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Clayton J. Hilmert^a, Ai Ni Teoh^a & Michael M. Roy^{bc}

^a Department of Psychology, North Dakota State University, Fargo, ND, USA.

^b Department of Psychology, Elizabethtown College, Elizabethtown, PA, USA.

^c Department of Music, North-West University, Potchefstroom, South Africa.

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Effort and negative affect interact to predict cardiovascular responses to stress

Clayton J. Hilmert^{a*}, Ai Ni Teoh^a and Michael M. Roy^{b,c}

^aDepartment of Psychology, North Dakota State University, Fargo, ND, USA; ^bDepartment of Psychology, Elizabethtown College, Elizabethtown, PA, USA; ^cDepartment of Music, North-West University, Potchefstroom, South Africa

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Objective: Although traditional models posit that negative emotional responses to stress increase cardiovascular reactivity (CVR), laboratory studies have generally not found a strong emotion–CVR association. In this paper, we took a multidimensional approach to examining psychological reactions to stress in three studies.

Methods: In each study we assessed the amount of effort exerted by a participant and the negative affect (NA) felt by the participant with different self-reported measures and an effort behavioural measure.

Results: Our findings consistently demonstrated that NA was associated with CVR when effort was relatively high, but not when effort was relatively low.

Conclusion: This suggests that the weak NA–CVR correlations reported in past research may have been confounded by a third effort-related variable and that CVR is significantly associated with NA under certain circumstances. Furthermore, our findings suggest that by considering the multidimensional nature of psychological responses to stress, we may come to better understand the links between stress-related emotion and physiology.

Keywords: cardiovascular reactivity; effort; laboratory stressor; negative affect; stress

Associations between emotional responses and cardiovascular reactivity (CVR) to acute stress in the lab have been inconsistent. The fact that reported correlations are generally weak and range from $r = .13$ to $.34$ (Feldman et al., 1999) challenges the notion that the heart and emotions are intimately associated and the validity of laboratory research studying psychophysiological stress reactivity (cf. Gerin et al., 1999; Schwartz, 1999). In this paper, we reconsider the association between negative emotion and CVR in three stress-reactivity studies using a multidimensional approach to measuring psychological responses to stress. We hypothesised that the inconsistent and generally weak correlations between negative emotion and CVR reported in previous experiments may have been due, in part, to a third factor moderating the emotion–CVR association, namely, the amount of effort put forth during the stressor.

*Corresponding author. Email: clayton.hilmert@ndsu.edu

Cardiovascular reactivity, negative affect and health

According to the reactivity hypothesis (Krantz & Manuck, 1984) chronic, large increases in blood pressure in response to stress, or CVR, cause wear and tear on the cardiovascular system that, over time, indicates, and likely contributes to the development of cardiovascular disease (CVD; Treiber et al., 2003). This idea has spurred a great deal of research focusing on factors that augment and attenuate cardiovascular responses to stress. One common approach has been to induce acute stress in the laboratory while blood pressure and heart rate (HR) are monitored and psychosocial variables are manipulated (see Kamarck & Lovallo, 2003, for review).

Traditional stress and health models posit that stress-related negative emotion is associated with physiological arousal, including CVR, and consequently is related to the development of CVD (Cohen, Kessler, & Gordon, 1997; Lazarus & Folkman, 1984; Lovallo, 2004). Epidemiologic research has shown that anxiety, depression, anger and psychological disorders that involve intense negative emotions have been linked to CVD (Kubzansky, 2007; Ormel et al., 2007). Excessive CVR to stress seems a logical pathway from negative emotions to cardiovascular health. However, despite their common association with CVD, negative emotion has not been strongly associated with CVR in past studies; findings have been inconsistent with some studies reporting no associations (e.g. Warner & Strowman, 1995) and others reporting weak associations (Feldman et al., 1999). In general, the pathways linking negative emotion to CVD remain unclear.

Multidimensional stress responses

In a stressful laboratory setting, participants' psychological responses are likely complicated and multifaceted (Russell & Feldman Barrett, 1999). Thus, when investigating a stress-related emotion-CVR connection we must consider that an array of psychological responses might be elicited by stress. These responses could involve multiple emotions experienced simultaneously (Lane & Schwartz, 1987), or various blends of emotional valence (positivity-negativity) and emotional energy (activation; Russell & Feldman Barrett, 1999). Here, we took the latter perspective and considered emotional responses to stress with a bidimensional circumplex, involving some degree of affective valence – positive to negative – and activation – invigorated to fatigued (see Figure 1; Thayer, 1989; Russell, 2003). Conceiving of emotional responses to stress as involving two continuous dimensions rather than as involving a unidimensional discrete emotional response affords more precise measurement and description of the emotional experience (Russell, 2003).

