Constraints for emotion specificity in fear and anger: The context counts

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Abstract

We investigated psychophysiological responses to fear and anger inductions during real-life and imagination. Female participants (N = 158) were assigned to a fear-treatment, fear-control, anger-treatment, or anger-control group. Context (real-life, imagination) was varied in two sessions of fixed order. Eleven self-report and 29 somatovisceral variables were registered. Results showed that (a) except during anger imagination, control groups were emotionless; (b) in control groups, contexts prompted diverging somatovisceral responses, but similar emotion self-reports; except during fear imagination, the emotion inductions (c) were successful and (d) produced specific emotion reports; (e) during real-life, somatovisceral fear and anger responses exhibited a marked cardiovascular defense reflex; (f) in addition, real-life fear showed an adrenaline-like specific response pattern, whereas real-life anger showed specific forehead temperature and EMG extensor increases, accompanied by an elevated DBP during imagination. A Component Model of Somatovisceral Response Organization is proposed.

Descriptors: Emotion, Specificity, Defense reflex, Fear, Anger, Context

Somatovisceral activation constitutes the interface between brain and behavior. The study of somatovisceral responses is therefore an important field of psychological inquiry because, beyond mere reflexlike and homeostatic reactions, such responses may inform about the psychobiological processes leading to anticipated or actual behavior. However, diverse influences may simultaneously impinge on the somatomotor and the autonomic nervous systems, threatening the internal validity of studies by allowing alternative ways of interpreting empirical data.

Research about the physiological specificity of emotions is a pertinent example. For instance, the finding of physiological differences between fear and anger inductions (Ax, 1953) could have been misrepresented as evidence for emotion specificity simply because the inductions differed both physically and in their behavioral demands (Stemmler, 1984). Similarly, Stemmler’s (1989) report that emotional autonomic patterning was specific to the context of emotion induction was criticized by Ekman (1994) for measuring physiology “a considerable period” after the induction was over, suggesting the emotional processes had already subsided.1 Boiten (1996) argued that the directed facial action task used by Ekman, Levenson, and collaborators (Ekman, Levenson, & Friesen, 1983; Levenson, Carstensen, Friesen, & Ekman, 1991; Levenson, Ekman, & Friesen, 1990; Levenson, Ekman, Heider, & Friesen, 1992) exerted its autonomic effects primarily through effort-related changes in respiration rather than through the activation of emotion circuits in the brain. Levenson and Ekman (submitted) in turn argue that Boiten has misrepresented their results. In sum, many concerns prevail about the internal validity of studies in this field, and opinions about the empirical validity of physiological emotion specificity are strongly diverging.

Conceptual objections against the view that somatovisceral responses could be specific for emotions (James, 1884), or at least for some (Ekman, 1992), came in two waves. During the first wave of objections it was argued that autonomic activity is largely undifferentiated and unitary, eliminating any basis for specific bodily emotion signatures (Cannon, 1929; Duffy, 1962; Mandler, 1975; Schachter & Singer, 1962). The second wave of objections is more recent: Instead of reflecting the arousal of a particular emotion, somatovisceral processes would indicate the demands of an action disposition, action tendency, or action proper (Davidson, 1993, 1994; Frijda, 1986; Lang, Bradley, & Cuthbert, 1990). Moreover, emotions could motivate actions that, across emotions, were likely to overlap.

The first objection against somatovisceral emotion specificity, that is, that autonomic activity is largely undifferentiated, is no longer tenable. There is ample evidence from both physiology (Hilton, 1975; Hilton & Redfern, 1987; Jänig, 1988; Jänig & McLachlan, 1992) and psychophysiology (Berntson, Cacioppo, & Quigley, 1991; Fahrenberg, 1987; Lacey, 1967; Stemmler, 1992a; Stemmler & Fahrenberg, 1989) that somatovisceral processes are
highly patterned and are likely to reflect the activity of multiple activation/inhibition systems. The second objection, that somato-visceral activation during emotions reflects action tendencies but not emotions per se, is a serious challenge to the notion of somato-visceral emotion specificity. This argument was motivated by the low consistency of somatovisceral configurations in studies of emotion (see reviews by Cacioppo, Klein, Berntson, & Hatfield, 1993; Stemmler, 1998). In addition, there is ample evidence that not only physical tasks, body postures, and their associated motor behaviors, but also psychological influences (e.g., attention, mental effort) exert an effect on somatovisceral activation. In the following, the term “context” will be used to denote this ensemble of non-emotional physical, behavioral, and psychological factors that influence somatovisceral activation during emotion.

Beyond the context, emotions might still influence somatovisceral activation. But their effects could well be confounded with context effects, rendering their separate identification difficult. Because contexts vary widely across emotion studies, a low degree of consistency of somatovisceral emotion configurations would be predicted. Theoretically, one of us has discussed context-deviation specificity as a model of emotion specificity (Stemmler, 1984, 1989, 1992b) which—rather than the model of absolute specificity—should be applied when there are context–emotion confounds. Context-deviation specificity views emotion specificity as a conditional concept. An emotional “stimulus” is supposed to modify a context-bound physiological pattern. Thus, somatovisceral emotion and context effects are usually confounded. Emotion specificity could then be demonstrated only after this confound has been pulled apart. Emotion specificity is found whenever there are systematic and specific deviations of the emotion-plus-context pattern of physiological reactivity from the context-alone pattern. Absolute emotion specificity assumes that, even across different contexts, at least some emotions are accompanied by specific physiological patterns. However, neither context–emotion confounds nor the validity of context-deviation specificity have yet been studied systematically. The present investigation is a first attempt to fill this gap. In particular, we compared a real-life with an imagination context with respect to emotion specificity.

In this report, we chose to study the negatively valenced emotions of fear and anger. Reasons for this choice were threefold. First, the database for fear-anger contrasts is larger than for any other emotion combination. Second and perhaps more importantly, fear and anger have been induced in different contexts, including “real-life” and imagination settings, and the directed facial action task. Apart from fear and anger, only happiness has been induced during real-life inductions (see the studies reviewed by Cacioppo et al., 1993). Finally, somatovisceral differences between fear and anger, should they exist, could not be attributed to their common negative valence.

