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## Life Events and Acute Cardiovascular Reactions to Mental Stress: A Cohort Study

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# Life Events and Acute Cardiovascular Reactions to Mental Stress: A Cohort Study

Anna C. Phillips, Douglas Carroll, Christopher Ring, Helen Sweeting, and Patrick West

## **Abstract**

**Objective:** This study addressed the issue of whether frequent exposure to life events is associated with aggravation or blunting of cardiovascular reactions to acute mental stress.

**Methods:** In a substantial cohort of 585 healthy young adults, systolic and diastolic blood pressure and pulse rate were recorded at rest and in response to a mental arithmetic stress task. Participants indicated, from a list of 50 events, those they had experienced in the last year.

**Results:** There was an overall association between life events and blunted cardiovascular reactivity that was driven by variations in the frequency of exposure to desirable events. The total number of events and the number of personal events were negatively associated with systolic blood pressure and pulse rate reactions to acute stress, whereas the number of work-related events was negatively associated with diastolic blood pressure and pulse rate reactivity. The negative association between total events and systolic blood pressure reactivity was stronger for women than men, whereas men exposed to frequent undesirable events showed enhanced diastolic blood pressure reactivity. The blunting of pulse rate reactivity associated with frequent personal life events was evident particularly for those who had a relatively large number of close friends.

**Conclusions:** The nature and extent of the association between life events exposure and stress reactivity in young adults depends on the valence of the events together with the sex of the individual and their social network size.

**Key words:** acute stress, cardiovascular reactivity, life events, sex, social support.

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

Exaggerated cardiovascular reactions to acute stress have been implicated in the development and expression of cardiovascular disease (1-5). However, the influence of contextual factors on the magnitude of acute stress reactivity has received, as yet, relatively sparse attention (6, 7). Potentially paramount among such factors are individuals' recent histories of exposure to life events.

It has been hypothesised that high levels of background stress may be sensitising, serving to increase cardiovascular reactions to acute stress (8). There is some evidence in support of this hypothesis. For example, the frequency of chronic stress exposures, lasting nine months or more, but not more episodic stress exposures, was found to be positively associated with blood pressure and heart rate reactions to a mental arithmetic stress task (9). Further, children and adolescents with high levels of ongoing background stress showed larger increases in diastolic blood pressure and total peripheral resistance to a battery of stress tasks than those with little background stress (10). In contrast, more studies examining the effects of differences in exposure to life events on acute stress reactivity have found a negative relationship, i.e. that greater life events exposure is associated with blunting of cardiovascular reactivity. In an early study, high scores on a life events inventory were associated with reduced diastolic blood pressure reactions to mental arithmetic stress tasks, but only for participants with a positive family history of hypertension (11). A study focusing on occupational demands found that high demands were negatively related to cardiovascular reactions to a structured interview and a cognitive distraction task (12). Two studies of adolescents have also found associations that similarly suggest that high life events exposure blunts cardiovascular reactions to acute laboratory stress. Life event scores were inversely related to heart rate and blood pressure reactions to mental arithmetic, a video game, and the cold pressor test (13), and to a car-driving simulation task (14). Further, in a study of young to middle-aged adults, those with higher scores on a composite measure of stress displayed lower systolic blood pressure reactions to mental arithmetic and public speaking tasks (15). There are also studies that have found no relationship between life stress and acute cardiovascular reactivity. For example, no difference was reported in cardiovascular reactions to mental arithmetic and speech stress tasks between elderly individuals subject to the stress of

care-giving and age- and sex-matched controls (16). Further, participants high and low in recent life events did not differ in cardiovascular reactions to stressful film presentations (17), a stressful teaching exercise (18), a brief intelligence test challenge (19), and a mental arithmetic stressor (20).

