2(2) = 18.28, p < .001$ . In contrast, participants who reported never smoking had less abdominal adiposity than those who smoked or were ex-smokers,  $F(2,1640) = 10.40, p < .001, \eta^2_p = .013$ . The summary statistics are presented in Table 3.



In the analyses which simultaneously adjusted for age cohort, sex, occupational group, PASAT performance scores, medication, and smoking status, as well as baseline cardiovascular levels, the positive association between body mass index and DBP reactivity was no longer statistically significant, although the negative association with HR reactivity was,  $\beta = -.15$ ,  $t = 6.25$ ,  $p < .001$ ,  $\Delta R^2 = .020$ . The negative association between waist-hip ratio and HR reactivity remained significant,  $\beta = -.09$ ,  $t = 4.09$ ,  $p < .001$ ,  $\Delta R^2 = .009$ , but the positive relationships with blood pressure reactivity did not. Obese participants still exhibited much smaller HR reactions to stress than their non-obese counterparts,  $F(1,1627) = 16.71$ ,  $p < .001$ ,  $\eta^2_p = .010$ , but not smaller SBP reactions. In order to illustrate which covariates were largely responsible for abolishing the significant positive associations between body mass index and DBP reactivity, and waist-hip ratio and SBP and DBP reactivity, we present the full regression summaries in Table 4. Aside from  $\beta$ , we also report the  $R^2$  for the model with just the covariates entered, and then the full model with the independent variable added.

[Insert Tables 3 and 4 about here]

### ***Sensitivity analyses***

Participants were also split into three groups to determine whether those with normal body masses ( $< 25 \text{ kg/m}^2$ ), differed in HR reactivity from those who were overweight but not obese ( $>25$  but  $< 30 \text{ kg/m}^2$ ), and whether the overweight in turn differed from the obese. This issue was addressed by ANCOVA using the fully adjusted model. There was a negative linear relationship between body mass status and HR reactivity,  $F(2,1603) = 14.94$ ,  $p < .001$ ,  $\eta^2_p = .018$ , which is illustrated in Figure 1.

[Insert Figure 1 about here]

### ***Body mass index, abdominal adiposity, obesity and cardiovascular reactivity: prospective analyses***



Reactivity did not predict the rise in either body mass index or waist-hip ratio in the five years between the third and fourth follow-ups. However, HR reactivity did predict whether participants became obese or remained or became non-obese, OR (95%CI) = 0.97 (0.95 – 0.99),  $p = .02$ ; high HR reactivity was associated with a reduced likelihood of becoming obese. The respective mean (SD) reactivity values for those who became obese and those who remained or became non-obese were 6.8 (9.09) and 9.2 (10.04) bpm. This analysis was repeated adjusting for all the previous possible confounders (age cohort, sex, occupational group, PASAT performance scores, medication, and smoking status). The association between becoming obese or not and HR reactivity was unchanged, OR (95%CI) = 0.97 (0.95 – 0.99),  $p = .01$ . Finally, we also examined this issue using an analysis with obesity at the fourth follow-up as the outcome and obesity at the third follow-up as a covariate. The result remained unchanged; HR reactivity predicted whether or not participant were obese at the fourth follow-up, taken into account their obesity status at the earlier follow-up, OR (95%CI) = 0.98 (0.96 – 0.99),  $p = .008$ .

## Discussion

Participants with large body mass indices and waist-hip ratios were characterized by higher resting blood pressure levels and those defined as obese not only had relatively high resting blood pressure, but also higher resting HRs. These results are not without precedent. Numerous studies now attest that adiposity and obesity are positively related to resting blood pressure (14, 15); in addition, modestly higher basal HRs in obese versus lean participants has been reported in some, although not most, studies (55). However, in contrast to expectations based on previous research, the most consistent and robust findings from the present cross-sectional analyses of reactivity were negative associations between measures of adiposity and HR reactivity; obese participants exhibited smaller HR reactions to the stress task than their non-obese counterparts and the greater the body mass index and waist-hip ratio, the lower the HR reactivity. Although positive associations emerged between body mass index and waist-hip ratio and blood pressure

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reactivity, these did not withstand adjustment for possible confounders in multivariate regression models.

The present results for cardiovascular reactivity are seemingly at odds with those from previous studies (25, 26, 35-38). Although painting a far from consistent picture, in general these studies suggest that abdominal adiposity, in particular, is positively related to reactivity, especially for parameters such as DBP that reflect stress-induced changes in systemic vascular resistance. However, with one exception (38), these studies either tested small numbers of participants or samples that were not representative of the general population and hardly, if at all, adjusted for possible confounders. In contrast, the present sample was large, recruited using established sampling techniques, and representative of the community, West of Scotland, from which it was drawn. Further, the importance of covariate adjustment is demonstrated clearly in the present study where the positive associations between body mass index and waist-hip ratio and blood pressure reactivity were abolished with control for a range of candidate confounders. Only one study to date has examined the prospective association between reactivity and change in adiposity over time; no association emerged between the 3-year upward drift in body mass index and waist-hip ratio and earlier cardiovascular reactivity (38). In contrast, in the present study HR reactivity predicted which participants were more likely to have become obese over the subsequent five years. In line with the direction of the cross-sectional associations between heart reactivity and measures of adiposity, high HR reactivity appeared protective, reducing the likelihood of becoming obese.