Because measures of similarly valenced discrete emotions (e.g. anger, sadness and anxiety) are strongly correlated (Carroll, Yik, Russell, & Feldman Barrett, 1999), past laboratory studies of emotion and CVR that employed discrete negative emotion measures likely captured the degree of emotional negativity (valence) experienced by participants performing stressful tasks. However, none have considered both degree of negativity and the level of activation felt in response to a stressor. In the present studies, we considered a valence dimension of the stress response with measures of discrete negative emotions and an activation dimension of the stress response with measures of effort exerted or motivation felt during a stressful task.

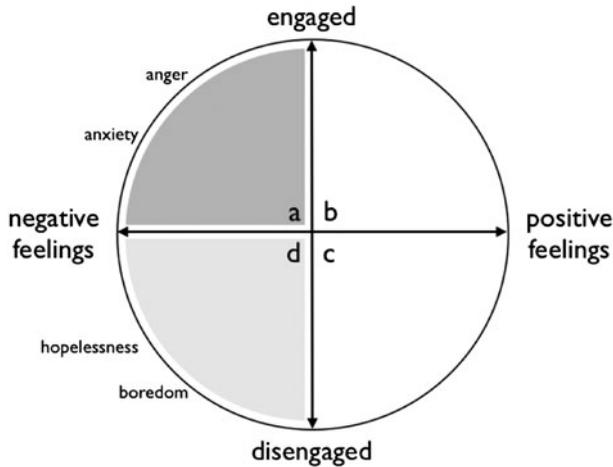


Figure 1. The stress response circumplex. Stress responses to the left of the y -axis in the shaded regions involve negative feelings coupled with some degree of engaged/motivated responding (quadrant a; e.g. anger, anxiety) or disengaged/enervated responding (quadrant d; e.g. hopelessness, boredom). We hypothesise that NA will be more strongly associated with CVR in quadrant a than in quadrant d (shown with darker and lighter shading, respectively). Stress responses to the right of the y -axis involve positive feelings and are beyond the scope of this paper.

Cardiovascular reactivity and activation

In the following studies we measured activation with self-reports of effort (Study 1) and motivation (Study 2). In addition, because the activation dimension of an emotional experience can be affected by and reflected in behaviour (Thayer, 1989) we utilised a behavioural measure of effort in Study 3. Unsurprisingly, effort has been associated with CVR to stress in past research (e.g. Wright & Kirby, 2001). For example, studies that involved avoiding an aversive stimulus (e.g. shock) by completing an easy, moderately difficult or impossible task (Elliott, 1969; also, classical aversive condition paradigms; e.g. Obrist, 1981) generally found that a moderately difficult task induced greater increases in blood pressure and HR than easy tasks and impossible tasks (Obrist, 1981). As there was no action possible in the impossible condition, lower CVR in this condition and the easy task condition supports the conclusion that CVR is proportional to the amount of effort exerted to avoid shock (e.g. Elliott, 1969; Obrist, 1981; Wright & Kirby, 2001). Further, if emotional intensity is greatest in the impossible task condition, as some have assumed (e.g. Obrist, 1981), then it might be concluded that CVR is not associated with emotion.

It is our contention that a situation that elicits intense negative emotion and prohibits any recourse is a qualitatively different stress-emotion episode than a situation that elicits intense negative emotion and allows for alternatives to passive acceptance. The qualitative difference, in our view, is the level of emotional activation that is present in a hopelessly impossible stressful situation compared to a situation that is stressful, but where effort could result in escape or victory. Thus our current interest is in how effort and emotion interact to predict CVR. An easy/moderately difficult/impossible stress task manipulation does address this interaction as all

combinations of high and low effort and emotion are not included. Depending on whether you consider the moderately difficult condition a high or low negative emotion condition, either a high effort and high emotion condition or a high effort and low emotion condition is not represented.

Cardiovascular reactivity, emotion and effort

Few studies have considered emotion, effort and their combination as predictors of physiological functioning. In one study, Shapiro, Jamner, Goldstein, and Delfino (2001) examined how emotion and energy-related variables were associated with average daily ambulatory blood pressure. Specifically, the authors examined interaction effects of discrete negative emotion (stressed, anxious and angry) measures, and an energy-/effort-related mood (tired) measure on blood pressure and HR. Results indicated that there were significant negative emotion by energy interactions. Participants who reported high *stress* and low *tiredness* (high energy) had higher daily diastolic blood pressure (DBP) over a five-day period than those who reported other combinations of *stress* and *energy* (Shapiro et al., 2001). Although Shapiro et al. did not consider acute responses to stress, this study suggests that an energy-related state in an acute stress situation may interact with a negative emotion measure to predict CVR.

In the following studies we considered: (1) how negatively participants felt and (2) how much effort they put forth during stressful laboratory tasks. We assumed that effort reflected how activated participants felt and that activation and negative affect (NA) varied independently in response to a stressful task. Our general hypothesis was that the association between NA and CVR would be moderated by effort. That is, we expected the strength of the association between negative feelings in response to stress and cardiovascular responses to stress to depend on how activated the participant felt during the stressor.