There are at least 15 studies that have compared fear and anger in two or more somatovisceral responses (Adsett, Schottstaedt, & Wolf, 1962; Ax, 1953; Boiten, 1996; Chessick, Bassan, & Shattan, 1966; Funkenstein, King, & Drolette, 1954; Levenson et al., 1990, 1991, 1992; Miller et al., 1987; Roberts & Weerts, 1982; Schachter, 1957; Schwartz, Weinberger, & Singer, 1981; Sinha, Lallowlo, & Parsons, 1992; Sinha & Parsons, 1996; Stemmler, 1989). Findings across these studies demonstrated few consistent somatovisceral differences between fear and anger. During anger, diastolic blood pressure, total peripheral resistance, and somatomotor activity increases were larger than during fear (in five out of eight, three out of four, and two out of six studies, respectively). During fear, finger temperature decreases and cardiac output increases were larger than during anger (in three out of seven and in two out of four studies, respectively). The present investigation sought to replicate these consistent findings but also to extend the present knowledge in using a much broader sample of somatovisceral variables than did most of the studies cited above. A broader sample of variables might allow conclusions about the physiological regulation patterns underlying emotion responses that could reach beyond what can be inferred from a few variables alone.

All of the studies cited above used a within-subjects control condition against which the emotion effects were compared. This procedure is perfectly well-suited for determining whether there are somatovisceral emotion differences, but the control–emotion comparison is confounded by context differences if they exist. Thus it will be difficult to determine what the context-free emotion signatures might look like. To illustrate, context effects were likely to operate in diastolic blood pressure, even though the small number of studies defies any definitive conclusions. All real-life ($N = 5$) and all imagination ($N = 3$) studies found significant diastolic blood pressure increases during anger compared to the control condition. In contrast, during fear, all real-life ($N = 4$) but none of the imagination ($N = 3$) studies found significant diastolic blood pressure increases. As a consequence, only two real-life studies reported significantly larger diastolic blood pressure increases during anger than fear, but all of the three imagination studies did. Thus, depending on the emotion induction context there is a real risk for the test of emotion specificity to fail for artificial reasons.

To determine what the context-free emotion signatures might look like, a control condition as similar as possible to the emotion induction period needed to be constructed—except for the emotion effect per se (Boiten, 1996; Nyklicek, Thayer, & van Doornen, 1997; Stemmler, 1992b). In this study, we tried to approach this goal through a between-subjects control design where the control group received exactly the same treatment as the treatment group with the one difference that controls were completely preinformed about the impending emotion inductions. This control procedure should alleviate the emotional meaning of the impending emotion induction but leave the nonemotional context effects almost unchanged. Our expectation of a negligible emotional engagement of the control group is based on the powerful effects appraisals can have under conditions where individuals have to make sense of their environment. Referring, for example, to the cognitive-motivational-relational theory of emotions, Lazarus (1991) distinguishes among three forms of primary appraisal: goal relevance, goal congruence, and type of ego involvement. If an individual is convinced that the impending threat to his or her self- or social esteem is just fake, the person will not very much care about the forthcoming encounter (goal relevance), the threat will not be strongly inconsistent with his or her goals (goal congruence), and the individual’s self- or social esteem will not be markedly endangered or assaulted, as in fear and anger, respectively.

In sum, the aims of the present study were fourfold. First, we tried to determine context effects of two alternative induction methods: real-life and imagination. If present, such effects could blur the effects of emotion on somatovisceral responses. In contrast to somatovisceral responses, we expected only minor influences of the context on self-reports of emotion, because feeling states reflect meaning structures that are largely independent of situational properties. Second, we asked what the emotion responses to fear and anger emotion inductions look like, if—in light of our claim of context–emotion confounds—emotion and context effects are pulled apart by contrasting appropriate treatment and control groups. This second aim also touches upon the question of whether these emo-
tion responses are consistent across real-life and imagination contexts. Third, using a broad sample of somatovisceral variables, we tried to identify the regulatory patterns underlying fear and anger responses. Finally, we wanted to replicate the small and fragile set of findings suggesting a physiological differentiation of fear and anger.

Method

Participants
One hundred and ninety-seven participants were recruited by flyers distributed on a university campus and by local newspaper ads mentioning a study of stress and strain. Inclusion criteria were female gender, German native language, age between 18 and 45 years, right-handedness, and normal weight according to the body-mass index (23 kg/m² ± 5 kg/m²). Exclusion criteria were studying psychology, current medical treatment, taking medication affecting the circulation, or pregnancy. We chose a female sample because gender differences in self-reported emotions point to a larger female expressiveness of negative emotions (Manstead, 1992). Thirty-nine participants declined after the introductory session or were excluded, leaving N = 158 participants for statistical data analysis.2 The average age of these participants was 25.3 years (range 18–45 years); mean body-mass index was 21.1 kg/m² (range 16–31 kg/m²). Seventy-seven percent of the participants were university or high school students, the remaining 23% were employees from various professions. Participants were paid 90 DM for approximately 6 hr involvement in the study.

Experimental Design
Participants were assigned to one of four experimental groups formed by the combination of Emotion (fear, anger) × Group (treatment, control). Participants were randomly assigned to groups with the restriction of homogeneity of trait anxiety in fear groups and of trait anger in anger groups. As a result, fear groups were virtually identical in STAI trait anxiety (M = 40.0, 40.9; SD = 8.0, 9.4; N = 38, 41, for treatment and control groups, respectively) and anger groups in STAXI trait anger (M = 22.2, 21.9; SD = 4.9, 5.4; N = 40, 39, same order as above). Context (real-life, imagination) was a within-subjects factor. Real-life inductions were always performed first. Imagination inductions were performed one week later in a second experimental session.

