Int. J. Behav. Med. (2010) 17:189–194
DOI 10.1007/s12529-009-9063-3

Emotion, Stress, and Cardiovascular Response: An Experimental Test of Models of Positive and Negative Affect

Haulie Dowd · Alex Zautra · Michael Hogan

Published online: 18 September 2009

Abstract
Background The nature of the relationship between positive affect (PA) and negative affect (NA) has been a topic of debate for some time. In particular, there are gaps in our knowledge of the independent effects of PA and NA on health under stress.
Purpose The study examined the effects of a laboratory-induced stressor on the experience of PA and NA, and the effects of affect on cardiovascular (CV) reactivity and recovery.
Method A sample of 56 female college students was randomly assigned to a public speaking (stress) task or a silent reading (control) task. Pre- and posttask PA and NA were measured using the Positive and Negative Affect Schedule (PANAS Watson J Pers Soc Psychol 54:1,063-1,070, 1988). Baseline, task, and posttask cardiovascular measures were also recorded.
Results The results indicated that PA and NA responded differently to the stressor and contributed independently to the prediction of both CV reactivity and recovery. Of particular interest was the finding that higher levels of both PA and NA predicted greater CV recovery.
Conclusion Results are discussed in light of the debate concerning the (in)dependence of positive and negative emotions and the importance of understanding the dynamics of emotions, stress, and health.

H. Dowd · M. Hogan (✉)
Department of Psychology, NUI,
Galway, Ireland
e-mail: michael.hogan@nuigalway.ie

A. Zautra
Department of Psychology, Arizona State University,
Phoenix, AZ, USA

Keywords Stress · Affect · Cardiovascular reactivity · Health

Introduction

For some time now there has been a debate on the nature of the relationship between positive affects (PA) and negative affects (NA). Some argue for a bipolar model with PA and NA inversely correlated (e.g., [1]). Others argue for a bivariate model with independent but covarying PA and NA (e.g., [2]). Most researchers investigating the relative merits of bipolar versus bivariate models of affects and other psychological states have constructed and tested measurement models with which to gage the relative independence of positive and negative experience. Another approach, and the one adopted here, has been to examine whether distinguishing between positive and negative states is useful in understanding how stressful events influence behavior. In this study, we examine the merits of a bivariate versus bipolar approach to the prediction of affective and cardiovascular (CV) responses to a laboratory stressor.

Increasingly, researchers have pointed to the beneficial effects of positive emotion on health and longevity (see [3]) independent of negative emotions. Fredrickson and Levenson [4] found direct evidence that positive emotion can quicken cardiovascular recovery. More generally, the dynamic model of affect (DMA; 10 proposes that PA and NA often function independently in shaping health outcomes, and that stress may alter the way PA and NA function. Indeed, evidence from several quarters points to the conclusion that positive events and emotions have their greatest influence on negative emotions and CV health when the person is under stress (see [5] for a review). In a meta-analysis on the effects of laboratory induced stressors, Feldman et al. [6] found an association

between an increase in negative emotion and CV reactivity. The amount of variance accounted for was small and ranged between 2% and 12%. However, this research did not examine positive emotions. Maier, Waldstein, and Synowski [7] found that PA before a stressor, but not NA was associated with more CV reactivity during a stressor task. Somewhat typical of studies of this kind, Maier et al. [7] did not examine CV recovery. Consequently, there are gaps in our knowledge about the independent effects of positive and negative effect on CV reactivity and recovery during a stress episode.

A good test of the idea that positive emotions have their greatest influence on CV health when the person is under stress is to examine the effects of PA and NA on CV reactivity and recovery in a group exposed to stress and group not exposed to stress. The current experiment examined changes in both affect and cardiovascular responding in the context of a public speaking task (experimental stress group) and a silent reading task (control group). We predicted a significant effect of public speaking, but not silent reading, on PA, NA, and CV reactivity. Consistent with bivariate models of affect (cf. [8]), we predicted independent effects of stress on PA and NA and independent effects of NA and PA on CV reactivity and recovery. Finally, consistent with DMA we predicted that independent effects of NA and PA on CV reactivity and recovery would be greatest in the experimental stress condition.

