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SELF-REPORTED STRESSORS, SYMPTOM COMPLAINTS AND PSYCHOBIOLOGICAL FUNCTIONING I: CARDIOVASCULAR STRESS REACTIVITY

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Abstract—Taking into account neuroticism and lifestyle variables (smoking and alcohol consumption), cardiovascular and psychological reactions to stressful films were studied in four groups of subjects selected on self-reported levels of recent stressor load and symptom complaints (low-load/low-symptoms; low-load/high-symptoms; high-load/low-symptoms; high-load/high-symptoms). The films were known either to stimulate or to depress cardiovascular activity. The results showed that psychological reactions (distress; deactivation; openness/involvement) were associated with group membership and condition. In addition, high-symptom subjects were characterized by faster resting heart rates and smaller ECG T-wave amplitudes (TWAs), reflective of greater sympathetic tone on the myocard. Further, low-symptom subjects, in particular those labeled as stress-resistant (high-load/low-symptoms), exhibited larger myocardial responses to the cardiovascular-stimulating films than did high-symptom subjects. Low-symptom subjects showed predicted variations in physiological responses to the different films, whereas the responses by the high-symptom subjects showed lesser variation across films. It is concluded that the cardiovascular responses of low-symptom subjects more accurately followed the energetic demands of the body, whereas the high-symptom subjects were in a comparatively enduring state of arousal.

Keywords: Stressors, self-reported; Symptom complaints; Psychobiological functioning; Psychoneuroendocrine variables; Endocrine responsivity.

INTRODUCTION

A number of studies have investigated the ways in which emotional demands affect cardiovascular health, for example, in relationship with job stressors [1]. Similarly, the ways in which behavioral traits such as Type A behavior or other psychosocial factors affect cardiovascular reactivity to a short-term challenge or stressor have been studied extensively in the laboratory [2]. However, few studies have linked these two approaches, investigating the effects of exposure to stressful life conditions on cardiovascular responsivity to an acute, laboratory stressor. As expressed in the old adage "bad things come

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in threes", rarely do stressful things happen in the absence of other events. How previous or current real life stressor exposure affects responsivity to taxing events is a complex question, requiring measurement of not only the number, nature, and intensity of the events, but also of the extent and adequacy of coping. Given the (moderate) relationships between measures of experienced life events and health outcomes, it is important that the underlying mechanisms be clarified.

To our knowledge, only 7 investigations [3-9], yielding contradictory findings, have examined differences in cardiovascular reactions to standard stressors as a function of level of real-life stressors. Pardine and Napoli [3] failed to find any differences in baseline levels or in cardiovascular reactivity between students reporting many life events and a control group reporting few life events. However, the high-load individuals took longer to return to baseline levels following termination of the stressful stimulation. The authors suggest that these observations are in support of the slow-unwinding hypothesis put forward by Frankenhaeuser [10]. However, a more recent attempt to replicate these findings failed [4].

Jorgesen and Houston [5] tested the relation between differences in blood pressure responsivity and aspects of self-reported stressful life events. A significant inverse relation between diastolic blood pressure responsivity and level of life stressor exposure was observed. Comparable results were reported by Schaubroeck and Ganster [6], who found, amongst others, consistent negative relations between objective occupational demands and cardiovascular responsivity to acute challenges in the laboratory. Previously, Siegrist [7] showed that exposure to chronic occupational stressors was associated with reduced cardiovascular responsivity to a standard mental stressor at the end of a working day. In contrast, a positive association has been reported between the density of subjects' neighborhoods and the amplitudes of their cardiovascular reactions to a challenging task [8], reflecting higher stressor load leading to greater reactivity.

Finally, Opmeer and associates [9] reported a decreased heart rate reactivity after subjects experienced a demanding working day in comparison to responses following a restful day.

Fewer studies have focussed on the relationship between symptom reporting and psychophysiological functioning, though experiencing symptoms may serve as a sign of inadequate coping. As with the available studies of stressor load and reactivity, studies of symptom reporting and responses to acute stressors are marked by varied outcomes. Gannon and coworkers [11] found physiological reactivity to be a function of the relationship between symptom reporting and number of hassles. Those who had a strong positive association between symptom reporting and hassles showed greater reactions and slower recovery. Wientjes et al. [12] compared subjects with high and low scores on a symptom checklist and found that high-symptom subjects showed the lowest cardiovascular reactivity to standard laboratory stressors.

In sum, especially in comparison to studies dealing with single-challenge reactivity, there are only a limited number of studies addressing the relationship between cardiovascular reactivity and level of exposure to real-life stressors and/or subjective well-being. These few available studies fail to present a clear set of results. An additional observation is that the laboratory tests of reactivity most often involve measurements of responses to typical standard laboratory stressors such as mental arithmetic, Stroop test, or reaction time tasks. By design, these tasks are typically new to the subject, in order to control for prior experience. Although requiring considerable mental effort, these tasks often lack any strong specific emotional component and ecological validity.

Finally, attention is generally focussed solely on the sympathetic branch of the autonomic nervous system, ignoring indications that the parasympathetic system also may play an important role in cardiovascular stress reactions as well as in onset of myocardial dysfunction [13].

Related to this issue is the general tendency to appraise cardiovascular or, more generally, sympathetic reactivity only as a negative phenomenon with possible harmful long-term health consequences. It has often been suggested that individuals will differ as to their ability to respond effectively to new threats or challenges based on some discernable variable. Recently, Dienstbier [14] put emphasis on the positive aspects of peripheral arousal, introducing the concept "physiological toughness" and contending that repeated exposure to intermittent stressors leads to low sympathetic base rates, strong reactivity, and quick demobilization of energy. Therefore, differences in sympathetic reactivity alone may be inadequate for distinguishing among those that are under higher or lower stress loads with high or low perceived states of well-being. Instead, it is hypothesized that the full context of responses – reflecting cardiovascular stimulation and inhibition along with resting levels and recovery rates – will better represent the conditions of those who appear stress resistant versus stress vulnerable.