A notable feature of this experiment was the use of multiple induction periods, that is, several inductions of the same emotion right after one another. This procedure allowed us to aggregate measurements across emotion induction periods provided homogeneity of response profiles was given. Aggregation increases measurement reliability.

The choice of a between-subjects design for the factors Emotion and Group is quite unusual in psychophysiological emotion research, because a within-subjects design has the virtue of controlling sources of variance between individuals that may be large in somatovisceral variables. However, the internal validity of within-subjects designs is easily threatened (Cook & Campbell, 1979). Relevant problems are the interaction of adaptation effects (fatigue, habituation, motivation, etc.) with presentation order and changing interpretations or expectations on the part of the participants. To be sure, presentation order of fear and anger inductions could in principle be balanced across participants, but obviously not the order of treatment and control conditions (at least not with our control group procedure). Of equal importance is the consideration that, from the perspective of the experimenter, real-life inductions always carry an element of surprise and dissembled intentions. Thus, it is not unlikely that, in a within-subjects design, the second emotion induction has less of an impact than the first one. For these reasons we chose a between-subjects design.

Setting and Apparatus
The experimental room (4 × 3.4 m) was sound-attenuated and air-conditioned and had a largely nontechnical appearance. Participants sat comfortably in a reclined position. Electrodes were connected to a customized headbox (NeuroScan), where signals were preamplified with a gain of 30 (input impedance of differential inputs 20 MΩ). Transducer-based signals were relayed through an input box to Biopac couplers. Right in front of the participant’s seat was a customized ergometer (Kettler EX-1), which could be pedaled while seated. Three LEDs signaled the participant to slow down, speed up, or continue at the same pace. A custom-made hand dynamometer with a built-in indicator scale for pulling strength was mounted at the right side of the seat. Other equipment included two WS-A10EW Panasonic 160 W loudspeakers, a JVC TK-1281 video camera, and a 20-in. black-white computer monitor (Miro).

In an adjacent room were placed a 32-channel SynAmps Model 5083 amplifier with 16 bit A/D conversion (NeuroScan); a 16-channel Biopac system with couplers for skin conductance (GSR 100), skin temperature (SKT 100), pulse volume (PPG 100), respiration belt (RSP 100), general purpose couplers (DA 100), and with an MP 100 workstation with 16 bit A/D conversion; a Kardio-Dynagraph (Diefenbach) for impedance cardiography; a bosotron 2 (Bosch) blood pressure monitor; a Sony minidisk player MDS 501 for the delivery of instructions and loud noise; and other audiovisual equipment. A Macintosh Quadra 950 (Apple) microcomputer with a NB-D10-24 digital I/O card (National Instruments) and four serial I/O ports performed experimental control, data recording, data visualization, and data storage under LabView 3.1.1 (National Instruments).

With the exception of parts of the fear induction and the imagination session, participants were alone in the room. Whenever necessary, experimenter and participant communicated via intercom.

Procedure
Introductory session. Participants could familiarize themselves with the experimental room, some experimental tasks (standard tests), and the emotion self-report form. They also filled out trait anger and anxiety questionnaires needed for group assignments. Finally, participants signed an informed consent that also mentioned that it might be necessary to obtain a blood sample.

Experimental Session I. One to three weeks later, the first experimental session was scheduled. It took about 2.5 hr. Two female assistants positioned electrodes and transducers. The (male) experimenter explained the standard tests and the emotion self-report form. He reminded the participant to sit quietly to help prevent artifacts in the physiological recordings.

The standard tests began with a 10-min rest period during which participants should relax but keep their eyes open. The loud noise

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2Reasons to decline were the disturbing loud noise (N = 8) and the experience of claustrophobic feelings (N = 1). N = 15 participants gave no explanation for their declining. Reasons to exclude participants were unwillingness to openly answer the items of a personality questionnaire (N = 2), the association of the Milgram experiments (N = 2), and strong artifacts in the physiological recordings (N = 11).
task consisted of a 185-s 100 dBA loud white noise delivered over the loudspeakers. During the handgrip task, participants had to press the hand dynamometer for 2 min at 50% of their maximal voluntary force. The exercise task consisted of a graded ergometer exercise beginning at 25 W with a 20-W increase every minute. During the fifth and final minute, a load of 105 W was reached. Pedaling speed was 50–60 rpm. The tasks were introduced by 1-min prestimulus periods followed by an emotion self-report.

Figure 1. Sequence and duration of experimental conditions. Panel a visualizes the standard tests; panel b, real-life fear induction; panel c, real-life anger induction; and panel d imagination. Black horizontal bars denote periods of physiological data recording. An “X” identifies data recordings and emotion self-reports that were used as covariates in the ANCOVAs. A “Y” denotes data analyzed as dependent variables.

statistical comparison of responses among experimental groups after the emotion inductions.

The real-life fear induction³ comprised a 4-min rest period, a 1-min prestimulus period, and four induction periods, all of which were immediately followed by a 1-min data recording period and then by an emotion self-report. The experimenter explained that the aim of the current session was to study the physiological effects

³Details of the induction procedures are described in Stemmler, Heldmann, Pauls, and Scherer (1998).
of preparing and giving a speech. Participants were asked to sit quietly. Induction Period 1 began with the instruction that participants were to give a 5-min speech on the topic “arguments pro and con the European Union.” The speech should be prepared during the next 5 min. Participants should do their best. It would be necessary to record their speech. The speech would be evaluated for verbal intelligence and compared to a norm sample. Actually, the speech preparation lasted 11 min, because it was interspersed with three additional fear induction periods. Induction Period 2 comprised a mental arithmetic task. Participants had to subtract 1, 2, 3, and so forth from 1,000 and the ongoing intermediate results silently and as quickly as possible. After 1 min, participants were stopped to tell the current result. The experimenter commented on the poor performance and gave a new start number. One minute later, the subject was stopped again. Again, the result was claimed wrong. After data collection, the experimenter kept grumbling at the subject. During Induction Period 3, an anagram task was presented on the monitor. After 6 of 12 words, the experimenter in an angry voice quarreled with the participant for moving around in her seat. At the end of the anagram task, the experimenter rudely accused her of noncompliance. After data collection, participants were unhooked, led to another room for a postexperimental interview, and then debriefed (see Figure 1, Panel c, for the exact timing of the real-life anger induction).