Method

Design and Statistical Analyses

A 2×2 (time: pretest/posttest)×(stress: stress/no stress) design was used. Analysis of variance (ANOVA) was used to examine the effect of stress on affect over time. Multiple regression was used to examine the effects of stress, affect, and stress×affect interactions on CV reactivity and recovery.

Participants

Participants were 56 women, with a mean age of 18.9 (*SD* = 2.87; *range* 17–35) years, recruited from a sample enrolled in an undergraduate course in psychology at NUI Galway, who received course credits for taking part in the study. Participants had to be between 17 and 40 years of age and have no history of hypertension to be included in the study. Of those who were within the appropriate age-range, none had a history of hypertension. A female only sample was selected to avoid confounding by gender differences in CV reactivity to stressors [9]. Based on available prescreening data, those assigned to the stress and no stress conditions were matched on measures of trait anxiety (STAI, Form Y-2, [10]). Caffeine and nicotine use was not restricted as this may have produced unpredictable effects of withdrawal on affect and CV reactivity to stress [11]. We recorded smoking behavior (number smoked daily and time smoking), but found no correlation with measures of affect or cardiovascular responding.

Affect Measurement

The PANAS [12] was used to measure PA and NA. The scale consists of 20 adjectives describing different feelings and emotions; ten describe positive moods, and ten negative moods.

Apparatus

A MS 700 automatic digital blood pressure monitor, manufactured by Mars Corporation, was used to measure systolic and diastolic blood pressure and heart rate.

Experimental Task

Participants in the experimental condition completed an evaluative speaking task. Each participant was asked to prepare a speech defending themselves in a role-play scenario where they had been falsely accused of shoplifting. This task has been shown to be successful in eliciting cardiovascular responses [13]. Participants in the control condition were asked to read psychological journal articles for the duration of the experiment.

Procedure

Informed consent was obtained, and participants were tested individually. After the digital blood pressure monitor was attached, baseline systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) readings were taken at the end of 1, 2.5, and 4 min. During this time, participants were asked to sit quietly and were offered magazines to read. Participants then completed the PANAS. Participants in the experimental condition were then given 3 min to prepare for the speech task, and, after a cue was provided to begin, they then had 5 min to deliver their speech. While participants spoke, SBP, DBP, and HR were measured at the end of 1, 2.5, and 4 min. Participants in the control condition read articles from psychological journals, during which time their blood pressure and heart rate measured at the same time intervals as those in the experimental group. At the end of the experimental phase, participants in both groups again completed the PANAS. Participants were then given 5 min to relax and SBP, DBP,

and HR recovery readings were taken at the end of 1, 2.5, and 4 min.

Results

Affect

Two 2 (time)× 2 (stress) ANOVAs run separately on the two affect measures revealed the following pattern of results: For PA, there was a main effect of Time, F (1, 54) = 8.98, p= .004, η^2 = .143, with PA before the experimental condition (time 1) significantly higher than PA after (time 2), and a time×stress interaction effect, F (1, 54) = 4.14, p= .047, η^2 = .071, with a significant decrease in PA for the stress group (p= .002) and a nonsignificant decrease for the no stress group (p> .05; see Table 1).

For NA, there was a main effect of Time, F (1, 54) = 12.32, p< .001, η^2 = .186, and stress, F (1.54) = 29.82, p< .001, η^2 = .356. NA was higher in the stress group and higher at time 2 compared with time 1. Further, there as a significant time×stress interaction effect, F (1.54)=25.64, p<.001, η^2=.322; NA for the no stress-group tended to reduce but not significantly (p>.05), whereas NA for the stress group increased from time 1 to time 2 (p<.001).