The following studies are secondary analyses of data from experiments that examined psychophysiological responses to stress (Study 1: Hilmert, Kulik, & Christenfeld, 2002; Study 2: Roy, Dirksing, & Christenfeld, 2005; Study 3: Hilmert, Christenfeld, & Kulik, 2002). These studies were not designed to test our hypothesis nor did they include analyses relevant to the current hypothesis. However, each included a manipulation of 'stress' and measures of NA and effort that were originally manipulation checks and possible mediators of condition effects on CVR. For our analyses we collapsed across study conditions, (e.g. no social support and social support conditions), thus we assumed NA varied within each data-set (Lazarus & Folkman, 1984). Furthermore we assumed that effort varied, as there were no a priori intentions to control effort. In other words, we expected that when performing a stressful task, like performing a speech to an audience, these samples experienced a range of both negativity and activation.

The following studies involved a typical laboratory stressor (public speaking; Studies 1 and 3) and an atypical stressor (anagram-solving competition; Study 2). In all three studies, after informed consent was provided, resting baseline measures of blood pressure and HR were taken. Participants then performed a stressful task while their blood pressure and HR continued to be recorded. Self-reports of effort and NA were measured immediately after the task.

Study 1: Effort × ‘nervous’ during public speaking

In Study 1, we examined if a self-reported effort by ‘nervousness’ interaction predicted CVR to a speech task. The data were from a social support study (Hilmert, Kulik et al., 2002). We collapsed across experiment conditions prior to analyses.

Method

Participants and task

A total of 59 female undergraduates from the University of California, San Diego participated in this study for class credit.

Participants were asked to give a speech to a peer stranger on abortion. The audience member was a confederate of the experiment and was trained to react to the participants’ speeches supportively or non-supportively depending on the experimental conditions. Half of the participants performed the speech in the presence of an experimenter while the others performed it in the absence of an experimenter (Hilmert, Kulik et al., 2002). Immediately after the speech task, the participants completed questionnaires containing the relevant NA and effort measures.

Cardiovascular, negative affect and effort measures

Beat-to-beat blood pressure and HR measures were taken using an Ohmeda Finapres finger cuff on the participant’s non-dominant hand. All experimenters were trained to use the Finapres such that CVR was measured accurately and reliably (Kurki, Smith, Head, Dec-Silver, & Quinn, 1987; Smith, Wesseling, & DeWitt, 1985). Measures taken during an initial five-minute resting period were combined into average systolic blood pressure (SBP), DBP and HR baselines. Beat-to-beat cardiovascular measures were also taken continuously during the speech task and averaged. The baseline measure was then subtracted from the average task measure to calculate SBP, DBP and HR reactivity.

To measure NA, we used a single-item self-report of how nervous the participant felt during the task. Ratings of nervousness were on a five-point Likert scale from 1 (*not at all*) to 5 (*very much*). Effort was measured with a single-item self-report of how much effort the participant exerted during the task on a five-point Likert scale from 1 (*no effort*) to 5 (*a lot*).

Analyses overview

First, data were collapsed across conditions. To examine if the public speaking task had an overall effect on cardiovascular functioning, repeated measures *t* tests compared mean baseline and task levels of SBP, DBP and HR. Next, to judge the degree of independence of our psychological measures we examined the correlation between effort and ‘nervousness’. We also examined correlations between cardiovascular baseline measures and, effort, NA and CVR. Baseline measures were not significantly associated with effort or NA in any study ($ps > .10$) and, therefore, these associations are not considered again. Baseline measures were significantly, negatively associated with respective SBP and HR reactivity measures in Study 1 and HR reactivity in Study 2 ($ps < .05$). In these instances, regression analyses were run both with and without baseline as a covariate.

To examine if an effort by emotion interaction predicted CVR, we first used separate hierarchical regression analyses predicting SBP, DBP and HR reactivity. We statistically accounted for variance in CVR associated with experimental conditions by entering a condition variable in the first step of regression analyses. This strategy made our analyses rather conservative as some condition (i.e. support vs. non-support and experimenter present vs. absent) effects likely accounted for a portion of the variances in emotion and effort. In the second step of the regression analyses, independent z-scored effort and nervous measures were entered. Finally, in the third step of the analyses we entered an effort by NA interaction variable that was computed with the standardised variables. We further examined effort by NA interaction effects by graphing the interactions and with simple slopes analyses in which associations between NA and CVR are examined at 1SD above and 1SD below mean effort (Aiken & West, 1991). Finally, we compared the effect size of the association between NA and CVR when effort was a moderator and when it was not by using the Fisher's Z transformation test (one-tailed).

Results and discussion

Preliminary analyses

The public speaking task significantly increased SBP, DBP and HR (Table 1). The correlation between effort and nervousness was negative, but not quite statistically significant, $r(57) = -.23$, $p < .08$. Baseline SBP and HR were significantly negatively correlated with respective reactivities ($r_s(57) = -.32$ and $-.33$, respectively, $ps < .05$).