The aim of the present study, then, was to qualitatively compare psychobiological responses of subjects under high or low load and reporting many or few symptoms. Subjects were selected based on the extremity of their scores on stressor questionnaires and a symptom checklist. The concepts "stress-vulnerability" and "stress-resistance" were operationalized as, respectively, reporting a low load with many symptoms and reporting a high load with a low level of symptoms. Traditional views would predict that low-load subjects and those with few symptom complaints should respond less to the stressful stimuli than would their high-load and high-symptom counterparts. In contrast, but in concordance with Dienstbier's [14] conception of physiological toughness, one would predict low base rates, high reactivity, and quick recovery for stress-resistant individuals, that is, the high-load/low-symptom group.

It was expected that our selection procedure would maximize the likelihood of observing differences between these groups. Mindful of the remarks by Watson and Pennebaker [15] that self-reports of stressors and symptoms are both strongly associated with negative affectivity or neuroticism, this factor was also taken into account.

To conduct responsivity comparisons based on real-life stressor exposure, subjects were exposed to emotional, stressful films, which allowed for the systematic measurement of both sympathetically and vagally mediated cardiovascular stress responses. The films were of life-relevant stressful situations that have been shown to reliably elicit psychophysiological and emotional responses in previous research [16–18]. More precisely, the Driving Test and Rape films stimulate cardiovascular activity, as evidenced by increased heart rate, shorter pulse transit times (PTTs), and flattened ECG T-wave amplitudes (TWAs). In contrast, the two other films (Death Bed and Surgery) have been shown generally to induce an inhibition of the cardiovascular system, as reflected in a decrease in heart rate. Therefore, as a consequence of the type of stimuli used, we could systematically study both the activating and deactivating effects of stressful stimulation on the cardiovascular system, without the confounding of differences in mental or physical effort (this in contrast to typical laboratory stressors such as mental arithmetic, Stroop-test, or reaction time tasks).

The measurement strategy employed was designed to better reflect the dynamics of cardiovascular responsivity and adaptation. Cardiovascular measures with parameters such as TWA, PTT and heart rate variability (VAR) were used because these may provide more unequivocal information with respect to changes in autonomic balance, with TWA and PTT as potential indices of sympathetic tone on the myocard and VAR reflecting vagal influences on myocardial functioning (for a discussion see [16–20]). The design allowed study not only of baseline levels, but also of adaptation over the experimental session and reactions to different types of films. Therefore, in our analyses we make a distinction between baseline-levels, reactivity to films, and adaptation ("unwinding") over the experimental session.

METHODS

Subjects

Ninety-one males (age range 23–53 years) participated in this study, which formed part of the project "Psychosocial and psychobiological determinants of health and disease" at the Vrije Universiteit of Amsterdam. Participants were selected from an original sample of approximately 875 males who had participated in an earlier questionnaire portion of the project [21, 22]. This larger group had been recruited in two ways: about 200 participants were randomly sampled from two small villages in the Mid-Brabant area in the Netherlands; the remaining 675 had responded to advertisements in local newspapers and magazines and to announcements in several companies and clubs.

In order to assure that group selection was based on current stress profiles, subjects participated in the laboratory portion of the study within 4 to 6 weeks following completion of the questionnaires. As soon as questionnaires had been returned, scores on the Recently Experienced Events Questionnaire (REEQ) [23], based on Rahe's Recent Life Changes Questionnaire [24]; the Everyday Problem Checklist (EPCL) [25–27]; and the Hopkins Symptoms Checklist (HSCL) [28, 29] were calculated. Subsequently, raw data were transformed to z-scores utilizing the parameters obtained in a previous sample [25, 26] consisting of 461 males in the same age range (mean and standard deviation: REEQ: 19.02 [14.72]; EPCL: 25.63 [22.48]; HSCL 17.98 [17.78]). In order to be selected for the present study, participants had to meet the following criteria: either high (positive z-scores) or low scores (negative z-scores) on the REEQ and EPCL (reflecting high or low psychosocial load) as well as high or low scores on the HSCL (reflecting high or low levels of symptom complaints).

The following four groups were composed: (1) low-load, low-symptoms [LL/LS] (N = 23), (2) low-load, high-symptoms [LL/HS] (N = 22), (3) high-load, low-symptoms [HL/LS] (N = 22), and (4) high-load, high symptoms [HL/HS] (N = 24). It must be noted that the criteria for "high" and "low" differed between groups, because of the correlation between the self-reports of stressors and symptoms (r is approximately

Table I.—Characteristics of the four groups of subjects in terms of personality and coping measures (means + SD)

	LL/LS	LL/HS	HL/LS	HL/HS	*F*-values: Load	Symptoms	Interaction
N	23	22	22	24			
REEQ	6.7 (4.0)	9.2 (5.3)	38.3 (15.0)	63.2 (19.9)			
EPCL	8.5 (6.2)	13.2 (5.1)	51.9 (13.5)	98.5 (34.7)			
HSCL	5.0 (3.3)	34.0 (18.8)	11.0 (2.7)	67.3 (22.4)			
Age	39.9 (5.8)	38.4 (6.4)	35.3 (6.1)	38.1 (6.9)	3.5	<1.0	2.6
Neuroticism	4.7 (3.8)	12.6 (7.6)	6.9 (4.2)	24.0 (9.0)	24.1***	82.3***	11.2**
Cigarettes/day	2.8 (5.6)	9.3 (10.3)	5.2 (13.6)	10.9 (10.4)	1.5	8.4**	<1.0
Drinks/week	10.3 (8.5)	13.5 (14.8)	10.7 (8.7)	9.2 (7.1)	<1.0	<1.0	1.2

** $p < 0.01$
*** $p < 0.001$

0.52, see [21]). This implied that "high" and "low" in the HL/HS and LL/LS, respectively, were more extremely defined than in the two discordant groups.