Cardiovascular Responding

Average heart rate, systolic blood pressure, and diastolic blood pressure were calculated for each of the three periods: baseline, task, and posttask. Table 1 lists means and SDs. Controlling for baseline cardiovascular responding, a series of six forward step-wise multiple regressions were conducted to examine the effect of group (stress, no stress), pretask PA and NA, PA×NA, and group×affect interactions on heart rate reactivity and recovery and systolic and diastolic blood pressure reactivity and recovery. There was an effect of stress group on systolic and diastolic blood pressure reactivity and recovery (see Table 2). A series of six one-sample t tests indicated no significant changes in BP or heart rate over time in the silent reading group. The public speaking group showed significant increases in systolic and diastolic BP reactivity during the task and significant decreases in systolic and diastolic BP recovery after the task (p<.05 for all four comparisons).

Both pretask PA and NA accounted for significant variance in systolic reactivity and recovery. However, significant stress group×affect interaction effects suggested that PA and NA were moderating systolic reactivity and recovery differently under conditions of stress versus no stress. Post hoc regression revealed that higher PA predicted with higher systolic BP reactivity for the stress group, β=.689, t=3.49, p<.005. Both higher PA, β=.558, t=3.00, p<.01, and higher NA, β=.468, t=2.86, p<.01, were significant predictors of greater systolic BP recovery. In the control group, higher pretask NA alone predicted lower systolic BP reactivity, β=−.338, t=−2.20, p<.03. A stress group×PA interaction effect was observed for both diastolic BP reactivity and recovery. Higher pretask PA predicted less diastolic BP reactivity (β=−.653, t=−3.106, p=.005) and recovery (β=−.552, t=−2.663, p=.014) in the control group. In the stress group, there were no effects of affect on diastolic reactivity or recovery. There were no effects of stress or affect on pulse reactivity and recovery.

Discussion

The current study examined the effects of stress on the experience of positive and negative affect (PA and NA), and the effects of affect and expectancy on cardiovascular reactivity and recovery. We found that, compared with the pretask and posttask affective experiences of a control group reading psychology journal articles, those participants who were asked to deliver a speech into a camera reported a significant increase in NA and a significant reduction in PA. However, the effect of stress on NA was far more pronounced than the effect of stress on PA, with the time×stress interaction effect accounting for 32.2% of the variance in NA, but only 7.2% of the variance in PA.

Table 1 Means and SDs of pulse, systolic BP, Diastolic BP, PA, and NA for stress (N=28) and no stress (N=28) groups

	No stress			Stress		
	Baseline	Task	Posttask	Baseline	Task	Posttask
Pulse (b/min)	73.71 (11.12)	71.33 (10.95)	70.94 (10.48)	80.49 (11.70)	81.90 (16.87)	76.27 (11.33)
Systolic (mmHg)	99.74 (12.07)	98.75 (11.61)	98.52 (12.24)	101.49 (10.78)	128.21 (17.92)	102.00 (11.73)
Diastolic (mmHg)	64.43 (7.32)	63.58 (8.01)	63.54 (7.75)	66.64 (8.14)	92.39 (21.83)	68.68 (9.91)
PA	29.32 (6.28)	–	28.71 (7.28)	27.93 (5.12)	–	24.75 (7.30)
NA	12.79 (3.08)	–	11.82 (2.91)	14.89 (3.44)	–	20.21 (6.60)

Table 2 The effects of stress group, affect, and stress×affect interactions on systolic and diastolic reactivity and recovery

Dependent variable			Predictor variables			
	F	*p*		R^2	β	*p*
Systolic reactivity	19.68	.000				
			Baseline SBP	0.010	−.103	.180
			Stress	0.552	.793	.000
			PA before	0.058	−.856	.002
			NA before	0.033	−.641	.016
			PA×NA	0.000	.015	.864
			Stress×PA	0.079	1.026	.000
			Stress×NA	0.042	.717	.008
Diastolic reactivity	6.74	.000				
			Baseline DBP	0.015	−.129	.246
			Stress	0.365	.654	.000
			PA before	0.037	−.699	.067
			NA before	0.000	.023	.949
			PA×NA	0.012	−.129	.298
			Stress×PA	0.049	.816	.037
			Stress×NA	0.000	.044	.902
Systolic recovery	27.51	.000				
			Baseline SBP	0.000	−.004	.953
			Stress	0.602	.828	.000
			PA before	0.024	−.552	.021
			NA before	0.036	−.664	.005
			PA×NA	0.004	.076	.332
			Stress×PA	0.041	.741	.003
			Stress×NA	0.053	.814	.001
Diastolic recovery	6.37	.000				
			Baseline DBP	0.001	−.030	.788
			Stress	0.292	.585	.000
			PA Before	0.033	−.665	.086
			NA before	0.001	−.117	.750
			PA×NA	0.012	−.131	.295
			Stress×PA	0.056	.877	.027
			Stress×NA	0.007	.299	.417