Real-life anger inductions for control groups started with a detailed explanation of the actual aims of the study. The participants were told that they had been assigned to a control group. They would be asked to give a free speech that would be evaluated. But actually, they only needed to read aloud a written text. Her reading performance would not be evaluated. The experimenter put down a page with the text and introduced the assistants who would later bring in the syringes or supposedly evaluate the speech. Controls were assured that no attempt would be made to draw blood. Finally, all instructions she would hear would come from tape. To demonstrate, the experimenter ran one instruction track. During the experiment, control subjects heard the same tape as treatment subjects.

The real-life anger induction comprised a 4-min rest period, a 1-min prestimulus period, and three induction periods, all of which were immediately followed by a 1-min data recording period and then by an emotion self-report. During Induction Period 1, participants were given a series of 15 items selected for high error rates from a test of general knowledge. They had to say loudly “I don’t know” if they did not know an answer. After the second item, which was practically unsolvable, participants were asked to speak louder to compensate for an alleged malfunction of the intercom. After the eighth item, the experimenter interrupted again and said in an annoyed tone that he could not understand her. After the task, participants were told that they had only one-third correct. After the registration period, the experimenter claimed seeing movement artifacts and reminded the participant to sit quietly. Induction Period 2 comprised a mental arithmetic task. Participants had to subtract 1, 2, 3, and so forth from 1,000 and the ongoing intermediate results silently and as quickly as possible. After 1 min, participants were stopped to tell the current result. The experimenter commented on the poor performance and gave a new start number. One minute later, the subject was stopped again. Again, the result was claimed wrong. After data collection, the experimenter kept grumbling at the subject. During Induction Period 3, an anagram task was presented on the monitor. After 6 of 12 words, the experimenter in an angry voice quarreled with the participant for moving around in her seat. At the end of the anagram task, the experimenter rudely accused her of noncompliance. After data collection, participants were unhooked, led to another room for a postexperimental interview, and then debriefed (see Figure 1, Panel c, for the exact timing of the real-life anger induction).

Real-life fear inductions for control groups started with the same general explanations as in the real-life fear control groups. The participant was forewarned that the comments of the experimenter during the following tasks had the goal of inducing anger in the participants. The comments were bogus, and did not really apply to her performance. Nevertheless she should try her best to solve the tasks. All instructions including the experimenter’s insults would be replayed from tape. An example track with an abusive comment was played. Then the real-life anger induction as described above followed.
Experimental Session II. About one week later, participants returned to the lab. Standard tests were identical to the first session, but emotion inductions now used emotional imagery. Participants performed two distinct, consecutive imagination tasks: Reliving a personal emotional episode and recalling the real-life emotion induction. Only the latter recall will be used here, because it (a) leaves imagination content largely constant across participants, (b) automatically includes treatment versus control group information, and (c) best equates the stimulus and background information across real-life and imagination contexts.

Fear and anger imagination followed the same scheme. After a 4-min rest period with ensuing emotion self-report, the reliving and then the recall task began, each introduced by a 1-min pre-stimulus period. Participants recalled the induction periods one by one (four periods for fear, three periods for anger groups). The male assistant supported the recollections encouraging especially the recall of bodily sensations during that period (focus on response propositions; Miller et al., 1987). He then asked the participant to imagine that situation as vividly as possible and to leave her eyes open. With a button press he started a 1-min data recording period. Then he continued with the next induction period. Following the last recall, participants completed the emotion self-report for the most intensively recalled period and then scored the vividness of her imagery. Finally, participants were unhooked and led to another room for a postexperimental interview (see Figure 1, Panel d, for the exact timing of the imagination task).

Variables
Self-report of emotion. Participants performed an 11-point intensity rating on six unipolar (0 = “not applicable,” 10 = “completely applicable”) and five bipolar (5—0—5) scales tagged by one to four descriptive adjectives. The scales were selected to capture (a) expected emotional feelings such as shame (embarrassed/ridiculed/ashamed/foolish), fear (frightened/timid/afraid/scared), sadness (sad/depressed/miserable/dejected), happiness (happy/gay/cheerful/delighted), and anger (angry/annoyed/mad/sore); (b) the bodily sensation of a pounding heart; (c) feelings of arousal and valence such as tense versus relaxed (nervous/restless/tense/wound up versus calm/relaxed/serene/ease), active versus tired (energetic/activeanimated/lively versus tired/fatigued/sluggish/exhausted), positive versus negative (positive/pleasant versus negative/unpleasant); (d) cognitive states such as alert versus confused (alert/attentive/receptive/abrupt versus confused/baffled/perplexed); and (e) motivational states such as interested versus bored (curious/interested/interested) or indifferent/indifferent/dull).

Self-reports of emotion were always collected right after physiological registration periods. Because we could not be sure that the emotions induced would last longer than 1 min, participants were not asked to rate their momentary affective state. Instead, participants were asked to recall and rate the emotional state that prevailed right before the registration period, that is, at the end of the preceding condition. Ratings were obtained during standard tests for the end of the rest period, for the loud noise and the handgrip tasks, and for the final minute of the exercise task; during real-life emotion inductions, for the end of the rest period, and separately for each of the three (anger) or four (fear) induction periods; during the imagination of emotion, for the end of the rest period and for the most intense of the recall periods.

Imagery vividness. Vividness of imagination was assessed on a 7-point scale ranging from “perfectly clear and as vivid as the actual experience” (1) to “not clear or vivid, but recognizable” (4) to “no image present at all, you only ‘knowing’ that you are thinking of the situation” (7).