Neuroticism was measured applying the Dutch Personality Inventory (DPI) [30]. In addition, information was collected concerning lifestyle, in particular, smoking and alcohol consumption. Thirteen potential candidates, for different reasons, did not accept the invitation to take part in the laboratory study. All participants received 50 Dutch guilders for their participation. For a full description of the groups' characteristics, see Table 1.

Stimuli

Subjects were exposed to six short (5- to 8-min) films. The first two were 'buffer' films to allow subjects to become acquainted with the experimental procedures, followed by four experimental, stressful films. The four stressful films represented (1) a driving test; (2) a woman dying at home and her funeral; (3) abdominal surgery after a car accident; and (4) rape. The first three films were chosen because previous research [16–18] and a pilot study (testing the rape film) had shown that they evoke strong psychological and differential psychophysiological stress reactions. As already indicated, the Death Bed and Surgery films both had reliably induced suppression of the cardiovascular system (CVS− films), whereas Driving Test and Rape had a general stimulating effect on the cardiovascular system (CVS+ films). For a more extensive description of the contents of the films, see Hettema et al. [31].

The sessions took place in a 7.2- × 4.6-m room that was designed to resemble a cinema. The subjects were seated in a comfortable chair about 4.5 m before a film screen (3.4 × 1.8 m). The projection quality was excellent and the room was completely darkened during the films and dimly lit between films. Registration equipment and the projector were housed in adjacent rooms.

Measures

Psychological measures. After each experimental condition, each subject completed an Adjective Checklist (ACL), which was based on one originally developed by Kjellberg and Bohlin [32]. It consists of 30 adjectives such as tense, sleepy, tired, curious, attentive, etc. Participants indicated on a 4-point scale (not at all, a little, rather, very) the degree to which the item applied to them while they were viewing the film that had just finished. Previous research [16] showed that three dimensions of this ACL can be distinguished: (1) Distress (with high loadings for restless, jittery, and tense), (2) Deactivation (with high loadings for drowsy, lethargic and dreamy), and (3) Openness/involvement (with highest loadings for interested, alert and attentive).

Cardiovascular measures. The following psychophysiological measures were obtained or calculated in order to obtain information concerning the relative contribution of both sympathetic and vagal influences on cardiovascular functioning [16–20]: ECG inter-beat interval (IBI), TWA, PTT, and VAR. ECG (Ag-AgCl) electrodes were attached to the left lower rib, just below the right clavicle, and to the left wrist. A photoelectric earpiece was attached to the pinna of the right ear for making plethysmographic recordings.

All analogous physiological signals were amplified and recorded by a Beckman R611 Dynograph recorder. In addition, they were stored on magnetic tape (Hewlett Packard, 3968A) for off-line analysis in case of breakdown of the computers that controlled the on-line data collection procedures. For an extensive description of the automatized data collection and preprocessing, the reader is referred to Geenen and Aalders [33].

IBIs were measured by feeding the analogous signal into a locally made timer circuitry that calculated the time between the successive R-waves. TWA was operationalized as the difference between the maximum amplitude of the ECG in the interval between 150 and 350 msec after the R-wave, minus the zero level, which was determined after 60 percent of the time of the preceding IBI had expired. The plethysmographic signal was amplified by a locally made device. Its output and the analogous ECG signal were led into a pulse maximum detector, which measured the time between the apex of the R-wave and the peripheral pulse. At each heart beat, the preceding IBI, PTT, and TWA were sampled and stored in a file.

Timing and marking was done by a Falcon computer. IBIs were used to calculate the means and the variability (VAR), which was defined as the sum of the absolute differences between successive IBIs, as described by Mulder [34].

Procedures

Potential participants were visited at their homes, at which time extensive information was given about the experimental procedures and informed consent obtained.

To control for diurnal variation of hormonal measures, which also were obtained in this study, the experimental sessions always started at 9:15 A.M. Before the start of each session, several procedures related to blood sampling for hormonal measurement occurred (see [35]). Subsequently, the electrodes and transducers for psychophysiological recording were applied.

After the subjects were connected, they were given written instructions informing them that they would be viewing 6 films depicting situations from daily life. In addition, it was emphasized that it was important to sit quietly while watching the films. Finally, it was stated that the subjects could stop their participation at any moment. When the subjects indicated that everything was clear, an adaptation period of 10 min started, followed by the initial baseline measurements. All subjects then saw the same 6 films in the same order (two buffer films followed by Driving Test, Death Bed, Surgery, and Rape). After each film, blood samples were taken and the psychological reactions were measured.

The session was concluded with a 1-min rest condition in which participants were instructed to relax. In between the films and during the concluding rest condition, music was played over speakers. A session lasted about 2½ h.

RESULTS

Association between independent variables and neuroticism

In order to have an impression of the relationship between the independent (self-report) measures and neuroticism, Pearson *r*'s between these variables were computed: Neuroticism-EPCL, $r = 0.60$; Neuroticism-REEQ, $r = 0.56$; and Neuroticism-HSCL, $r = 0.88$ (all p values were less than 0.01). Therefore, neuroticism scores were entered as covariates for all analyses.

Data were analyzed applying repeated measures analysis of covariance (ANCOVA), subjected to Greenhouse-Geisser corrections of the degrees of freedom. In addition to neuroticism, smoking and alcohol consumption were entered as covariates.[1]

Psychological reactions

The ACL data were used to obtain information concerning the psychological effects of the film stimuli. Following Vingerhoets [16], the raw ACL data were subjected to a Principal Components Analysis with Varimax rotation. This yielded three factors with eigenvalues above 2.0. The three factors could be labeled as follows: "Distress", "Deactivation", and "Openness/Involvement". For each of these factors, individual factor scores were calculated for every condition. Figure 1 shows the uncorrected group means for each ACL factor. Subsequently, $2 \times 2 \times 7$ repeated measures ANCOVAs were carried out with Psychosocial Load and Symptoms as between-subjects variables and Condition (the 6 post-film and the one final rest measurements) as the within-subjects variable.