Hypothesis testing

There were no significant main effects of effort on SBP, $\beta = .15$, $t = 1.15$, $p > .05$, DBP, $\beta = .15$, $t = 1.20$, $p > .05$, and HR reactivity, $\beta = .09$, $t = .72$, $p > .05$. Stronger feelings of nervousness were significantly associated with greater SBP reactivity, $\beta = .29$, $t = 2.27$,

Table 1. The means and standard deviations of cardiovascular readings during baseline and stressful task in studies 1, 2 and 3.

Cardiovascular Index	Stage	Study 1	Study 2	Study 3
Systolic blood pressure (mmHg)	Baseline	108.88 ^b (15.50)	111.91 ^b (13.81)	108.80 ^b (14.26)
	Task	154.04 ^a (18.92)	140.28 ^a (18.88)	161.04 ^a (21.92)
Diastolic blood pressure (mmHg)	Baseline	61.76 ^b (8.50)	69.42 ^b (9.92)	66.47 ^b (9.10)
	Task	88.36 ^a (11.86)	85.74 ^a (12.21)	100.54 ^a (13.71)
Heart rate (bpm)	Baseline	77.35 ^b (10.68)	78.67 ^b (12.73)	78.41 ^b (9.63)
	Task	91.68 ^a (12.50)	90.89 ^a (13.87)	94.43 ^a (15.01)

Note: The values in the parentheses were the corresponding SDs. Baseline and task values for each measure and study with different superscripts differed significantly from each other at $p < .05$.

$p < .05$, and approached statistical significance for DBP reactivity, $\beta = .25$, $t = 1.91$, $p = .06$. The main effect of nervousness was not statistically significant for HR reactivity, $\beta = .08$, $t = .59$, $p > .05$.

The regression analyses also revealed a significant effort by nervousness interaction effect on SBP reactivity, $\beta = .27$, $t = 2.23$, $p < .05$, and on DBP reactivity, $\beta = .25$, $t = 2.03$, $p < .05$. The former result remained significant when baseline SBP was included as a covariate. Simple slope analyses showed that blood pressure reactivity was positively associated with nervousness when participants were putting forth relatively high effort, and was unrelated to nervousness when participants reported relatively low effort (see Table 2 for simple slope regression coefficients). Figure 2A shows the effort by nervousness interaction effect on SBP reactivity. As shown in the figure, the combination of high effort and high nervousness was associated with greater SBP reactivity than other combinations. The same pattern was found for DBP reactivity. The effort by nervousness interaction effect was not significant on HR reactivity, $\beta = -.09$, $t = -.66$, $p > .05$ and remained such when HR baseline was controlled for.

The results from Experiment 1 showed that effort and NA interacted to predict CVR.

When effort was not considered, NA accounted for 8.41% of the variance in SBP reactivity and 6.25% of the variance in DBP reactivity. When effort was considered and was relatively high (+1SD), NA accounted for 30.58 and 24.50% of the variance in SBP and DBP reactivity, respectively. Effect size comparisons using the Fisher's Z transformation test revealed that the association between NA and CVR when effort was high (+1SD) was significantly stronger than the overall association between NA and CVR for SBP, $z = 1.69$, $p = .05$. There was a similar association for DBP, though it did not reach statistical significance, $z = 1.56$, $p < .06$.

Table 2. NA associations with CVR when effort was high or low: standardised and unstandardised regression coefficients of simple slope analyses.

	High effort	Low effort
Study 1		
Systolic blood pressure reactivity (mmHg)	.55** (9.36)	.02 (.29)
Diastolic blood pressure reactivity (mmHg)	.50** (4.09)	-.00 (-.03)
Heart rate reactivity (bpm)	-.01 (-.07)	.16 (1.75)
Study 2		
Systolic blood pressure reactivity (mmHg)	.27* (3.97)	-.04 (-.62)
Diastolic blood pressure reactivity (mmHg)	.22 [†] (1.85)	-.06 (-.54)
Heart rate reactivity (bpm)	.27* (2.81)	-.12 (-1.24)
Study 3		
Systolic blood pressure reactivity (mmHg)	.52** (10.56)	-.01 (-.23)
Diastolic blood pressure reactivity (mmHg)	.46* (6.00)	-.21 (-2.73)
Heart rate reactivity (bpm)	.56** (7.23)	-.15 (-1.88)

Notes: [†] $p < .09$, * $p < .05$, ** $p < .01$. The values in the parentheses are the corresponding unstandardised regression coefficients (B).

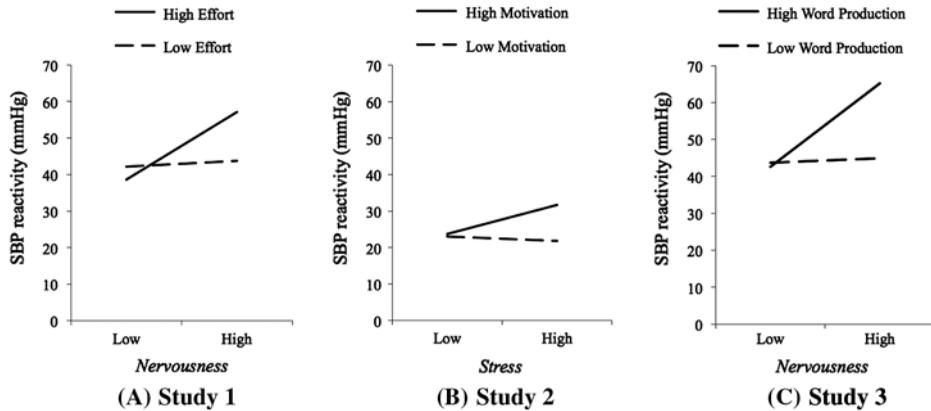


Figure 2. The interaction effect between effort and NA on SBP reactivity in studies 1, 2 and 3.