Sympathetic nervous system blockade studies indicate that cardiac reactivity in the context of mental stress reflects  $\beta$ -adrenergic activation (47, 56). Substantial research has now been directed at the role of the sympathetic nervous system in adiposity and obesity (57). A complex, although still incomplete, story is beginning to emerge. For example, although data are scarce, there is evidence that, at least in genetically susceptible populations, low sympathetic nervous system activity, as indexed by muscle sympathetic nerve activity, may be associated with weight gain, abdominal adiposity and the

pathogenesis of obesity (58). The present prospective association between low HR reactivity and an increased likelihood of becoming obese is certainly in line with this notion. In the absence of data, we are reluctant to speculate why a sympathetic nervous system that is less responsive to challenge may confer risk for developing obesity. However, others have postulated reduced sympathetic nervous system activity may increase food intake (58), as has been demonstrated in rodents (59, 60).

On the other hand, there is reasonably consistent evidence that once individuals have become obese, they are characterized by high basal sympathetic nervous system activity (57). This may be regionally specific, though, with the renal and skeletal muscle circulations, but not the cardiac circulation, being targets for increased basal sympathetic tone (61). For example, muscle sympathetic nervous system activity shows a strong positive association with adiposity and obesity (62, 63). Further, higher renal but lower cardiac indicators of sympathetic nervous system activity (nor-epinephrine spill-over and regional oxygen consumption) have been reported in obese normotensives (64). However, there is also evidence from studies of HR variability suggesting a shift in sympatho-vagal balance in obese individuals; an increase in the low to high frequency ratio suggests sympathetic nervous system predominance (65 – 67). In contrast, there is emerging evidence that whereas sympathetic tone may be elevated in obese individuals in the resting state, their sympathetic nervous systems may be less responsive to stimulation. For example, there is evidence of a postprandial sympathetic nervous system response, as indicated by higher plasma nor-epinephrine concentrations and an increased low to high frequency ratio in the HR variability spectrum after ingestion of a meal (66, 67). However, this latter effect was reported to be significantly smaller in obese versus lean individuals (66). Importantly, the changes in HR and muscle sympathetic nerve stimulation following infusion of anti-hypertensive and anti-hypotensive drugs were significantly smaller in the obese than the non-obese (62). Our findings for HR reactivity, then, are what would be expected if a high body mass index, abdominal adiposity, and obesity are associated with generally blunted sympathetic nervous system responses to challenge.

The present study is not without limitations. First, we measured only blood pressure and HR. It could have proved instructive to have the sort of comprehensive assessment of haemodynamics afforded by impedance cardiography. This would have allowed us to determine the origins of variations in heart rate reactivity. Although an increase in afterload as a consequence of vascular reactivity must remain a possibility, it is more likely that heart reactivity with the present stress exposure is, as we infer above, a function of increased sympathetic drive to the heart. Whatever the case, the decision to test participants in their own homes and the size of the sample precluded the use of impedance cardiography. Second, performance on the stress task was adopted as a measure of task engagement. Although this seems reasonable, in retrospect it might have proved useful to have included self-report measures of stress task impact. Nevertheless, performance score correlated with HR reactivity in the expected direction. Third, determining causality is impossible from cross-sectional study and still fraught with pitfalls in prospective analyses (68). However, we did adjust statistically for a broad range of potential confounders. Nevertheless, residual confounding as a consequence of poorly measured or un-measured variables cannot be wholly discounted.

This is the first large scale community study to examine the association between body mass index, abdominal adiposity, obesity and cardiovascular reactions to acute psychological stress. High body mass index, abdominal adiposity and obesity were all negatively associated with HR reactivity in cross-sectional analyses; the lower the reactivity, the greater the adiposity. In prospective analyses, low HR reactivity was associated with increased risk of becoming obese over a five year period. On balance, then, our analyses suggest that low HR reactivity, probably by reflecting generally blunted sympathetic nervous system reactions to a variety of acute challenges, may be a risk marker for developing obesity.

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Table 1.