No main effects for Psychosocial Load or Symptoms were found. However, the Symptom by Load interaction was significant for the Distress factor when neuroticism was covaried ($F(1, 84) = 4.10$, $p < 0.05$). The mean Distress levels for the 4 groups were LL/LS = −0.40, LL/HS = 0.12, HL/LS = −0.05, and HL/HS = 0.34, but none of the means proved significantly different from another using Duncan's test on the means.

All three of the ACL factors showed significant differences over the 7 conditions: Distress ($F(3.58, 522) = 31.39$, $p < 0.001$); Deactivation ($F(3.44, 522) = 24.97$, $p < 0.001$), and Openness/Involvement ($F(3.23, 522) = 4.58$, $p < 0.01$). Analyses of the means confirmed that the Rape film was more distressing than any other condition with the Surgery and Death Bed films being more distressing than the Resting and Buffer conditions (p values were less than 0.05). In contrast, the final resting condition

[1]To test if the groupings adequately reflected the four possible combinations of high and low load and symptoms, all data analyses were re-run using the load and symptoms scores as covariates. In no case was there any alteration of outcome. Therefore, the groupings were considered to be robust.

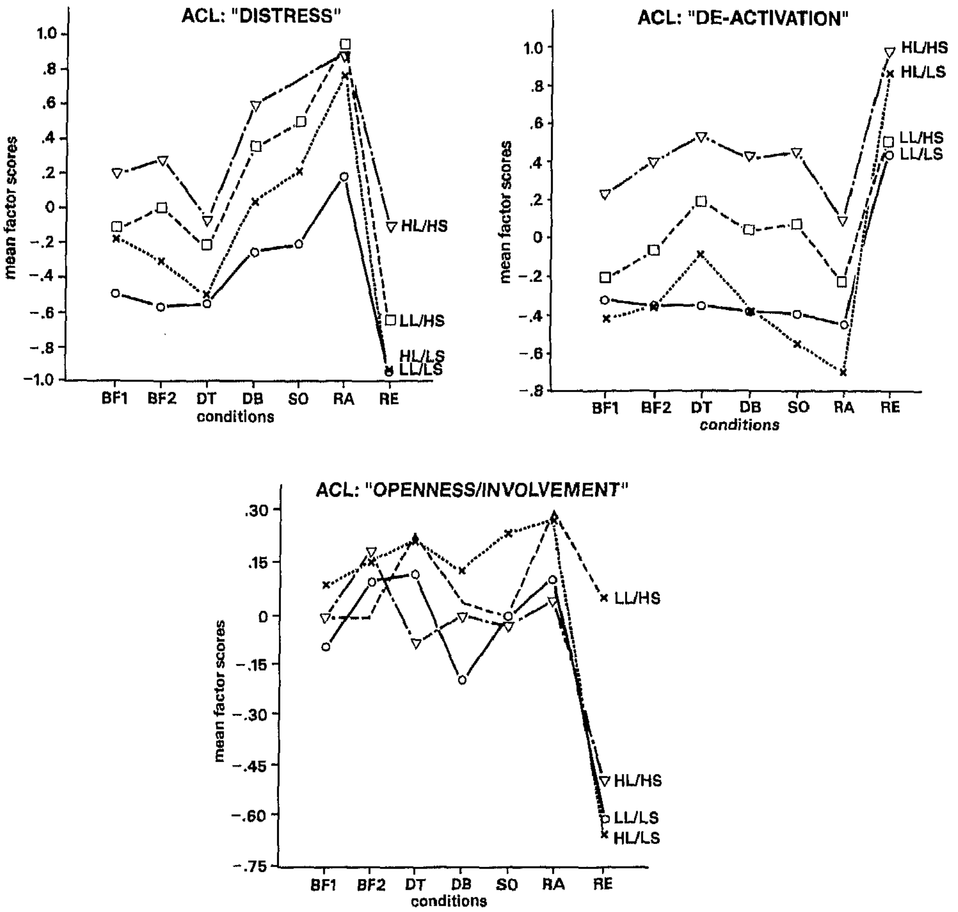

Fig. 1. Uncorrected mean ACL factor scores for the four subject groupings (LL/LS, LL/HS, HL/LS, and HL/HS), represented per condition.

was characterized as being the least distressing, most deactivating, and the one with the lowest level of involvement (p values were less than 0.05).

Mood responses among the groups over measurements were similar, except between the high-and low-symptom groups on Deactivation ($F(3.44, 552) = 3.31, p < 0.05$). Comparisons of the means revealed that the low-symptom subjects reported higher activation throughout the session, except during the final resting period when the groups showed almost identical, increased levels of deactivation (p values were less than 0.05).

In order to test the affective comparability of the films that were expected to result in either greater inhibitive (Surgery, Death Bed) or stimulating (Driving Test, Rape) responses, mean ACL responses were combined based on film type. This resulted in a new 2 × 2 × 4 ANCOVA with Neutral (Buffer) films, "CVS-Inhibition" (CVS−) films, "CVS-Stimulation" (CVS+) films, and Final Resting period representing the 4 within-subject conditions. Again, significant condition effects were found for the three ACL factors ($F(1.81, 261) = 21.77, p < 0.001$; $F(1.54, 261) = 37.69, p < 0.001$; and

$F(1.56, 261) = 6.46, p < 0.01$ for Distress, Deactivation, and Openness/Involvement, respectively). The Stimulation and Inhibition films were found to be more distressing than either Buffer films or Rest conditions, and Rest remained the most deactivating and least involving (p values were less than 0.05), but the CVS− and CVS+ films were similar on each of the mood dimensions.