Clearly, there is little consensus as to whether life events exposure is associated with increased or decreased reactivity to acute laboratory stress. It is difficult at this stage to know what may explain such variations in results. Although there are exceptions, it would appear that those studies which have focussed on chronic or background stress have tended to observe sensitisation, whereas those measuring life events exposure have tended to observe blunting. However, only further research will bring the necessary resolution. One difficulty is that, with the exception of two sizable population studies, both in youths (10, 14), most of the previous studies assessing general life events have tested modestly sized samples. It is possible that larger adult sample sizes are required to clarify the precise nature of the relationship between life stress and acute stress reactivity. Large samples permit the examination of possible moderators. Given that both sex and occupational status have been associated with cardiovascular reactivity in this study population and others (21, 22) (23, 24), they would suggest themselves as candidate moderators. Further, it has been reported that a high frequency of life events sensitises cardiovascular stress reactivity although only in individuals with large support networks (8).

The present study, then, examined the relationship between life events exposure and acute cardiovascular reactions to mental stress in a large cohort of young adults, encompassing both men and women, and approximately equal numbers from manual and non-manual occupational groups. Social network size was also measured. On the basis of the prevailing impression from previous research, it was hypothesised that individuals reporting high numbers of life events would be characterised by blunted rather than enhanced cardiovascular reactivity to acute stress. However, it was also hypothesised that this association would be moderated by such factors as sex, occupational status, and social network size.

## **Method**

### ***Participants***

Data are derived from the youngest cohort of the West of Scotland Twenty-07 Study who have been followed up at regular intervals since the baseline survey (aged 15) in 1987 (25). Participants were from the Glasgow area and were all between 23 and 25 years old at the third follow-up when data on life events and cardiovascular reactivity were collected. These data were available for 585 participants, with a mean age of 23.7 (SD = 0.57) years and a mean body mass index of 24.6 (SD = 4.08) kg/m<sup>2</sup>. There were 269 (46%) men and 316 (54%) women, and 254 (43%) came from manual and 331 (57%) from non-manual occupational households.

### ***Apparatus and Procedure***

A description of much of the testing procedure is available elsewhere (26). Testing sessions were conducted by trained nurses in a quiet room in the participants' homes. Demographic information was obtained by interview. Household occupational status was classified as manual and non-manual from the occupation of the head of household, using the Registrar General's (27) classification system of occupations. Head of household was either the participant, if working and living independently, or the parent with the higher occupational status, if the participant was a student or lived with their parents. Height and weight were measured and body mass index computed.

The inventory used to measure life events exposure is an elaboration of similar inventories used in previous waves of the study which have been described elsewhere (28). The inventory provided participants with a list of 50 life events and they had to indicate any they had experienced in the last 12 months, by selecting 'yes' from the binary 'yes'/'no' response options. The events were identified as those appropriate to young adults and covered three broad domains: work, education, and money (14 events); family life (18 events); personal life (18 events). Examples of the first of these were 'you were sacked from your job or training scheme', 'you failed a very important exam', 'you started college or university'. Examples of the second were 'your mother, father, brother,

or sister died', 'your parents decided to separate or divorce', and 'your mother or father got a new job', and examples of the last were 'you had sexual problems', 'you got into trouble with the police', and 'you got married'. Following previous practice (28) events were also classified as undesirable (30 events), desirable (10 events), and ambiguous (10 events). The distribution of undesirable to desirable events across the three domains was as follows: 6:3 work; 16:1 family life; 8:6 personal life. Finally, given the results of a previous study on chronic stress (9), the frequency of exposure to the seven events related to death and serious illness in family and close friends was also calculated for each participant; this provided a measure of exposure to the most serious of negative events. Participants were also asked to indicate the number of close friends they could rely on for support as a measure of social network size.

The stress task was the paced auditory serial addition test (PASAT), which has been shown in numerous studies to reliably perturb the cardiovascular system (26, 29-31). 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.

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate (PR) were determined by an Omron (model 705CP) semi-automatic sphygmomanometer. The Omron 705CP is a semi-automatic blood pressure measuring device recommended by the European Society of Hypertension (32). Following questionnaire completion (approximately 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 PR was taken. Task instructions were then given and the participant allowed a brief practice to ensure that



they understood the requirements of the PASAT. Two further SBP, DBP, and PR readings were taken during the task, the first initiated 20 seconds into the task (during the first slower sequence of numbers), and the second initiated 110 seconds later (at the same point within the first of 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 PR for each participant.