Study 2: Stress \times motivation during an anagram competition

The purpose of Study 2 was to test our hypothesis with a different laboratory task and different measures of effort and NA (Roy et al., 2005). The task in this study was an anagram competition task that required effort to succeed (i.e. to win the competition) and manipulated perceived task difficulty. The measures of effort and emotion were self-reports of ‘motivation to win’ and ‘stressfulness’, respectively.

Method

Participants and task

There were 121 (68 females) undergraduates from the University of California, San Diego that participated in the experiment for class credit.

The competitive anagram race was with another undergraduate who was actually a confederate of the experiment. As each participant and the confederate completed 1 of 12 pages each containing 8 anagram problems, they rang a bell to indicate they were moving on. In this experiment, perceived task difficulty was manipulated by coordinating whether the confederate was consistently ahead of (rang his bell before) the participant, consistently behind (rang his bell after) the participant on each of the 12 anagram pages or the confederate switched from being behind/ahead for the first six pages to being ahead/behind for the last six pages. After all 12 pages of anagrams were completed by one of the competitors, questionnaires measuring NA and motivation were completed.

Measures and analyses

SBP, DBP and HR readings were recorded with an Ohmeda Finapres, and the calculation of CVR was identical to that in Study 1. After the task, the participants rated how stressed they felt and how motivated they were to win [1 (*not at all*) to 7 (*very much*)].

We performed the same analyses as in Study 1. Once again, we collapsed across conditions, and in hierarchical regression analyses, we controlled for condition in step 1.

Results and discussion

Preliminary analyses

The anagram competition task significantly increased blood pressure and HR from a resting baseline (Table 1). In contrast to Study 1, the correlation between motivation to win (effort) and stressfulness (NA) was positive, $r(119) = .21$, and statistically significant, $p < .05$. Baseline HR was significantly correlated with HR reactivity [$r(119) = -.30$, $p < .01$].

Hypothesis testing

Regression analyses revealed that self-reported stress was not significantly associated with CVR (SBP reactivity, $\beta = .11$, $t = 1.23$; DBP reactivity, $\beta = .08$, $t = .84$; and HR reactivity, $\beta = .07$, $t = .83$; all $ps > .05$). Greater motivation was significantly associated with larger increases in CVR (SBP reactivity, $\beta = .18$, $t = 1.91$, $p < .06$; DBP reactivity, $\beta = .22$, $t = 2.36$, $p < .05$; HR reactivity, $\beta = .30$, $t = 3.32$, $p < .05$).

The stress by motivation interaction was marginally significant for SBP reactivity, $\beta = .17$, $t = 1.81$, $p = .07$, not statistically significant for DBP reactivity, $\beta = .15$, $t = 1.64$, $p = .10$, and significant for HR reactivity, $\beta = .20$, $t = 2.29$, $p < .05$. This result remained significant when we controlled for baseline HR. Although the interaction results did not reach statistical significance for SBP and DBP reactivities, to see if data were in line with the same patterns as those found in Study 1, we performed simple slope analysis on the three CVR measures.

Simple slope analyses showed that when participants reported being relatively highly motivated (+1SD) during the competition, self-reported stress was significantly, positively associated with SBP and HR reactivity, and marginally significantly associated with DBP reactivity (Table 2). In contrast, when participants reported having relatively low motivation (-1SD), stress was not significantly related to CVR (Table 2). Figure 2B shows the stress by motivation interaction effect on SBP reactivity. Effects on DBP and HR had the same pattern.

The results of Study 2 showed that an effort by NA interaction effect on CVR is not specific to reports of nervousness and effort, but to the other relevant constructs related to NA and effort. Also, the effort by NA interaction effect was observed in response to a competitive anagram task, suggesting that the effect is not specific to public speaking. NA accounted for more variance in CVR when motivation to win was considered and was high (7.18, 4.71 and 7.02% of the variance in SBP, DBP, and HR reactivity, respectively) than when effort was not accounted for (1.21, .64 and .49% of the variance in SBP, DBP and HR reactivity, respectively). However, Fisher's z transformation tests showed that these differences were not statistically significant, $zs < 1.10$, $ps > .05$.

Study 3: Nervous \times word production during public speaking

To further extend the findings of Studies 1 and 2, we examined a behavioural indicator of effort that would avoid post-task self-report biases. A behavioural measure of effort allowed us to see if the effort by NA interaction effect on CVR depended on subjective recollections of effort or if actual effort expended during a stressful task would lead to the same pattern of results.