Physiological functioning

Physiological activation levels. In order to assess differences in physiological activation levels at rest and during the films, repeated measures ANCOVAs were conducted on each psychophysiological measure obtained during the 21 measurement points (7 precondition measurements and 7 × 2 condition measurements) from initial baseline through the end of the final rest period. Repeated measures ANCOVAs conducted on IBI, VAR, TWA, and PTT showed significant changes over time ($F(6.86, 1700) = 31.51, p < 0.001$; $F(6.45, 1740) = 2.82, p < 0.01$; $F(1.87, 1740) = 20.49, p < 0.001$; and $F(4.05, 1720) = 11.69, p < 0.001$, respectively). These effects over time reflect changes in responsivity during the films, which are analyzed more thoroughly in the following section.

Significant effects of symptoms and/or load categorization were found for IBI and TWA, but not for VAR and PTT. Inter-beat intervals were significantly smaller for high-symptom complaints subjects, reflecting faster heart rates, than for low symptom subjects ($F(1,82) = 5.27, p < 0.05$). Similarly, the pattern of IBIs over time differed between high- and low-symptom subjects ($F(6.86, 583) = 1.93, p = 0.06$).

Post hoc analyses revealed that the HL/HS group exhibited faster IBIs than did the HL/LS group during the time period between the first rest period through the rest period that followed the final buffer film. Subsequently, during the rest and film periods that were associated with the stressful films, IBI rates did not differ among any of the groups. The IBI rates then diverged again during the final rest period, with the HL/HS group again exhibiting IBIs that were significantly faster than those shown by the HL/LS group (p values were less than 0.05). The other two groups (LL/LS and LL/HS) fell between these two groups in IBIs during the beginning and end rest periods.

Significant differences among the four groups also appeared for TWA over time ($F(1.87, 1740) = 3.27, p < 0.05$). Post hoc comparisons showed significant differences in TWA throughout the session between HL/LS and LL/HS groups, with HL/LS subjects exhibiting a larger mean TWA than LL/HS and the other two groups falling between these two. This would be suggestive of lesser sympathetic myocardial tone for the HL/LS subjects throughout the session compared with LL/HS subjects. However, it should be noted that, since TWA is not a calibrated measure, caution must be exercised when making between-group comparisons.

Physiological reactivity

Cardiovascular reactivity to the films was calculated as the difference between the average of the two measurement points during each film and the immediately preceding rest period. Repeated measures ANCOVAs conducted on each of the psychophysiological measures showed significant effects based on film type (IBI: $F(1.95, 172) = 39.63, p < 0.001$; TWA: $F(1.96, 174) = 10.61, p < 0.001$; PTT: $F(1.76, 172) = 36.30, p < 0.001$;

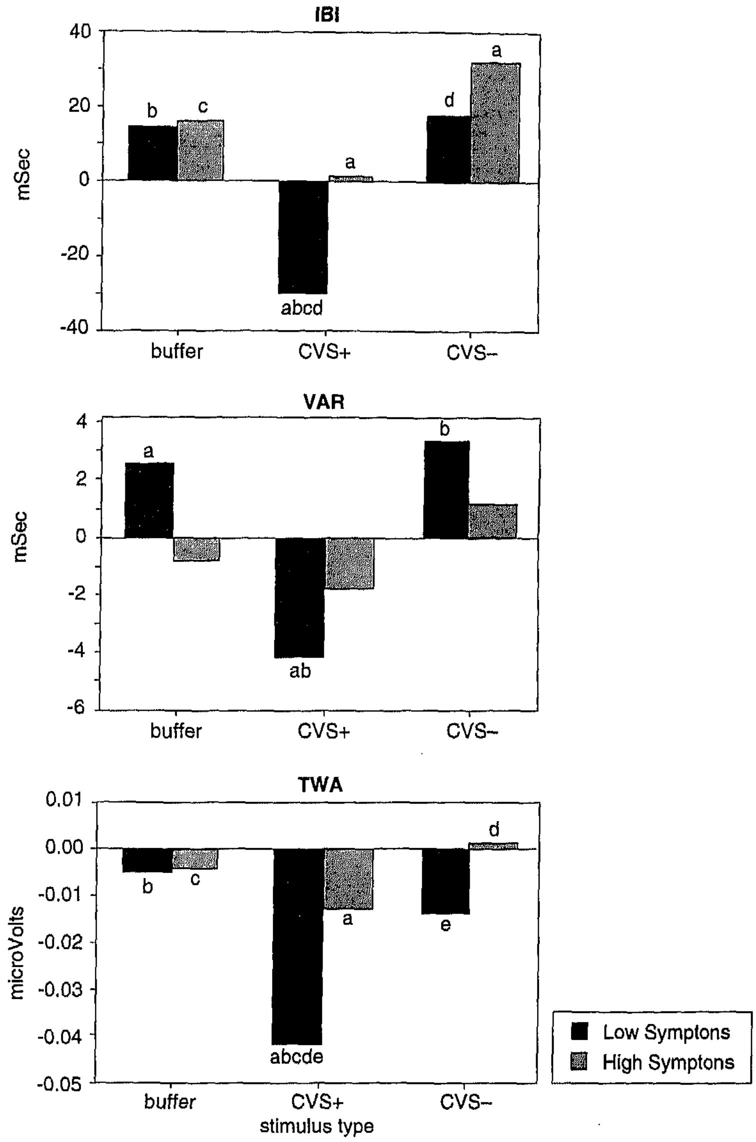

Fig. 2. Reactivity differences between high-symptom and low-symptom reporting groups. Like subscripts (a through d, $p < 0.01$; e, $p < 0.05$) indicate significant differences.

VAR: $F(1.96, 174) = 10.66, p < 0.001$). Post hoc analyses revealed significant decreases from prefilm rest in PTT for all 3 film types. Significant decreases in IBI, TWA, and VAR were found during the CVS+ films only, whereas significant increases in IBI occurred during the buffer and CVS– films (all p values were less than 0.01). The increases in VAR during the CVS– films approached, but did not reach, significance.