Also, for participants delivering a speech to camera (N= 28), PA and NA both contributed independently to the prediction of CV reactivity and recovery. Higher pretask PA was associated with greater systolic blood pressure reactivity. Surprisingly, both higher pretask PA and higher pretask NA independently predicted swifter recovery. In the control group, higher pretask NA predicted less systolic BP reactivity, and higher baseline PA predicted less diastolic BP reactivity and recovery.

These findings are interesting for a number of reasons. First, they add to the existing body of knowledge on the relationship between PA, NA, and stress when we found that, although PA decreases as NA increases during a stress episode, the magnitude of the changes accounted for by stress differ. This supports the view that, as neither the bipolar nor bivariate approach adequately explains the dynamics of affect during a stressful episode, a contextual model may be more appropriate.

Consistent with this contextual view, we observed different effects of pretask affect on patterns of cardiovascular reactivity and recovery under different task conditions. How might we explain the counterintuitive finding that the more intense were positive emotions prior to delivering a speech, the greater were systolic blood pressure changes from pretask to task (i.e., SBP reactivity)? Importantly, researchers have identified different patterns of sympathetic nervous system response associated with "distress" and "effort" For example, Blascovich and Tomaka [14] have noted that an "effort" or "challenge" response is associated with increased cardiac responding

and reduced vascular responding, whereas a "distress" or "threat" response tends to produce reduced cardiac responding and increased vascular responding. The effort/challenge pattern of physiological responding is much more likely when personal abilities are appraised as congruent with situational demands.

In the current study, it may be that those participants with higher baseline PA were also those who perceived themselves as having higher self-efficacy when they entered the laboratory situation (i.e., personal abilities were appraised as congruent with situational demands). When it came to delivering their speech, they may have perceived it as more of a challenge than a threat, and this in turn would explain why they had higher systolic reactivity when compared to those with lower baseline PA. However, we did not have available a full hemodynamic profile [15], and further studies in this area need to tease apart the relationship between the hemodynamic profile and the performance profile of those who experience different amounts of both negative and positive emotions when engaging in potentially "challenging" or "stressful" tasks.

Also consistent with the bivariate view, was the finding that both more positive and more negative emotion prior to the speech were associated with greater systolic blood pressure changes from task to posttask (i.e., SBP recovery). Rather than speculating about distinct or relative "challenge" versus "stress" patterns of responding, an alternative hypothesis is that when entering a situation where one is uncertain about the nature of the task to be performed, emotions associated with both perceived "challenge" and potential "threat" can be coactive. This greater state of activation could be characterized as greater "emotional complexity" and a "readiness" to engage the task and defend against any lasting impact of the stressor on physiological equilibrium. Alternatively, it might reflect a more "conflicted" state. After completing the task, it is conceivable that those participants who were in this "conflicted" state (i.e., experiencing both relatively high PA and NA simultaneously) would also be most likely to experience the greatest "relief". However, if this were true, we might have expected to observe a significant PA×NA interaction effect on recovery. We did not observe such an effect. As such, we found no evidence in support of the conflicted state hypothesis.