Method

Participants and task

Sixty-four undergraduate females from the University of California, San Diego participated in this experiment. To induce stress, the participants performed a five-min public speaking task on the topic 'College is a valuable asset' in front of two audience members described to the participants as experts or novices in public speaking (Hilmert, Christenfeld et al., 2002). During the speech, audience members either provided positive (supportive) or neutral (non-supportive) feedback to the participants. Once again, as the experimental conditions were not relevant to the present hypotheses, we collapsed data across conditions.

Measures and analysis

This experiment measured CVR using the same method as Studies 1 and 2. The NA measure asked 'how nervous the audience made you feel', [1 (*not at all*) to 5 (*very much*)]. To assess effort in Study 3 the number of words spoken by the participant during the speech task was counted. The assumption was that more words produced indicated greater effort (Hilmert, Christenfeld et al., 2002). The number of words produced ranged from 270 to 1064 words. We performed the same series of analyses as in Studies 1 and 2.

Results

Preliminary analyses

The public speaking task significantly increased blood pressure and HR from a resting baseline (Table 1). Consistent with Study 1 and in contrast to Study 2, correlation analyses revealed a significant negative association between nervousness and word production, $r(62) = -.38$, $p < .01$. Baseline cardiovascular measures were not significantly correlated with reactivity measures in this study (all $r_s > .05$).

Hypothesis testing

After controlling for condition, separate regression analyses revealed that participants who reported being more nervous during their speech had significantly greater SBP reactivity, $\beta = .25$, $t = 2.04$, $p < .05$. Self-reported nervousness was not significantly associated with DBP reactivity, $\beta = .13$, $t = .97$, $p > .05$, or with HR reactivity, $\beta = .21$, $t = 1.70$, $p > .05$. Also, regression analyses showed that there was a significant effect of word production on SBP reactivity such that participants who produced more words (put forth more effort) had greater SBP reactivity, $\beta = .25$, $t = 2.04$, $p < .05$. There were no significant main effects of word production on DBP, $\beta = .13$, $t = .99$, $p > .05$, and HR reactivity, $\beta = .05$, $t = .42$, $p > .05$.

The nervousness by word production interaction explained a significant amount of variance in SBP, $\beta = .23$, $t = 2.00$, $p = .05$, DBP, $\beta = .29$, $t = 2.43$, $p < .05$ and HR reactivity, $\beta = .31$, $t = 2.71$, $p < .05$. Simple slope analyses confirmed that when participants were putting forth relatively more effort (+1SD), as indicated by producing more words, there was a significant positive association between nervousness and CVR. However,

when participants were putting forth relatively little effort ($-1SD$) there was no association between nervousness and CVR (see Table 2). Figure 2C shows the nervousness by word production interaction effect on SBP reactivity. The interaction effect patterns for DBP and HR reactivity were similar.

Similar to Studies 1 and 2, NA accounted for more variance in CVR when effort was accounted for and high than when effort was not accounted for, $z_s > 1.77$, $ps < .05$. Specifically, when the word production was not taken into account, NA explained for 6.25, 1.69 and 4.41% of the variability in SBP, DBP and HR reactivity, respectively. When the number of word production was taken into account and effort was high ($+1SD$), NA explained 26.94, 21.53 and 31.25% of the variance in SBP, DBP and HR reactivity, respectively.

Meta-analyses

We conducted meta-analyses on the primary results of Studies 1, 2 and 3. These analyses followed the procedures described by Rosenthal (1984). First, we converted p values into standardised z scores. Then we summed the z scores and divided the sum by the square root of the number of studies to get a combined z score. The p value associated with the combined z score indicates the overall significance level (one tailed).

We first examined whether, overall, the NA by effort interaction effect was statistically significant. The results were significant for SBP reactivity, $z = 3.41$, $p < .001$, DBP reactivity, $z = 3.46$, $p < .001$ and HR reactivity, $z = 2.44$, $p < .01$. The overall effect sizes for SBP, DBP and HR reactivity were .22, .23 and .15, respectively.

Next we tested whether, overall, the association between NA and CVR was significant when effort was high. That is, we analysed the results of simple slopes analyses. The results showed that when effort was high, the combined z tests were significant for SBP reactivity, $z = 4.71$, $p < .001$, DBP reactivity, $z = 4.04$, $p < .001$ and HR reactivity, $z = 3.00$, $p = .001$. When effort was low, the combined z tests were not significant for SBP, DBP and HR reactivity, $z_s < -.17$, $ps > .19$. Thus, these analyses suggest that overall, there was a significant positive association between NA and CVR when effort was high, but not when effort was low.

To estimate the overall effect size of NA on CVR when effort was high or low, we combined the effect sizes of our findings. The effect size estimates were the beta (standardised) coefficients of the simple slope analyses that regressed CVR on NA when effort was high or when effort was low. We transformed each of the effect sizes to a Fisher's z coefficient. Next we computed mean z scores by summing the respective z s and dividing the sum by the number of studies (i.e. 3). The mean z scores were then each transformed back into an r coefficient (Rosenthal, 1984).