Significant differences were found for the symptoms by film type interactions for IBI, TWA, and VAR ($F(1.95, 172) = 5.97, p < 0.01$; $F(1.96, 174) = 3.20, p < 0.05$;

and $F(1.96, 174) = 3.41$, $p < 0.05$; respectively) (Fig. 2). Post hoc analyses indicated that low-symptom subjects showed greater decreases in IBI (i.e., increased heart rate) and TWA during the CVS+ films than did high symptom participants (p values less than 0.01). Additionally, while the low symptom participants showed significant differences in IBI reactivity to the CVS+ films in comparison with both the CVS− and the buffer films, high symptom participants showed differences only between the CVS+ and CVS− films (all p values were less than 0.01). Moreover, only for the low-symptom individuals was TWA found to decrease significantly during the CVS+ films, reflecting greater sympathetic activation of the myocard (p values were less than 0.01). High-symptom participants' TWAs showed no appreciable changes from rest for any film type.

Similarly, low symptom reporting was associated with greater variation in VAR depending on film type (p values were less than 0.01), whereas high symptom reporting was not associated with appreciable VAR differences between films.

The psychosocial load by film type interaction proved to be significant for VAR ($F(1.96, 174) = 7.38$, $p < 0.01$). Post hoc analyses revealed that only the marked decreases in VAR exhibited during the CVS+ films by high-load participants were significant ($p < 0.01$).

Post hoc analyses of the 4 separate groups for each of the physiological variables that had significant load by film type or symptom by film type interactions revealed that for both TWA and VAR, the HL/LS group showed the largest responses during the CVS+ films in the direction of greater sympathetic activation (p values were less than 0.05).

Unwinding

Of further interest was the question of whether there was evidence in favor of the slow unwinding hypothesis, which would be apparent if there were significant group by condition interactions in ANCOVAs on all baseline measurements (in-between films and final rest). However, for none of the variables was this interaction significant.

DISCUSSION

The aim of the present study was to systematically investigate psychological and psychophysiological—in particular, cardiovascular—reactions to emotional films as a function of self-reported stressor load and symptom complaints. Given the topical discussion of the influence of negative affectivity or neuroticism on such self-reports [15, 36], individual neuroticism scores were taken into account in data analysis. Although the groupings were made based on scores appearing above or below the medians found in the larger parent sample, symptom scores tended to be higher among high-load subjects and, likewise, load scores were higher among high symptom reporting subjects. Possible threats to the validity of the factorial design were tested by reanalyzing all data with the z-transformed load and symptom scores as covariates. These analyses revealed no significant contribution of these scores over that of the designated groupings. This, then, confirmed the adequacy of the symptom and load groupings used in the present study.

The ACL results suggest the following conclusions:

(1) When neuroticism is applied as a covariate, there are no substantial overall differences for any of the mood variables between the high and low symptoms groups;

(2) Neither load nor symptom level differentially affected mood responses; and
(3) The films evoked psychological reactions, which differed significantly in intensity. These reactions failed to systematically correlate with psychophysiological reaction patterns; both increases and decreases in cardiovascular arousal were associated with increased feelings of distress. This was as predicted for these films – (dis)stressful emotional responses would be reliably found either with cardiovascular stimulation (Driving Test, Rape) or with cardiovascular deactivation (Death Bed, Surgery).

Altogether, then, the interactive effects of the two primary aspects of stress (stressors and strains) could be explored in their effects on exposure to subsequent emotional challenges with different characteristics. While there is some controversy surrounding the validity of symptom measures as indicators of stressor effects, primarily because of the relatively strong relationship with neuroticism [15], symptom reporting may help clarify who is and who is not adequately coping with current stressors. Symptom levels, independent of neuroticism, were found to differentiate between response patterns of subjects with comparable stressor loads. The pattern of responses was parallel to what a toughness [14] model would predict,[2] with those who seem to cope with life stressors more adequately (HL/LS) generally showing lower resting sympathetic activation and greater responsivity when presented with a stressor, especially in comparison to their high-symptom counterparts. Larger TWAs, indicative of lower sympathetic tone, were consistently found with the HL/LS group and resting periods were accompanied by the lowest heart rate levels for this same group. In contrast, LL/HS subjects showed the smallest TWAs and HL/HS subjects the fastest heart rates at rest. Thus, in both cases were high symptom levels found to be associated with greater myocardial sympathetic tone.

Low-symptom subjects were also notable in their different, presumably more adequate, responsiveness to the films in comparison with high-symptom subjects. Not only were low-symptom subjects' heart rate responses larger than were high-symptom subjects' when presented with the CVS+ films, but VAR's increases during CVS− and its decreases during CVS+ films indicate more appropriate responses by low-symptom subjects for the type of stimuli. This appropriate variation in response occurred most markedly in the HL/LS group. These response patterns and distinctions were missing with high-symptom subjects, who reacted more uniformly, independently of film type.

Our findings lend support to Watson and Pennebaker's view [15] that self-reports of stressors, symptoms and disturbed mood are determined by neuroticism, that is, a more general tendency to complain. However, at the same time, our physiological

[2] An anonymous reviewer suggested the interesting alternative interpretation that the pattern of responses shown among HL/LS subjects could be a reflection of repressive coping [37]. Several studies [38–41] have shown repressive copers to report comparatively low levels of negative affect to various challenges while displaying elevated physiological responses. Rather than being physiologically toughened, some proportion of the HL/LS group may be more likely to underreport undesirable emotions. Unfortunately, no measures were used in the present study that could support or refute this alternative explanation. However, the high stressors' scores within this group seem at odds with a repressive coping explanation. Additionally, recent data [41] suggest that the response dissociation only occurs under conditions in which the person is under direct, evaluative, observation. In the present study, the challenges were of a non-embarrassing quality, with emotional responses recorded privately. Yet, cardiovascular responses differed and no differences were found among the groups on emotional responses. This would argue against repressive coping as an explanation for the results reported here.

data strongly contradict the views of Watson and Pennebaker [15] that high symptom reports merely reflect a tendency to complain rather than being a valid index of disturbed somatic functions. Even with the strong correlation between neuroticism and the symptom measure ($r = 0.88$), the results of the ANOVAs were hardly affected by introducing this measure as a covariate. This further suggests that perhaps symptom reporting represents a particular class of neurotic responding that is independently predictive of (physiologic) response. This issue needs further study.