Physiological variables. Twenty-nine physiological parameters were derived from the somatovisceral recordings.¹ Electrode sites were cleansed with alcohol and rubbed with OmniPrep paste to ensure electrode impedances below 10 kΩ. If not otherwise noted, BIOPAC Sigma Gel 100 electrode paste was used. Participants were grounded at the left mastoid.

The electrocardiogram (ECG) was recorded through Ag/AgCl surface electrodes (8 mm sensor diameter, In Vivo Metric) from a point below the right clavicle and the left lateral margin of the chest. Filters were set to DC and 125 Hz (12 dB/octave), amplification was 200, and sampling rate was 250 Hz. Parameterization was performed with the program BIO25 (Fahrenberg & Foerster, 1989) yielding heart period (in milliseconds), P-wave and T-wave amplitudes (in millivolts), Pe-Qs time (end of the P-wave to start of the Q-wave; in milliseconds), relative Q-T time (systolic time: start of the Q-wave to end of the T-wave, relativized by Bazett’s frequency correction to a heart rate of 60 bpm; in square root of milliseconds), and ST-segment (determined as the amplitude of the ECG-curve at a point 80 ms past the J-point, which was defined as the first point with a null potential after the S-peak; in millivolts). Heart period variability (HP-variability; in milliseconds squared) was calculated as the mean square of successive heart period differences.

The impedance cardiogram (ICG) was recorded with four band electrodes, two placed around the neck and two around the thorax. A current of 0.5 mA and 33 kHz was fed through the outer electrodes. The dZ/dt signal was amplified by 30 with a filter setting of DC and 125 Hz and a sampling rate of 250 Hz. Single epoch parameterization was performed with the program BIO25. Left ventricular ejection time (LVET; in milliseconds) was determined as the time between B-point (notch at the E-wave upstroke) and X-point; pre-ejection period (PEP; in milliseconds) was the time between the Q-wave in the ECG and the B-point. Stroke volume (SV; in milliliters) was estimated with the Kubicek equation. Other variables included cardiac output (CO; in liters per minute), ventricular ejection speed (in ohms per second), the Heath index (in ohms per second squared), and R-Z time (time between R-wave in the ECG and E-wave maximum in the ICG; in milliseconds).

Systolic and diastolic blood pressure (SBP and DBP; in millimeters of mercury) were measured intermittently by a noninvasive automatic inflation system which allowed for one measurement per minute. The cuff (50 cm x 13 cm) with an in-built piezoelectrical microphone was applied on the left arm. Total peripheral resistance (TPR; in dyn x s x cm⁻⁵) was estimated from mean blood pressure and CO.

Peripheral pulses were detected at the volar surface of the distal phalanx of the left middle finger (TSD 100, BIOPAC). To avoid both external light from interfering with the photoplethysmograph and cooling of the hand (with concomitant vasoconstriction), the left hand was placed in a mitten. The signal was amplified by 50 with filters set to DC and 10 Hz at a sampling rate of 250 Hz. The BIO25 program extracted from this signal pulse volume amplitude (in arbitrary units). Pulse transit time (PTT; in ms) was determined as the time between the R-wave in the ECG and the systolic peak in the pulse signal.

¹EEG variables and voice parameters were also obtained, but they are not analyzed here.
Skin temperatures (in degrees Celcius) were measured at the volar surface of the left little finger’s distal phalanx and at the forehead with a TSD 102a fast response thermistor (BIOPAC). Sensitivity was $1.39^\circ C/V$ (with a midpoint at 32.2$^\circ C$); the frequency range was DC—0.15 Hz; sampling rate was 1 Hz.

Respiratory excursions were recorded with a thoracic strain gauge transducer (TSD101, BIOPAC). Amplification was 50, filters were set to DC and 10 Hz; sampling rate was 100 Hz. The BIO25 program determined respiration rate (in min$^{-1}$) as the frequency with peak amplitude in the power spectrum of 10-s data windows (with Hanning window).

Electrodermal activity was recorded with a constant voltage of 0.5 V at the volar surfaces of the proximal phalanges of the index and ring fingers on the left hand. Ag/AgCl electrodes (TSD 103, BIOPAC) had a surface of 0.38 cm$^2$; they were filled with a 0.05 molar sodium chloride Unibase emulsion. Sensitivity was 100 $\mu S/V$ with a frequency range of DC—10 Hz and a sampling rate of 100 Hz. Phasic responses greater than 0.078 $\mu S$ (minimal slope of 0.007 $\mu S$/s, maximal half recovery time of 10 s) counted as skin conductance responses (SCR). BIO25 parameterization yielded SCR-amplitude (in microsiemens) and the number of SCR per minute. Skin conductance level (SCL; in microsiemens per square centimeter) was separated from the raw signal with a 0.15-Hz low-pass filter and sampled with 1 Hz.

Electromyograms (EMG; in microvolts) were obtained from the m. extensor digitorum, m. zygomaticus major, and m. corrugator supercilii of the left side by through Ag/AgCl surface electrodes (arm 8 mm, face 4 mm sensor diameter, In Vivo Metric). Amplification was 1,000 with filters set to 10 Hz and 1 kHz After rectification, the signals were low-pass filtered at 40 Hz (24 dB/octave) and sampled at 100 Hz.

The vertical electrooculogram was derived from 4 mm Ag/AgCl electrodes (In Vivo Metric) placed on the vertical midline of the left eye right above the eyebrow and about 0.8 cm below the lower lid. Amplification was 200; frequency range was DC — 30 Hz, and sampling rate was 100 Hz. The BIO25 program determined the number of eyeblinks per minute (Eyeblink-No.).

Periods of physiological data recording were always 1 min long. During standard tests, nine periods were obtained: 1st, 5th, and 9th minute of the rest period as well as prestimulus and task periods of the loud noise and the handgrip tasks (exercise phases were not used because of their relatively high artifact rate). The three covariates for the analyses of emotion self-reports were taken from the rest period, the loud noise, and the handgrip task. With this analysis, we intended to control the extent physiological between-subjects variance (Marwitz & Stemmler, 1998) both at rest and in response to physical challenges differing in the kind of physiological demand (for an overview including the white noise and the handgrip tasks, see Buell, Alpert, & McCrory, 1986; Stemmler, 1992a). Emotion self-reports were also analyzed with ANCOVA, because random group differences in these scales might have existed already before the emotion inductions were introduced. In such cases, ANCOVA can be superior to an analysis of difference scores.