### ***Data Analyses***

One-way ANOVAs were used to test for differences in life event frequencies between sexes and occupational status groups. Repeated measures (baseline, task) ANOVAs were used to establish that the increases in SBP, DBP, and PR to the PASAT were statistically significant. Eta-squared ( $\eta^2$ ) is reported as a measure of effect size. Correlational analyses were then undertaken to determine the association between the numbers of life events experienced (overall and for the three domains) and cardiovascular reactivity, as well as the association between reactivity and the numbers of undesirable, undesirable, and severe events experienced; given that few participants ( $N = 19$ ) were exposed to more than one severe event, this last variable was transformed into a binary no exposure/exposure variable. Analysis then proceeded using hierarchical linear regression, in which at step 1, various possible confounders (i.e., body mass index, performance score on the PASAT, occupational class, sex) were entered. Given the almost complete lack of age variation in this cohort, age was not entered at step 1. In each of the models, the life events measure was entered at step 2. Moderation analyses were then undertaken, again using hierarchical regression, to test whether associations between life events and reactivity were moderated by sex, occupational status, and social support. As recommended to avoid multicollinearity (33, 34), the independent and potential moderator variables were mean centred and their products derived to test for interaction effects. The potential confounders were entered at step 1, with the exception of sex in the models testing sex as a main effect and occupational status in the models testing occupational status as a main effect. The main effects (using mean centred

variables) were entered at step 2, and the interaction at step 3. Significant interaction effects were then plotted using values corresponding to one SD above and below the mean of the predictor variable.

## **Results**

### ***Descriptive statistics***

The mean total number of life events experienced was 5.3 ( $SD = 3.30$ , range = 0 - 18). For the three domains the mean numbers of events experienced was: 2.6 ( $SD = 1.95$ , range = 0 - 9) for work, education, and money, 1.0 ( $SD = 1.34$ , range = 0 - 8) for family life, and 1.8 ( $SD = 1.50$ , range = 0 - 9) for personal life. The mean ( $SD$ ) number of undesirable events experienced was 1.8 ( $SD = 1.97$ , range = 0 - 13), of desirable events was 1.2 ( $SD = 1.02$ , range = 0 - 5), and of severe events was 0.3 ( $SD = 0.52$ , range = 0 - 4). The mean number of close friends was 4.5 ( $SD = 3.29$ , range = 0 - 30). The average performance score on the PASAT was 45.3 ( $SD = 1.50$ , range = 7 - 59) out of a possible 59. Men reported more total life events,  $F(1,583) = 9.57$ ,  $p = .002$ ,  $\eta^2 = .016$ , work-related events,  $F(1,583) = 11.31$ ,  $p = .001$ ,  $\eta^2 = .019$ , and personal events,  $F(1,581) = 4.28$ ,  $p = .04$ ,  $\eta^2 = .007$ , than women. Men also reported more undesirable events than women,  $F(1,566) = 11.95$ ,  $p = .001$ ,  $\eta^2 = .021$ . There were no significant differences in the total numbers of life events, work-related, family, or personal events reported by participants from manual and non-manual occupational households. There was a tendency for those from non-manual occupational households to report more desirable events,  $F(1,577) = 3.57$ ,  $p = .06$ ,  $\eta^2 = .006$ , and for those from manual occupational households to report more undesirable events,  $F(1,566) = 3.47$ ,  $p = .06$ ,  $\eta^2 = .006$ . There were no sex or occupational status differences in exposure to serious negative events. The means and standard deviations are presented in Table 1.

[Insert Tables 1 and 2 about here]

### ***Cardiovascular Reactions to Mental Stress***

Table 2 presents the mean baseline and task values. The increases in SBP, DBP, and PR to the mental stress task were substantial,  $F(1,584) = 597.06$ ,  $p < .001$ ,  $\eta^2 = .498$ , F

(1,584) = 335.38,  $p < .001$ ,  $\eta^2 = .365$ , and  $F(1,584) = 520.99$ ,  $p < .001$ ,  $\eta^2 = .471$ , respectively.