The cardiovascular findings reported here and the differences in endocrine functioning between both groups [see 35] clearly indicate that symptom reporting, more so than self-reported exposure to real-life stressors, is associated with psychophysiological arousal and responsivity. More precisely, lower symptom levels were predictive of lower psychophysiological base rates and larger – according to Dienstbier [14], more appropriate – responsivity. This study therefore provides evidence that stress outcomes (as measured by symptom reporting) and how they compare with reported stressor load may at least serve as a marker for physiological base rates and responsivity consistent with the physiological toughness model [14], rather than supporting the more traditional views considering high reactivity as *necessarily* bad and potentially harmful.

Although stress-load was of minimal importance when symptomatology was not considered, the present data do not necessarily contradict previous findings of studies [3–7] showing effects of stressful experiences on psychobiological functioning. The positive correlations between self-reported stressors and symptoms were relatively high (the correlations between HSCL and the stressor measures EPCL and REEQ were 0.53 and 0.47, respectively; see [21]). Thus, the present samples of HL/LS and LL/HS subjects actually overrepresent the corresponding groups in the population at large. Our selection procedures thus may have reduced the probability of replicating previous findings of direct stress-load and sympathetic activation connections.

Our findings thus challenge the traditional view of high reactivity being harmful for health and emphasize the need for a critical reappraisal and shading of that hypothesis. Our view is further supported by the results of a study by Ahern and collaborators [42], which showed that in post-infarction patients, low reactivity to a challenge was associated with future cardiac arrest. Moreover, there is a remarkable correspondence between the reaction patterns of low-symptom subjects found here and those of highly physically fit subjects [43, 44] to laboratory stressors.

Since recent research [45] has shown that, regardless of objective health status, subjective well-being is an important predictor of mortality, investigations aimed at the associations between subjective measures and psychobiological functioning deserve further exploration.

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REFERENCES

1. KARASEK RA, THEORELL T, SCHWARTZ JE, SCHNALL PL, PIEPER CF, MICHELA JL. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Am J Public Health* 1988; **78**: 910-918.

2. MATTHEWS KA, WEISS SM, DETRE T, DEMBROSKI TM, FALKNER B, MANUCK SB, WILLIAMS RB (Eds). *Handbook of Stress, Reactivity, and Cardiovascular Disease.* New York: Wiley, 1986.
3. PARDINE P, NAPOLI A. Physiological reactivity and recent life stress experience. *J Consult Clin Psychol 1983;* **51:** 467-469.
4. COHEN LH, SIMONS RF, ROSE SC, McGOWAN J, ZELSON MF. Relationships among negative life events, physiological reactivity, and health symptomatology. *J Hum Stress* 1986; **12(winter):** 142-148.
5. JORGENSEN RS, HOUSTON BK. Reporting of life events, family history of hypertension, and cardiovascular activity at rest and during psychological stress. *Biol Psychol* 1989; **28:** 135-148.
6. SCHAUBROECK J, GANSTER DC. Chronic demands and responsivity to challenge. *J Appl Psychol* 1993; **8:** 73-85.
7. SIEGRIST J. Occupational stress and cardiovascular reactivity in blue collar workers. *Work Stress* 1990; **4:** 295-304.
8. FLEMING I, BAUM A, DAVIDSON L, RECTANUS E, McARDLE S. Chronic stress as a factor in psychophysiological reactivity to challenge. *Health Psychol* 1987; **6:** 221-238.
9. OPMEER C, VAN DE HOFSTEDE J, HYNDMAN B, KRAMER F, POKORNY M. Vaststelling van vroege gezondheidseffecten van stress en opsporing van risicogroepen met behulp van provocatietests. Unpublished Report, Netherlands Institute for Preventive Health Care, Leiden, 1989.
10. FRANKENHAEUSER M. Psychoneuroendocrine approaches to the study of stressful person-environment transactions. In *Selye's guide to stress research,* Vol. 1 (Edited by Selye H) pp. 46-70. New York: Van Nostrand Reinhold Company, 1980.
11. GANNON L, BANKS J, SHELDON D, LUCHETTA T. The mediating effects of psychophysiological reactivity and recovery on the relationship between environmental stress and illness. *J Psychosom Res* 1989; **33:** 167-175.
12. WIENTJES CJE, DE WOLF-HOPMAN MM, GAILLARD AWK. Ademhaling en stress (Deel III). Onderzoek naar de effecten van kortdurende werkbelasting. Unpublished report. Institute for Perception/ TNO, Soesterberg, 1990.
13. VINGERHOETS AJJM. The role of the parasympathetic division of the autonomic nervous system in stress and emotions. *Int J Psychosom* 1985; **32:** 28-32.
14. DIENSTBIER RA. Arousal and physiological toughness: implications for mental and physical health. *Psychol Rev* 1989; **96:** 84-100.
15. WATSON D, PENNEBAKER JW. Health complaints, stress and distress: exploring the central role of negative affectivity. *Psychol Rev* 1989; **96:** 234-254.
16. VINGERHOETS AJJM. *Psychosocial Stress: An Experimental Approach. Life Events, Coping, and Psychobiological Functioning.* Lisse: Swets and Zeitlinger, 1985.
17. GEENEN MJM. *Psychophysiological Consistency and Personality.* Tilburg: Tilburg University Press, 1991.
18. LEIDELMEIER KCM. *Emotions: An Experimental Approach.* Tilburg: Tilburg University Press, 1991.
19. FERNANDEZ MC, VILA J. Sympathetic-parasympathetic mediation of the cardiac response in humans. *Biol Psychol* 1989; **28:** 123-133.
20. FUREDY JJ, HESLEGRAVE RJ, SCHER H. T-wave amplitude revisited: some physiological and psychophysiological considerations. *Biol Psychol* 1992; **33:** 241-248.
21. VINGERHOETS AJJM, VAN HECK GL. Gender, coping, and psychosomatic symptoms. *Psychol Med* 1990; **20:** 125-135.
22. VINGERHOETS AJJM, JENINGA AJ, JABAAY L, RATLIFF-CRAIN J, MOLEMAN P, MENGES LJ. The study of chronic stress: a psycho-biological approach. In *Theoretical and Applied Aspects of Health Psychology.* (Edited by Schmidt LR, Schwenkmezger P, Weinman J, Maes S) pp. 161-167. London: Harwood, 1990.
23. VAN DE WILLIGE G, SCHREURS P, TELLEGEN B, ZWART F. Het meten van 'life events': de Vragenlijst Recent Meegemaakte Gebeurtenissen (VRMG). [The measurement of life events: the Recently Experienced Events Questionnaire (REEQ).] *Ned Ts Psychol* 1985; **40:** 1-19.
24. RAHE RH. Recent life changes, emotions, and behaviors in coronary heart disease. In *Handbook of Psychology and Health Vol. V. Stress.* (Edited by Baum A, Singer JE) pp. 229-254. Hillsdale, NJ: Erlbaum, 1987.
25. VINGERHOETS AJJM, VAN TILBURG MAL. *De Alledaagse Problemenlijst (APL).* Lisse: Swets and Zeitlinger, 1994.
26. VINGERHOETS AJJM, JENINGA AJ, MENGES LJ. Het meten van chronische en alledaagse stressoren: Eerste onderzoeks-ervaringen met de Alledaagse Problemenlijst (APL) II. *Gedrag Gezondh* 1989; **17:** 10-17.
27. VINGERHOETS AJJM, JENINGA AJ, MENGES LJ. Chronic pain and everyday problems. *Int J Psychosom* 1987; **34:** 9-11.
28. DEROGATIS LR, LIPMAN RS, RICKELS K, UHLENHUTH EH, COVI L. The Hopkins Symptom Checklist, a self-report symptom inventory. *Behav Sci* 1974; **19:** 1-15.