ANCOVA permits conditional statements about treatment effects and controls for random pretreatment group differences in response level and reactivity. ANCOVA may reduce the error variance in group comparisons and consequently increase statistical power. In this study, ANCOVA is a valid statistical procedure to

### Data Analysis

**Preprocessing of physiological data.** For each physiological variable, a 158 subjects $\times$ 32 periods (anger) and a 158 subjects $\times$ 34 periods (fear) raw data matrix was constructed. Each cell within this matrix collected the raw data of 1-min periods. Because the basic time window for biosignal analysis differed across variables, the matrix cells contained either 60 data points (1-s time windows for EMG, SCL, skin temperature), number of beats-per-min data points (beat-by-beat time window for cardiovascular variables), 6 data points (10-s time windows for SCR, eyeblink, respiration rate), or 1 data point (blood pressure). First, in a computerized routine, these raw data were inspected for artifacts and deleted as necessary. Second, if within one cell more than 80% of the 1-s, 10-s, or beat-by-beat raw data were missing, the cell was set to missing data. Otherwise, medians (all of the ECG- and ICG-derived parameters, pulse volume amplitude, SCL, and skin temperature) or means (SCR, respiration rate, EMG, and Eyeblink-No.) were calculated per cell. Third, multivariate outlier detection on single blood pressure scores or on mean and median period scores was performed using JMP statistical software (SAS Institute Inc., 1995). Outliers were set to missing data. Overall, about 5% of the data were classified as missing. Finally, missing data were replaced with estimates computed from the overall statistical model, which specified Emotion, Group, Subject, Context, and Period. Period was nested within Groups and Contexts.

**Response scaling.** For the statistical analysis of group differences during emotion inductions, we used the analysis of covariance (ANCOVA) with multiple covariates. The five covariates for physiological data analyses were taken from the standard tests: the 5th minute of the rest period and the prestimulus and task periods of the loud noise and the handgrip tasks (exercise phases were not used because of their relatively high artifact rate). The three covariates for the analyses of emotion self-reports were taken from the rest period, the loud noise, and the handgrip task. With this analysis, we intended to control the extent physiological between-subjects variance (Marwitz & Stemmler, 1998) both at rest and in response to physical challenges differing in the kind of physiological demand (for an overview including the white noise and the handgrip tasks, see Buell, Alpert, & McCrory, 1986; Stemmler, 1992a). Emotion self-reports were also analyzed with ANCOVA, because random group differences in these scales might have existed already before the emotion inductions were introduced. In such cases, ANCOVA can be superior to an analysis of difference scores.

ANCOVA permits conditional statements about treatment effects and controls for random pretreatment group differences in response level and reactivity. ANCOVA may reduce the error variance in group comparisons and consequently increase statistical power. In this study, ANCOVA is a valid statistical procedure to

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5 Error sums of squares in Emotion $\times$ Group ANOVAs performed on difference scores during real-life emotion inductions were indeed markedly reduced by ANCOVAs with one to five covariates to 72%, 61%, 56%, 52%, and 48%. Numbers are averages across 29 physiological variables.

6 Specificity analyses were also performed using simple difference scores between response levels during emotion inductions and the 5th minute of the introductory rest period. Of the 29 t-tests reported in the column “Specific response 1” in Table 1, 23 had the same sign and significance status (6 negative sign and significant, 15 nonsignificant, 2 positive sign and significant). ANCOVA showed five significant variables and difference score analysis one significant variable that was nonsignificant in the other analysis. Of the 29 t-tests reported in the column “Specific response 1” in
test the null hypothesis of no conditional mean differences among groups (Huitema, 1980), because (a) participants had been randomly assigned to experimental groups and (b) the covariates (standard test periods) were assessed before any differential treatment had begun.

Statistical data analysis. Statistical data analysis was performed in three steps. First, homogeneity of multiple emotion induction periods of real-life fear and anger inductions was checked to determine which periods could be averaged for subsequent analyses. Second, ANCOVAs on averaged induction periods were performed for each variable. Third, these univariate tests were supplemented by multivariate tests across all variables to guard against an inflation of the alpha error in multiple univariate tests.

Homogeneity of response profiles during real-life emotion induction periods was determined by a test of profile parallelism (i.e., the interaction of variables × induction periods, see Morrison, 1976). This test was performed on response levels of variables standardized across all observations registered. Dependent variables in subsequent statistical analyses were the means across homogeneous induction periods. Forming means also allowed calculation of internal consistencies (Cronbach’s alpha) of the dependent variables. To guarantee maximum comparability, means of physiological data during the induction periods of the imagination context were obtained across the same periods as during the real-life context. Self-reports of emotion during imagination could not be averaged because only one rating was performed.

The overall statistical model used for hypothesis testing was a 2 (Group) × 2 (Emotion) × 2 (Context) ANCOVA followed up by a priori specified contrasts (see below). Using PROC MIXED of SAS/STAT (SAS Institute Inc., 1997), the error variance-covariance matrix was specified to be completely general (TYPE=UN option) and to allow for heterogeneous error variances within each Group × Emotion combination (GROUP option). These provisions tailored the statistical tests of contrasts to their appropriate error terms.

Ten contrasts were specified. They were devised to test for (a) emotion task effects (differences between the tasks per se, i.e., without an emotional tuning; comparison of fear and anger induction control groups, separately within the real-life and the imagination context), (b) context effects (comparison of control groups across contexts, separately for the fear and the anger inductions), (c) emotion responses (the effects emotions had over and above task effects per se; comparison of treatment and control groups, separately within real-life fear, real-life anger, and imagination anger conditions), and (d) specific emotion responses (the tetrad difference of the responses to the fear minus the anger emotion inductions in the treatment minus control group means, separately within each context).