### ***Associations between Life Events and Cardiovascular Reactivity***

There were no significant correlations between resting baseline cardiovascular activity and any of the life events measures. The outcome of correlational analyses examining the relationship between life event numbers and cardiovascular reactivity is presented in Table 3. The correlation coefficients are mainly negative, indicating that relatively frequent life event exposure was associated with blunting of cardiovascular reactivity. Statistically significant negative correlations emerged for SBP and PR reactivity and both total life events and personal life events, whereas DBP reactivity correlated significantly only with the number of events related to work, education, and money. There were also negative associations between the number of desirable events reported and SBP and DBP reactivity, although they did not quite meet the criterion for statistical significance. Exposure to undesirable events was not significantly related to cardiovascular reactivity and there were no differences in reactivity between those who had experienced a serious event and those who had not.

[Insert Table 3 about here]

The outcome of hierarchical regression, with SBP reactivity as the dependent variable and in which sex, occupational status, body mass index, and performance on the mental stress task were entered at step 1 and the life events variable at step 2, are presented in Table 4. Tables 5 and 6 present the outcomes for DBP and PR reactivity, respectively. Only the models in which life events were significantly associated with reactivity are presented. SBP and PR reactivity were predicted by total life events and personal events, and DBP and PR reactivity were negatively associated with work events; the greater the number of events, the less the reaction to stress in each case. Further, the extent of exposure to desirable events was negatively associated with all three cardiovascular reactivity measures. The number of undesirable events and the number of serious negative events experienced were not significantly related to reactivity.

[Insert Tables 4, 5, & 6 about here]

### ***Association between Social Network Size and Cardiovascular Reactivity***

There was no association between the number of close friends and cardiovascular reactivity.

### ***Moderation Analyses***

Moderation analysis was undertaken to determine whether the associations between life events and reactivity were moderated by sex (0 = men, 1 = women) and occupational status (0 = non-manual, 1 = manual). There was no evidence of moderation by occupational status. However, two significant sex  $\times$  life events interaction effects emerged. These analyses are summarised in Table 7. Presentation is purposively limited to the step 2 and 3 outcomes. In addition, step 3 reporting has been restricted to the main effects and interactions, as little changes for the other variables. Greater overall life events exposure was associated with a more pronounced blunting of SBP reactivity for women than for men. As recommended (33), subsequent analyses of the individual slopes showed that total life events was a significant negative predictor of SBP reactivity for women,  $B = -0.61$ , 95%CI =  $-0.98$  to  $-0.25$ ,  $t = 3.26$ ,  $p = .001$ , but not men,  $B = -0.11$ , 95%CI =  $-0.45$  to  $0.23$ ,  $t = 0.65$ ,  $p = ns$ , see Figure 1a. Conversely, for men more frequent exposure to undesirable life events was associated with enhanced DBP reactions to the mental stress task, whereas for women there was no relationship between exposure to undesirable events and DBP reactivity, see Figure 1b. Although different from one another, subsequent analyses, however, revealed that the individual slopes for men,  $B = 0.43$ , 95%CI =  $-0.07$  to  $0.94$ ,  $t = 1.69$ ,  $p = .09$ , and women,  $B = -0.37$ , 95%CI =  $-0.99$  to  $0.25$ ,  $t = 1.18$ ,  $p = .24$ , did not differ significantly from zero.

[Insert Table 7 and Figure 1 about here]

Moderation analyses were also undertaken using the social network measure. There was a significant interaction between the number of close friends and personal life events for

PR reactivity. This analysis is also summarised in Table 7, with the interaction displayed in Figure 2. Blunting of PR reactivity was associated with frequent personal life events for individuals with a relatively high number of close friends. For individuals with the mean number or greater than the mean number of close friends, higher personal life events exposure was associated with lower PR reactivity. Examination of the individual slopes confirmed this assertion. The slopes relating personal life events to PR reactivity were significant different from zero for participants with the mean number of close friends,  $B = -0.94$ ,  $95\%CI = -1.52$  to  $0.36$ ,  $t = 3.16$ ,  $p = .002$ , and for those with one standard deviation above the mean number of close friends,  $B = -1.69$ ,  $95\%CI = -2.65$  to  $0.74$ ,  $t = 3.48$ ,  $p = .001$ , whereas the slope was not significantly different from zero for participants with one standard deviation below the mean number of close friends.