When effort was high, the overall effect sizes on SBP, DBP and HR reactivity were .46, .40 and .29, respectively. When effort was low, the overall effect sizes on SBP, DBP and HR reactivity were $-.01$, $-.09$ and $-.04$, respectively. The effect sizes in high and low effort differed significantly from each other in SBP, DBP and HR reactivity, $z_s > 3.70$, $ps < .001$. In other words, when participants put forth effort, 20.70% of the variance in SBP, 16% of the variance in DBP and 8.47% of the variance in HR reactivity were accounted for by NA. When participants had low effort, NA explained only .01% of the variance in SBP, .83% of the variance in DBP and .14% of the variance in HR reactivity.

General discussion

Our hypotheses were supported in three stress–reactivity experiments. The interaction between how much NA participants experienced during a stressor and how much effort they put forth during the stressor predicted a consistent pattern of CVR. The results of the present studies indicate that NA was positively associated with CVR when participants devoted relatively high amounts of effort and was unrelated to CVR when participants devoted little effort. Furthermore, participants who exerted relatively high amounts of effort and had relatively strong NA responses to laboratory stressors tended to exhibit the greatest increases in blood pressure and HR in response laboratory stressor tasks.

Past laboratory studies of negative emotion and CVR found that negative emotion explained only 2–12% of the variance in blood pressures and HR (Feldman et al., 1999). Similarly, when effort was not taken into consideration our analyses revealed that NA explained 1.21–8.41% of the variance in SBP reactivity, .64–6.25% of the variance in DBP reactivity, and .49–4.41% of the variance in HR reactivity. However, our three studies revealed that, when effort was considered as a moderator and it was high, NA explained 20.70% of the variance in SBP reactivity, 16% of the variance in DBP reactivity and 8.47% of the variance in HR reactivity. NA was unrelated to CVR when participants put forth little effort.

Our results suggest that the effort by NA interaction effect on CVR may be robust. We found similar patterns of an effort by emotion interaction effect on CVR during two very different tasks. In addition to the speech and anagram competition tasks requiring very different behavioural responses, they elicited notably disparate effort-NA response patterns. During the speech tasks (Studies 1 and 3) greater effort was associated with less NA, while during the competitive anagram task (Study 2) greater effort was significantly associated with more NA. Despite these divergent psychological patterns, the same effort by emotion interaction effect on CVR was evident during both tasks, albeit to a somewhat lesser extent during the anagram competition.

We tested our hypothesis with three measures of effort, two self-reported measures ('effort' and 'motivation to succeed') and one behavioural measure of effort (number of words produced). Also, we had two self-reported measures of NA (stressfulness and nervousness) across studies. Despite this variation, the effort by NA interaction association with CVR remained apparent. These conceptual replications of the effort by NA interaction pattern suggest that this effect generalises across tasks and applies to effort and NA generally.

Theoretical implications

Our results suggest that to best understand the connections between stress psychology and stress physiology, we should consider the multidimensional nature of these responses (Hilmert & Kvasnicka, 2010). Here we have shown that variance in effort and NA responses to stress together influenced CVR. The reason why this interaction is associated with CVR, however, is not entirely clear.

Physical effort creates metabolic demand for increased blood flow to muscles being used (Obrist, 1981). Thus, physical effort can have a direct effect on cardiovascular functioning. Consistent with this, research has found that simply speaking causes blood

pressure increases (Linden, 1987). High NA may indicate that more effort may be necessary, causing CVR to exceed metabolic demands, particularly when some effort is already being exerted. When effort is not being exerted, it is not clear what NA may indicate that does not arouse the cardiovascular system. Perhaps it is a need to conserve energy for other, less demanding situations or perhaps energy is diverted elsewhere in these circumstances.

From another perspective it may be that different combinations of effort and NA in the present studies may have indicated qualitatively different emotional episodes (Russell & Feldman Barrett, 1999) that were more or less likely to be associated with CVR. That is, strong NA combined with high effort to complete a stressful task may have indicated an approach-emotion episode, perhaps resembling an anger response more than other combinations of effort and NA (see Figure 1; Carver & Harmon-Jones, 2009). This is consistent with theories of affect that focus on other dimensions of a circumplex model of emotions (Yik, Russell, & Barrett, 1999). For instance, a perspective that considers the NA and positive affect (PA) independent qualities of emotion (Watson & Clark, 1992), anger is clearly a high NA emotion because it has negative valence and high arousal characteristics.

Consistent with our findings, anger has been associated with CVR in past research (e.g. Chida & Hamer, 2008; Siegman, Anderson, Herbst, Boyle, & Wilkinson, 1992; Waldstein et al., 2000). In contrast, strong NA combined with little effort to complete the task may have indicated a withdrawal-emotion episode, resembling a sadness or hopelessness response more than other combinations of effort and NA. Consistent with our findings, sadness has not been associated with increased CVR in past research (Chida & Hamer, 2008; York et al., 2007).