We observed different effects of affect on patterns of cardiovascular responding in the control group. In particular, higher pretask NA predicted lower systolic BP reactivity, and higher baseline PA predicted lower diastolic BP reactivity and recovery. These results are not inconsistent with the pattern of results observed in the stress group, in the sense that they may represent an adaptive adjustment of cardiovascular responding for those who entered the laboratory ready for a "challenging" or a "stressful" task. In the absence of significant "challenge" or "stress" during the silent reading task, it may be that those who entered the laboratory anticipating task demands responded with a greater relative relaxation response. Again, further research using hemoodynamic profiling is needed to better understand these subtle relationships between emotions, task demands, and different patterns of cardiovascular responding associated with "distress" and "effort".

In their dynamic model of affect, Zautra and colleagues (cf. [8]) posit that the relationship between PA and NA is contextual, that is, changes as a function of ongoing events, and personality features. In our study, not only did PA and NA have independent effects on cardiovascular reactivity and cardiovascular recovery, the stress context appears to have made a difference in these relationships. Contextual models such as the DMA are needed to fully understand how affective processes may contribute to adaptive functioning. More generally, dynamic system modeling is needed to assess changing patterns of relationship between emotional responding, physiological responding, and behavioral performance [16].

Fredrickson and Lasoda [17] have suggested that a specific ratio of positive to negative affect may provide a balance of emotions under which humans flourish. However, any such search for the optimal interplay between positive and negative affective states needs to account for contextual factors that shape affective and task demands. Also, future research that examines the nature of stress reactivity itself will need to focus on the independent effects of both positive and negative affects and expectancies. A plan of research designed to map the subtle contextual dynamics in the relationship between emotions, stress, and health presents us with a formidable agenda, but the new insights gleaned will help us to better understand the conditions that maximize resilience in the face of stress.

References

1. Russell JA, Carroll JM. On the bipolarity of positive and negative affect. Psychol Bull. 1999;125:611–7.
2. Watson D, Clark LA. Towards a consensual structure of mood. Psychol Bull. 1985;98:219–35.
3. Vaillant GE. Aging well: surprising guideposts to a happier life from the landmark Harvard study of adult development. Boston: Little, Brown, and Company, 2002.
4. Fredrickson BL, Levenson RW. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. Cogn Emot. 1998;12:191–220.
5. Zautra AJ. Emotions, stress and health. Oxford: Oxford University Press; 2003.
6. Feldman PJ, Cohen S, Lepore SJ, Matthews KA, Kamarack TW, Marsland AL. Negative emotions and acute physiological responses to stress. Annals Behav Med. 1999;21:216–22.
7. Maier KJ, Waldstein SR, Synowski SJ. Relation of cognitive appraisal to cardiovascular reactivity, affect, and task management. Annals Behav Med. 2003;26:32–41.

8. Reich JW, Zautra AJ, Davis M. Dimensions of the affect relationship: models and their implications. Rev Gen Psychol. 2003;7:66–83.
9. Stroud LR, Niagura RS, Stoney CM. Sex differences in cardiovascular reactivity to physical appearance and performance challenges. Int J Behav Med. 2001;8:240–50.
10. Speilberger CD, Gorsuch DL, Lushene RE. Manual for the state-trait anxiety inventory. Palo Alto: Consulting Psychologists Press; 1970.
11. James JE, Gregg ME. Hemodynamic effects of dietary caffeine, sleep restriction, and laboratory stress. Psychophysiology. 2004;41 (6):914–23.
12. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. J Pers Soc Psychol. 1988;54:1063–70.
13. Hughes JW, Stoney CM. Depressed mood is related to high-frequency heart-rate variability during stressors. Psychosom Med. 2000;62:796–803.
14. Blascovich J, Tomaka J. The biopsychosocial model of arousal regulation. Adv Exp Soc Psychol. 1996;28:1–51.
15. Gregg ME, Matyas TA, James JE. A new model of individual differences in hemodynamic profile and blood pressure reactivity. Psychophysiology. 2002;39(1):64–72.
16. Chow SM, Ram N, Boker SM, Fujita F, Clore G. Emotion as a thermostat: representing emotion regulation using a damped oscillator model. Emotion. 2005;5(2):208–25.
17. Fredrickson BL, Losada MF. Positive affect and the complex dynamics of human flourishing. Am Psychol. 2005;60:678–86.