## **Discussion**

In a substantial cohort of young adults, the magnitude of their cardiovascular reactions to acute mental stress was negatively associated with the frequency of their exposure to life events. SBP and PR reactivity were inversely related to the total number of events and the number of personal events experienced in the previous year. DBP and PR reactivity were negatively associated with the number of work, education, and money related events. These findings resonate with those from other studies measuring stress as life events exposure (11, 13-14). However, it was the frequency of exposure to events designated as desirable that was consistently related to blunted cardiovascular reactivity in the present study. There were no overall significant associations between events designated as undesirable and derived as serious and cardiovascular reactivity. There are indications in previous studies of young samples, which find that life events exposure is associated with blunting of reactivity, that it is not the most serious and undesirable events that underlie the association. For example, in a small scale study of adolescents, those with the more modest cardiovascular reactions to stress reported significantly less severe events than those with the highest reactivity (13). In addition, blunting was found to be related only to variations in low subjective impact stress exposure (11). It has also been argued that the blunting of cardiovascular reactivity observed in a large scale study of adolescents occurred where stressful events had been resolved and no longer had a

negative impact (14). This sort of explanation would help reconcile the present results with those observed in the other substantial study of youths, in which heightened cardiovascular reactivity was observed for those who reported a single ongoing stress exposure but not for those who reported having resolved a significant life stress (15).

It is reasonable to conclude that the blunting of cardiovascular reactions observed in the present study did not reflect disengagement from the mental stress task, as this should have been reflected in poorer performance. Not only did the negative relationship between life events exposure and reactivity emerge following adjustment for the number of correct answers on the acute stress task, but subsequent analysis revealed no significant associations between life events scores and PASAT performance. The other parsimonious explanation for blunting enlists physiological adaptation (13), and has been referred to as the ‘inoculation effect’ (35). The assumption is that frequent exposure to life events causes a gradual decline in their impact on the cardiovascular system, so that when confronted by a further challenge, such as an acute stress task, individuals with high life events exposures show diminished reactivity. It has been long appreciated that desirable as well as undesirable experiences can perturb cardiovascular function (36).

The reactivity hypothesis, which considers that large magnitude reactions to psychological challenge play a role in the development and expression of cardiovascular disease (1-5), has been very influential, generating substantial numbers of studies. More recently, it has been argued that, in addition to the magnitude of cardiovascular reactions to stress, the reactivity hypothesis must also take into account individual differences in exposure to those life events that provoke cardiovascular reactivity, and that it is the product of exposure history and the reactivity magnitude that confers cardiovascular disease risk (6, 37). However, such versions of the reactivity hypothesis assume that reactivity magnitudes are independent of individuals’ exposure histories. The present results, indicating that that life events exposure and reactivity are linked, suggest that the simple multiplicative hypothesis requires revision.

The present study also provides provisional evidence of a complex interaction between the sex of the participant and the valence of life events and whether blunted or enhanced reactivity is observed. First, the negative association between life events and SBP reactivity was stronger for women than for men. Few studies in the life events and acute stress reactivity field, to date, have had the power to properly explore moderation effects. Since many of the studies showing the strongest blunting effects of supportive others on cardiovascular reactions to acute stress tested only young women (e.g. (38-40)), it may be a general phenomenon that more positive or less negative experiences exert a greater attenuating effect on acute stress reactivity in young women than in young men. Such speculation should be qualified by the conclusion drawn from a previous study that men are more likely than women to show blunted reactivity with high stress exposure (15), as well as by our failure to find a statistically significant interaction effect for sex and desirable events on SBP reactivity. Nevertheless, it is worth noting that this prior study was concerned with chronic undesirable stress and not low impact or desirable exposures and that the standardised regression coefficient we observed for the sex  $\times$  desirable events interaction was in the appropriate direction,  $B = -1.29$ ,  $SE = 0.84$ ,  $\beta = -.10$ ,  $\Delta R^2 = .004$ ,  $p = .13$ . Second, for men, relatively frequent exposure to undesirable events was associated with enhanced DBP reactivity, whereas, for women, frequency of undesirable events was unrelated to reactivity (see Figure 1b). The finding that the frequency of relatively undesirable events was associated with enhanced cardiovascular reactivity in males is not without precedent. Adolescent boys with the largest cardiovascular reactions to stress reported significantly more severe events than those with lower reactivity (13). It is possible that young men exposed to numerous undesirable events are more prone to show sensitization of reactivity. However, it is again necessary to qualify this conclusion. Sensitization of cardiovascular reactivity by frequent undesirable events has also been found in a student sample that included women as well as men (9) and, as indicated, greater blunting of cardiovascular reactivity in association with chronic stress in men than women has been inferred from the results of another study (15).