29. LUTEIJN F, HAMEL LF, BOUWMAN TK, KOK AR. *Hopkins Symptom Checklist (HSCL) Manual.* Lisse: Swets and Zeitlinger, 1985.
30. LUTEIJN F, STARREN J, VAN DIJK H. *Nederlandse Persoonlijkheidsvragenlijst. Handleiding (herziene uitgave).* Lisse: Swets and Zeitlinger, 1985.
31. HETTEMA PJ, VAN HECK GL, BRANDT C. The representation of situations through films. In *Personality and Environment. Assessment of Human Adaptation.* (Edited by Hettema PJ) pp. 113-128. Chichester: Wiley, 1989.
32. KJELLBERG A, BOHLIN G. Self reported arousal: further developments of a multifactorial inventory. *Scand J Psychol* 1974; **15:** 285-292.
33. GEENEN MJM, AALDERS AHJA. Computer-assisted recording of psychophysiological response patterns. In *Computers in Psychology: Applications in Education, Research and Psychodiagnostics* (Edited by Mulder LJM, Maarse FJ, Sjouw W, Akkerman A). Lisse: Swets & Zeitlinger, 1991.
34. MULDER G. The heart of mental effort. Unpublished thesis. State University of Groningen, 1980.
35. VINGERHOETS AJJM, RATLIFF-CRAIN J, JABAAIJ L, *et al.* Self-reported stressors, symptom complaints, and psychobiological functioning II: Psychoneuroendocrine variables. *J Psychosom Res* (this volume).
36. WATSON D. On the dispositional nature of stress measures: stable and nonspecific influences on self-reported hassles. *Psychol Inquiry* 1990; **1:** 34-37.
37. WEINBERGER D, SCHWARTZ G, DAVIDSON R. Low-anxious, high-anxious, and repressive coping styles: psychometric patterns and behavioral and physiological responses to stress. *J Abnorm Psychol* 1979; **88:** 369-380.
38. ASENDORPF J, SCHERER K. The discrepant repressor: differentiation between low anxiety, high anxiety, and repression of anxiety by autonomic-facial-verbal patterns of behavior. *JPSP* **45:** 1334-1346.
39. GUDJONSSON G. Self-reported emotional disturbance and its relation to electrodermal reactivity, defensiveness and trait anxiety. *Pers Indiv Diff* 1981; **2:** 47-52.
40. JAMNER L, SCHWARTZ G. Self-deception predicts self-report and endurance of pain. *Psychosom Med* 1986; **48:** 211-223.
41. NEWTON T, CONTRADA R. Repressive coping and verbal-autonomic response dissociation: the influence of social context. *JPSP* 1992; **62:** 159-167.
42. AHERN DK, GORKIN L, ANDERSON JL *et al.* Biobehavioral variables and mortality or cardiac arrest in the Cardiac Arrhythmia Pilot Study (CAPS). *Am J Cardiol* 1990; **66:** 59-62.
43. LAKE BW, SUAREZ EC, SCHNEIDERMAN N *et al.* The type A behavior pattern, physical fitness, and psychophysiological reactivity. *Health Psychol* 1985; **4:** 169-187.
44. DE GEUS EJ, VAN DOORNEN LJP, ORLEBEKE JF. Regular exercise and aerobic fitness in relation to psychological make-up and physiological stress reactivity. *Psychosom Med* 1993; **55:** 347-363.
45. MOSSEY JM, SHAPIRO E. Self-rated health: a predictor of mortality among the elderly. *Am J Public Health* 1982; **72:** 800-808.