An analysis of response profiles during emotion inductions supplemented the univariate analyses and also served to control for the accumulation of the alpha error present in multiple univariate, non a priori tests (see below). Two parameters of response profiles were analyzed, profile level and profile pattern. Profile level is the mean across profile variables. Levels were analyzed just like single variables; they depict the general response amplitude. Profile patterns denote the particular configuration of response variables. Differences between patterns were analyzed with multivariate analysis (Morrison, 1976), followed up by the contrasts specified above. To prepare for profile analysis, adjusted responses were derived from the ANCOVAs described above, standardized across all 158 (subjects) × 2 (sessions) observations ($M = 0, SD = 1$), and scaled to a common response direction. The latter objective was achieved by changing the polarity of the self-report variables happiness, active, positive, alert, and interested, and of the somatovisceral variables heart period, HP-variability, T-wave amplitude, ST-segment, Pe-Qs time, LVET, PEP, R-Z time, SV, TPR, pulse volume amplitude, PTT, and skin temperature at the finger. The test statistic reported in multivariate analyses is the $F$-approximation of Wilks’ lambda.

Unless otherwise noted, tests used an $\alpha$ of .05, two-tailed. For the following variables, one-tailed a priori tests were performed: We expected increases of self-reported fear during fear and of self-reported anger during anger inductions. For somatovisceral variables, we expected finger skin temperature decreases and CO increases during fear, and DBP, TPR, and EMG extensor increases during anger inductions. The substantive hypotheses behind the convergent validity contrasts postulated the equality of fear and of anger population means across contexts. In distinction to the common null hypothesis of equal population means, which is guarded by the alpha error against false rejection, the null hypothesis here is that population means are not equal. A small alpha would yield large confidence intervals around the sample means and tests of convergent validity would become extremely liberal. Therefore, the beta error needed to be controlled. Results of a power analysis indicated that for $\beta = 0.05$ the alpha error was to be set at a two-tailed $\alpha = .39$.

Results

Preliminary Analyses

Sequence effects of contexts. Contexts could not be counterbalanced, because the imagination task in Session II demanded reimagining the real-life emotion induction in Session I. To check whether the lack of counterbalancing might have had an impact on the results described later on, emotion self-reports and somatovisceral levels during the rest period of the standard tests were analyzed for session differences. Emotion self-reports exhibited session differences during rest in shame, fear, happiness, pounding heart, positive valuation, alertness, and interest, with higher ratings during Session I overall. This result suggests that at the beginning of Session I, affect intensity, but not negative affect in particular, was elevated. Levels of some somatovisceral variables were also elevated (DBP, SBP, SCL, EMG extensor) or reduced (PTT, pulse volume amplitude, skin temperature at the finger) indicating some degree of excess activation during the initial rest period (5th minute) of Session I. The consequence of these session effects will be smaller differences between standard tests and emotion induction periods during real-life inductions and larger differences during imagination. Thus, the size of emotion responses will tend to be underestimated in real-life compared to imagination inductions. This underestimation of real-life emotion responses is however

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Table 1. 27 had the same sign and significance status (1 positive and 1 negative sign and significant, 25 nonsignificant). In each analysis, one $t$ test was significant but nonsignificant in the other analysis. Overall, ANCOVA displayed slightly higher statistical power but a highly similar picture in the pattern of results as seen with difference score analysis.

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7 An a priori power analysis using the parameters of the respective Session × Group $F$ test ($df_1 = 1$, $df_2 = 154$, $N = 79$; estimated between session correlation $\rho = 0.50$ and under the assumption of a small effect size ($f^2 = 0.02$) indicated that for a beta error of 0.05, the alpha error equals 0.39.
more than compensated by the large effects of the real-life induction per se. Most importantly, it does not introduce a confound for the specificity analyses, which were conducted separately within each session.

Homogeneity and internal consistency of emotion induction periods. The four real-life fear induction periods exhibited significant heterogeneity in their somatovisceral profiles, \( F(84,841) = 1.48, p < .01 \). Exclusion of Induction Period 2 (threat of physical harm; chronotropic and inotropic activation were markedly lower here than in the other induction periods) made the remaining three profiles homogeneous, \( F(56,408) = 1.33 \). Self-report of emotion profiles in these three periods were also not heterogeneous, \( F(20,450) = 0.90 \). Thus, fear Induction Periods 1, 3, and 4 were aggregated. Internal consistencies of somatovisceral variables ranged from 0.79 to 0.99 with a median of 0.96; internal consistencies of emotion self-report ranged from 0.81 to 0.94 with a median of 0.88.

The three real-life anger induction periods were not heterogeneous in their somatovisceral profiles, \( F(56,414) = 0.74 \); neither were the profiles of emotion self-reports, \( F(20,450) = 1.30 \). Thus, anger Induction Periods 1, 2, and 3 were aggregated. Internal consistencies of somatovisceral variables ranged from 0.73 to 0.99 with a median of 0.92; internal consistencies of emotion self-report ranged from 0.84 to 0.93 with a median of 0.90.

Analysis of the Emotion Control Groups

Differences between emotion tasks. Within the real-life context, induction tasks per se (i.e., fear-control and anger-control responses) elicited identical emotion self-reports, as seen in equal profile levels and parallel self-report profiles. Somatovisceral profile levels were not different, but profile patterns were, \( F(28,126) = 1.69, p < .05 \). During the anger task, EMG zygomaticus activity was larger and respiration slower than during the fear task, \( t(73) = 3.00 \) and 3.04, respectively, \( p < .01 \). Within the imagination context, anger profile levels of emotion self-reports were larger than fear levels, \( t(75) = 2.35, p < .05 \). Self-report profiles were parallel. Anger-control compared to fear-control recalls produced larger self-reported anger and shame but less activity and alertness, \( t(75) = 2.06, 2.24, 2.72, \) and 2.33, respectively, \( p < .05 \). Neither somatovisceral profile levels nor patterns were different. Thus, with the exception of emotion self-reports during imagination, emotion task effects within contexts were negligible.