In the present study, social network size appeared to moderate the relationship between personal life events and PR reactivity. The observed negative association between

personal life events and PR reactivity was evident only for participants with the mean or higher than the mean number of close friends, a result that would appear to contradict the findings of the only other study to examine the interaction between life events and social network size, in which the combination of larger social networks and more frequent life events was associated with greater cardiovascular reactivity in men (8). However, in this latter study, contrary to the researchers' expectations, those with large social networks showed greater cardiovascular responses to a mental arithmetic stress irrespective of life events exposure. Only further research will help clarify the precise nature of the interaction between life events and social support in this context.

The present study suffers from a number of limitations. First of all, it was not designed specifically to explore the issue of whether and how life events are associated with the magnitude of cardiovascular reactions to acute mental stress. Thus, personality factors which could conceivably affect both the reporting of life events and reactivity, were not measured. However, other studies have failed to demonstrate that such variables had any substantial impact in this context (8). Further, many of the previous studies of life events and reactivity, particularly the few large scale studies, were similarly opportunistic. Second, life events checklists of the sort used in the present study are not without their limitations. It is self-evident that ostensibly the same event can impact very differently in different individuals. Nevertheless, in the present context, such checklists are the most common means of stress assessment and the alternatives are also not without difficulties. For example, measures of perceived stress and psychological well-being almost certainly suffer more than checklists of objective events from the reporting bias that arises from individual differences in negative affectivity (41). Third, only SBP, DBP and PR reactivity were measured, and only to one stress task. However, in a large cohort study, more comprehensive cardiovascular monitoring to a variety of stress tasks was not practicable. Further, SBP reactivity to the task used in this study has been found to predict prospective changes in resting blood pressure status (26). In addition, whether blunting or sensitization is observed is not easily attributable to the nature of the stress task. Of the studies which have used an obviously social stress task, one reported that stress blunts cardiovascular reactivity (15), two that stress aggravates cardiovascular



reactivity (10, 14) and two report no association (16, 18). Of the studies which have employed a mental arithmetic stress task, three found blunting (11, 13, 15), one observed enhanced cardiovascular reactivity (9), and one reported no effect (20). Fourth, it should be conceded that the present associations emerge from a substantial number of analyses and thus caution is warranted. However, the consistency of direction of correlation coefficients evident in Table 3 argues against the notion that the primary finding of blunting of reactivity arose by chance. Further, the associations are small in terms of the amount of variance explained. Nevertheless, the effect sizes that can be inferred from the two other large scale studies of life events and reactivity in young people are of the same order of magnitude (14, 15). In terms of the implications of effects of this size for smaller scale studies in the field, it is worth noting that low power constitutes a risk for type 1 as well as type 2 errors (42). Finally, our conclusions may only apply to relatively young samples, particularly given that there are age-related variations in cardiovascular reactions to stress (21) and an increased likelihood of exposure to severe life events with age.

In summary, in a large cohort of young adults, the frequency of exposure to life events in the previous year was negatively associated with cardiovascular reactivity to an acute mental stress task. However, this association was driven by variations in exposure to desirable events. In general, it would appear that where blunting of reactivity is observed in young cohorts, it is the frequency of the less negative or resolved events that is related to blunted cardiovascular reactions to acute stress. In addition, the negative relationship between events and SBP reactivity occurred for young women but not for young men. In men, relatively frequent exposure to undesirable events was associated with enhancement of DBP reactivity. Further, the blunting of PR reactivity associated with frequent personal events was manifest only for those who had a relatively large number of close friends. Thus, there would appear to be a complex interaction between the sex of the participant, social network size, and the valence of life events in determining whether, and to what extent, blunted or enhanced reactivity is observed.