Mean (SD) SBP, DBP, and HR baseline and reactivity by age cohort, sex, and occupational status

	<i>SBP</i>		<i>DBP</i>		<i>HR</i>	
	<i>Baseline</i>	<i>Reactivity</i>	<i>Baseline</i>	<i>Reactivity</i>	<i>Baseline</i>	<i>Reactivity</i>
<i>Age Cohort:</i>						
Youngest (N = 592)	120.0 (15.07)	10.1 (10.24)	73.4 (10.08)	6.8 (9.04)	67.5 (11.00)	10.0 (10.56)
Middle (N = 624)	127.1 (18.08)	12.3 (11.44)	80.6 (11.13)	7.1 (8.03)	66.7 (11.17)	7.7 (10.00)
Eldest (N = 431)	144.4 (21.68)	12.3 (13.92)	83.8 (11.17)	7.0 (8.92)	65.7 (9.92)	6.1 (7.74)
<i>Sex:</i>						
Male (N = 757)	134.7 (18.25)	12.8 (11.77)	81.2 (11.18)	7.2 (8.43)	64.7 (10.43)	8.7 (9.73)
Female (N = 890)	124.3 (21.07)	10.4 (11.70)	76.8 (11.56)	6.8 (8.81)	68.4 (10.84)	7.6 (9.83)
<i>Occupational Group:</i>						
Manual (N = 772)	130.5 (21.44)	11.1 (12.22)	79.3 (11.93)	6.5 (9.07)	67.0 (11.26)	6.9 (9.54)
Non-manual (N = 872)	127.8 (19.58)	11.8 (11.39)	78.4 (11.29)	7.3 (8.24)	66.5 (10.40)	9.1 (9.90)

Table 2.

Body mass index (BMI), waist-hip ratio, and obesity by age cohort, sex, and occupational status

	<i>N</i>		<i>BMI kg/m<sup>2</sup></i> <i>Mean (SD)</i>		<i>Waist-hip ratio</i> <i>Mean (SD)</i>		<i>Obesity</i> <i>N (%)</i>	
<i>Follow-up</i>	3rd	4th	3rd	4th	3rd	4th	3rd	4th
<i>Age Cohort:</i>								
Youngest	592	430	24.6 (4.07)	26.3 (4.57)	0.84 (0.08)	0.86 (0.08)	58 (10%)	75 (17%)
Middle	622	518	26.2 (4.20)	27.2 (4.42)	0.87 (0.08)	0.87 (0.08)	103 (17%)	112 (22%)
Eldest	430	328	26.3 (4.30)	27.0 (4.19)	0.88 (0.08)	0.88 (0.08)	64 (15%)	74 (23%)
<i>Sex:</i>								
Male	757	576	25.8 (3.76)	27.0 (3.74)	0.92 (0.06)	0.93 (0.06)	98 (13%)	117 (20%)
Female	887	700	25.7 (4.63)	26.7 (4.92)	0.81 (0.06)	0.82 (0.06)	127 (14%)	144 (21%)
<i>Occupational Group:</i>								
Manual	771	569	25.8 (4.23)	26.9 (4.39)	0.86 (0.08)	0.88 (0.08)	115 (15%)	119 (21%)
Non-manual	868	702	25.5 (4.27)	26.9 (4.46)	0.85 (0.08)	0.87 (0.08)	110 (13%)	142 (20%)

Table 3.

Body mass index (BMI), waist-hip ratio, obesity, and smoking status

		<i>N</i>	<i>BMI, kg/m<sup>2</sup></i> <i>Mean (SD)</i>	<i>Waist-hip ratio</i> <i>Mean (SD)</i>	<i>Obesity</i> <i>N (%)</i>
<i>Smoker:</i>	Current	593	24.7 (3.89)	0.86 (0.08)	55 (9%)
	Ex	338	26.6 (4.15)	0.87 (0.09)	64 (19%)
	Never	715	26.0 (4.44)	0.85 (0.08)	106 (15%)

Table 4: Final Hierarchical Regression Models Adjusting for Potential Confounding Variables

a) Body Mass Index and DBP reactivity	$\beta$	t	p	R <sup>2</sup>
Baseline DBP	-.28	14.19	<.001	
Age cohort	.14	5.42	<.001	
Sex	-.10	4.04	<.001	
Occupational group	-.03	1.11	.27	
PASAT performance score	.02	0.99	.32	
Medication status	.06	2.31	.02	
Smoking status	-.08	3.22	.001	.12 at step 1
Body Mass Index	.04	1.51	.13	.12 at step 2
b) Waist-hip Ratio and SBP reactivity	$\beta$	t	p	R <sup>2</sup>
Baseline SBP	-.36	12.91	<.001	
Age cohort	.25	9.40	<.001	
Sex	-.17	5.27	<.001	
Occupational group	.01	0.29	.78	
PASAT performance score	.13	5.49	<.001	
Medication status	.06	2.36	.02	
Smoking status	-.10	4.18	<.001	.13 at step 1
Waist-hip Ratio	.02	0.61	.55	.13 at step 2
b) Waist-hip Ratio and DBP reactivity	$\beta$	t	p	R <sup>2</sup>
Baseline DBP	-.37	14.36	<.001	
Age cohort	.13	5.18	<.001	
Sex	-.06	1.97	.05	
Occupational group	-.03	1.18	.24	
PASAT performance score	.03	1.06	.29	
Medication status	.06	2.40	.02	
Smoking status	-.08	3.38	.001	.12 at step 1
Waist-hip Ratio	.05	1.56	.12	.12 at step 2



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Figure 1: Estimated marginal means (SE) of heart rate reactivity for participants with BMIs in the normal range (n = 780), overweight (n = 608), and obese (n = 225).

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