The range of effort responses to a stressor may be evident in overt behaviour, as we saw with word production in Study 3. On the other hand, participants' behaviour is likely indicative of an underlying psychology, e.g. motivation or activation, which may be a key component determining if an emotional episode is significantly related to cardiovascular functioning and may be manifested in different ways; for instance, in some situations constraining a speech to fewer words may require more effort than producing more words. That is, perhaps it is possible to be energised and motivated, but to perform no overt act, perhaps, to simply endure the situation. Future research will have to untangle whether psychological activation, physical effort or qualitatively different emotional episodes best explain the effect we report here.

Practical implications

There is convincing evidence that over time large magnitude increases in blood pressure in response to stress may contribute to the development of CVD (Krantz & Manuck, 1984; Treiber et al., 2003). If CVR is primarily influenced by effort (Wright & Kirby, 2001), then it follows that effort can be bad for one's health (Hilmert & Kvasnicka, 2010). However, our findings suggest that high effort *combined with* strong NA leads to the largest magnitude CVR. It seems more likely that high effort-, strong NA-associated blood pressure responses to stress put individuals at risk for CVD more so than simply high effort alone.

It is conceivable that putting forth effort when there are few negative feelings is characteristic of a comparably constructive response to stress. Though there is reactivity associated with a high effort, weak NA response, it may not reach the magnitude necessary to damage the cardiovascular system and contribute to CVD. Also, the CVR associated with a weak NA, high effort response may involve a combination of underlying hemodynamic factors (e.g. vascular resistance, cardiac output) that is less harmful to health (Hilmert & Kvasnicka, 2010; Tomaka, Blascovich, Kelsey, & Leitten, 1993).

Assuming that strong NA combined with high effort puts a person at greatest risk of CVD relative to other combinations of NA and effort, our findings provide some potentially important information regarding how one should respond in a stressful situation. Putting forth effort in a stressful situation may be a good way to cope with a stressor, but only when negative feelings are minimal. Putting forth effort while feeling very negatively about a situation may be, as we have speculated, a response resembling anger, which research has repeatedly shown to be associated with high CVR and CVD (Smith, Glazer, Ruiz, & Gallo, 2004).

Another notable pattern in our results involved participants who reported high NA and low effort. The CVR of high NA, low effort participants was lower than that of the high NA, high effort participants. One implication of this pattern is that a high NA, low effort response to stress may not be a risk factor for CVD, at least not through a stress-CVR mechanism (Treiber et al., 2003). However, there may be other mechanisms through which a high NA, low effort or withdrawal response to stress impacts health. Depression and learned helplessness, both high NA, low effort/energy emotions have been associated with negative health outcomes, including CVD (Ormel et al., 2007). Thus, withdrawal may not always be a healthier response to stress than putting forth effort for reasons other than CVR.

Limitations and future directions

Although it is notable that we found the same pattern of results in three experiments not designed to test our hypothesis, our findings should be considered preliminary. Ideally, studies designed to independently manipulate NA and effort are needed. Similarly, a potential limitation of our studies is the use of single-item measures to assess NA. We note, however, that our interest was in indexing valence with the single-item NA measures and not measuring discrete emotions. As there are reliably strong correlations among ratings of similarly valenced emotions (Carroll et al., 1999), we are reasonably confident in the validity of the measures we used here. Nevertheless, future research should consider a broader range of negative emotion measures using multiple items and validated assessments of NA or valence quality.

Another limitation and potential future direction involves the inclusion of PA. It is not clear if there are different health benefits or costs to high or low effort while feeling intense PA, and whether associations between PA and CVR are moderated by an effort-related factor. The negative associations between PA and CVR have been documented (e.g. Steptoe, Gibson, Hamer, & Wardle, 2007), and therefore we expect that the effort-by-emotion interpretation is not likely for positive emotions. However, there is some suggestion that under certain circumstances, such as in patients recovering from coronary artery surgery, strong PA can be harmful to one's

health (Diener & Chan, 2011). Future research should consider an effort by PA mechanism to explain these findings.

Our analyses extend research on stress psychophysiology, suggesting that research focus on the multidimensional nature of psychological responses to stressors. There are likely other important interactions involving a variety of psychological and behavioural responses to stress and their associations with cardiovascular, neuroendocrine, immune and other physiological responses that have health-related implications. A better understanding of these associations may help us identify at-risk individuals and improve interventions that better target the psychological predictors of unhealthy physiological responses to stress.

Although our findings are correlational, they suggest that the emotional and effortful qualities of our responses to stress may have important implications for health. It is possible that the situational consequences of an effortful response to stress, for instance, a more hasty resolution may outweigh the effects of strong NA and high effort on CVR. On the other hand, when a response to stress involves intense NA, our results suggest that it may be best to step away until the NA subsides, at which point putting forth effort to cope with the stressor will be associated with less CVR and may be better for one's cardiovascular health.

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