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Table 1

Mean (*SD*) Life Events for Men and Women and for Participants from Manual and Non-Manual Occupational Households

	Men (N = 269)	Women (N = 316)	Manual (N = 254)	Non-manual (N = 331)
Total Life Events	5.78 (3.56)	4.94 (3.02)	5.24 (3.54)	5.40 (3.11)
Total Work Events	2.86 (2.04)	2.32 (1.84)	2.39 (1.98)	2.70 (1.92)
Total Family Events	1.00 (1.38)	0.97 (1.31)	1.06 (1.31)	0.93 (1.37)
Total Personal Events	1.92 (1.68)	1.67 (1.31)	1.80 (1.56)	1.77 (1.56)
Desirable Events	1.18 (0.98)	1.14 (1.05)	1.06 (1.04)	1.22 (0.99)
Undesirable Events	2.19 (2.24)	1.62 (1.68)	2.06 (2.04)	1.75 (1.91)
Serious Events (% with)	21	22	25	19

Table 2

Mean (*SD*) Cardiovascular Activity at Baseline and during the Mental Stress Task

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	Baseline	Task
SBP (mmHg)	120.0 (15.1)	130.1 (16.0)
DBP (mmHg)	73.3 (10.1)	80.2 (10.4)
PR (ppm)	67.5 (11.0)	77.6 (12.9)

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Table 3

Pearson Correlations Coefficients between Numbers of Life Events and Cardiovascular Reactivity

	SBP Reactivity	DBP Reactivity	PR Reactivity
Total Life Events	-.10*	-.06	-.09*
Total Work Events	-.06	-.12**	-.06
Total Family Events	-.07	.02	-.04
Total Personal Events	-.08*	.01	-.09*
Desirable Events	-.08 <sup>+</sup>	-.08 <sup>+</sup>	-.06
Undesirable Events	-.05	.02	-.05

<sup>+</sup>  $p = .06$ , \*  $p < .05$ , \*\*  $p < .01$

Table 4. Hierarchical Regression models for Life Events: SBP Reactivity

a) Total Life Events

	B	SE	$\beta$	$\Delta R^2$
Step 1				
Sex	-0.76	0.86	-.04	
Occupational status	-0.29	0.86	-.01	
BMI	0.00	0.10	.01	
Performance score	0.01	0.05	.07	.007
Step 2				
Sex	-1.06	0.86	-.05	
Occupational status	-0.36	0.86	-.02	
BMI	0.00	0.10	.00	
Performance score	0.01	0.05	.07	
Total life events	-0.34	0.13	-.11**	.012**

b) Personal Life Events

Step 2				
Sex	-0.90	0.86	-.04	
Occupational status	-0.26	0.86	-.01	
BMI	0.00	0.10	.00	
Performance score	0.01	0.05	.07	
Personal life events	-0.60	0.28	-.09*	.008*

c) Desirable Events

Step 2				
Sex	-0.74	0.86	-.04	
Occupational status	-0.50	0.87	-.02	
BMI	0.00	0.10	.01	
Performance score	0.01	0.05	.08	
Desirable events	-0.92	0.42	-.11*	.008*

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

Table 5. Hierarchical Regression models for Life Events: DBP Reactivity

a) Work Events

	B	SE	$\beta$	$\Delta R^2$
Step 1				
Sex	0.80	0.77	.04	
Occupational status	-0.01	0.77	.00	
BMI	-0.16	0.09	-.07	
Performance score	-0.01	0.05	-.01	.008
Step 2				
Sex	0.48	0.77	.03	
Occupational status	-0.27	0.77	-.02	
BMI	-0.18	0.09	-.08*	
Performance score	0.00	0.04	-.01	
Work life events	-0.56	0.20	-.12**	.014*

b) Desirable Events

Step 2				
Sex	0.73	0.77	.04	
Occupational status	-0.18	0.77	-.01	
BMI	-0.17	0.09	-.08	
Performance score	0.00	0.05	.00	
Desirable events	-0.74	0.38	-.08*	.007*

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

Table 6. Hierarchical Regression models for Life Events: PR Reactivity

a) Total Life Events

	B	SE	$\beta$	$\Delta R^2$
Step 1				
Sex	-0.21	0.88	-.01	
Occupational status	-0.64	0.88	-.03	
BMI	-0.36	0.11	-.14**	
Performance score	0.26	0.05	.20**	.006**
Step 2				
Sex	-0.54	0.88	-.03	
Occupational status	-0.72	0.88	-.03	
BMI	-0.39	0.11	-.15**	
Performance score	0.26	0.05	.21**	
Total life events	-0.38	0.13	-.12**	.013**

b) Work Events

Step 2				
Sex	-0.47	0.89	-.02	
Occupational status	-0.80	0.88	-.04	
BMI	-0.38	0.11	-.14**	
Performance score	0.26	0.05	.20**	
Work events	-0.44	0.22	-.08*	.006*

c) Personal Life Events

Step 2				
Sex	-0.32	0.88	-.02	
Occupational status	-0.55	0.88	-.03	
BMI	-0.39	0.11	-.15**	
Performance score	0.26	0.05	.21**	
Personal life events	-0.81	0.29	-.11**	.012**

d) Desirable Events

Step 2				
Sex	-0.11	0.88	-.01	
Occupational status	-0.52	0.88	-.02	
BMI	-0.39	0.11	-.15**	
Performance score	0.28	0.05	.22**	
Desirable events	-1.00	0.43	-.10*	.009*

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

Table 7. Hierarchical Regression models for Life Events: Moderation Analyses

a) Total Life Events and SBP reactivity

	B	SE	$\beta$	$\Delta R^2$
Step 2				
Occupational status	-0.36	0.86	-.02	
BMI	0.00	0.10	.00	
Performance score	0.01	0.05	.07	
Sex	-1.06	0.86	-.05	
Total life events	-0.34	0.13	-.11*	.012**
Step 3				
Sex	-1.06	0.86	-.05	
Total life events	-0.11	0.17	-.04	
Sex $\times$ events interaction	-0.50	0.26	-.11*	.006*

b) Undesirable Events and DBP reactivity

	B	SE	$\beta$	$\Delta R^2$
Step 2				
Occupational status	0.00	0.79	.00	
BMI	-0.15	0.10	-.06	
Performance score	0.00	0.05	-.02	
Sex	0.89	0.80	.05	
Undesirable events	0.11	0.20	.02	.002
Step 3				
Sex	0.87	0.80	.05	
Undesirable events	0.43	0.26	.09	
Sex $\times$ events interaction	-0.80	0.41	-.11*	.007*

c) Personal Events and PR reactivity

	B	SE	$\beta$	$\Delta R^2$
Step 2				
Sex	-0.18	0.88	-.01	
Occupational status	-0.64	0.88	-.03	
BMI	-0.38	0.11	-.14	
Performance score	0.25	0.05	.20	
Number of Close Friends	0.11	0.13	.04	
Personal Events	-0.85	0.29	-.12	.014**
Step 3				
Number of Close Friends	0.17	0.14	.05	
Personal events	-0.94	0.30	-.13	
Close friends $\times$ events interaction	-0.23	0.11	-.09*	.008*

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

### Figure Captions

Figure 1: a) Interactions between Total Life Events and Sex for SBP Reactivity. TLE = Total Life Events. Separate regression lines are plotted for Men and Women.  
b) Interactions between Total Undesirable Life Events and Sex for DBP Reactivity. TULE = Total Undesirable Life Events. Separate regression lines are plotted for Men and Women.  
 $\hat{Y}$  = predicted reactivity.

Figure 2: Interactions between Personal Life Events and Number of Close Friends for PR Reactivity. PLE = Personal Life Events. Separate regression lines are plotted for the mean, high (+1 *SD*), and low (−1 *SD*) number of close friends.  
 $\hat{Y}$  = predicted reactivity.