Differences between contexts. Fear-control during real-life did not differ from fear-control during imagination in self-report profile levels; however, the real-life context evoked higher sadness and lower tenseness and heartbeat sensations. In contrast, somatovisceral differences between contexts were remarkable. Compared to imagination, profile levels were higher, \( t(35) = 4.63, p < .01 \), and cardiac chronotropic and inotropic activations larger during real-life, as were eyewblinks-No. and SCR-amplitude. However, imagination produced larger activation in SBP, SCL, and skin temperature at the forehead (see “Context effect fear,” Table 1). Anger-control during real-life was different from anger-control during imagination in lower self-report profile levels, \( t(35) = 2.83, p < .01 \). The real-life context produced higher (though low) happiness and positive valuation and lower anger, heartbeat sensations, and tenseness. Somatovisceral profile levels, however, were higher during real-life than imagination, \( t(33) = 6.39, p < .01 \). Left-ventricular contractility, somatomotor activity, and electrodermal activity were larger during real-life (see “Context effect anger,” Table 1).

Analysis of the Emotion Treatment Groups

Responses to Emotion Inductions

Real-life context. In response to both the fear and the anger induction, self-report profile levels, \( t(74) = 9.00, 7.88, p < .01 \), and somatovisceral profile levels, \( t(72) = 10.81, 7.99, p < .01 \), were elevated in treatment compared to control groups. In addition, self-report profiles, \( F(10,145) = 6.61, 5.92, p < .01 \), and somatovisceral profiles of treatment and control groups, \( F(28,126) = 5.36, 3.45, p < .01 \), were not parallel. Compared to control groups, both fear and anger inductions led to feelings of strong negative valence in treatment groups, accentuated by the respective target emotions fear and anger. During fear, most of the somatovisceral variables showed changes in the direction of chronotropic, inotropic, pressor, vascular, and electrodermal activation. Anger led to a similar yet slightly less pronounced increase in somatovisceral activation (see “Emotion response fear r1” and “Emotion response anger r1”, Table 1).

Imagery vividness. The overall mean of the 7-point vividness rating was \( M = 3.43 \). Experimental groups did not differ in their ratings, \( F(3,153) = 0.60 \). In the postexperimental interview, participants were asked how well they could put themselves back into the situation. Ratings on a 4-point scale from 1 = “not at all” to 4 = “very well” indicated that participants could follow the task quite well (overall \( M = 2.67 \)). Again there were no group differences, \( F(3,154) = 0.75 \).

The imagination context. In this context, self-reports of emotion and somatovisceral arousal diverged markedly. In response to both the fear and the anger induction, self-report profile levels, \( t(74) = 5.03, 3.98, p < .01 \), were elevated in treatment compared to control groups; self-report profile patterns were different between these groups only during the anger induction, \( F(10,145) = 2.90, p < .01 \). The self-reported emotional changes paralleled the general negative and the target emotion specific pattern seen during the real-life inductions. In contrast, somatovisceral activity was much more dampened during imagination than during real-life, and it was elevated only in the anger induction, where profile levels, \( t(72) = 3.26, p < .01 \), and patterns, \( F(28,126) = 2.21, p < .01 \), differed between treatment and control groups. Anger recall led to increases in chronotropic activation, blood pressure, electrodermal, and forehead skin temperature (see “Emotion response anger i”, Table 1). Fear recall evoked no somatovisceral profile level or pattern changes.

Specific Responses to Emotion Inductions

Real-life context. Profiles of fear and anger emotion self-reports had different patterns, \( F(10,145) = 5.72, p < .01 \), but essentially equal levels. “Specific responses” (see Table 1 for \( t \) values) were found for self-reports of fear (effect size \( d = 1.19 \)) and anger \((d = -1.63)\), and only in these target emotions. Figure 2 clearly demonstrates specificity in these self-reports, as in the treatment but not the control group the induction’s target emotion was significantly elevated. In addition, as depicted in the contrast variable “sr” in the right panel of each plot in Figure 2, this elevation was much smaller in the nontarget condition. Somatovisceral fear and anger response profiles had specific profile levels, \( t(149) = 8 \) Profile pattern contrasts for within-subjects effects are not reported, because the statistical analysis software (PROC GLM of SAS/STAT, SAS Institute Inc., 1997) did not allow calculation of them.
Table 1. t-Values of Contrasts

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Shame</td>
<td>1.59</td>
<td>−1.66</td>
<td>4.54*</td>
<td>5.04*</td>
<td>4.68*</td>
<td>3.60*</td>
<td>−1.10</td>
<td>0.28</td>
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<tr>
<td>Fear</td>
<td>0.56</td>
<td>−1.04</td>
<td>6.47†</td>
<td>2.92*</td>
<td>4.10†</td>
<td>3.00*</td>
<td>3.72†</td>
<td>1.66†</td>
</tr>
<tr>
<td>Sadness</td>
<td>2.07*</td>
<td>−0.91</td>
<td>2.51*</td>
<td>3.74*</td>
<td>2.71*</td>
<td>2.57*</td>
<td>−0.96</td>
<td>−0.71</td>
</tr>
<tr>
<td>Happiness</td>
<td>−0.23</td>
<td>2.58*</td>
<td>−3.36*</td>
<td>−3.09*</td>
<td>−1.62</td>
<td>−0.79</td>
<td>0.36</td>
<td>−0.70</td>
</tr>
<tr>
<td>Anger</td>
<td>0.50</td>
<td>−2.03*</td>
<td>3.23*</td>
<td>8.01†</td>
<td>2.83*</td>
<td>5.14†</td>
<td>−5.11†</td>
<td>−2.90†</td>
</tr>
<tr>
<td>Heartbeat</td>
<td>−2.80*</td>
<td>−2.91*</td>
<td>7.89*</td>
<td>6.09*</td>
<td>2.89*</td>
<td>3.91*</td>
<td>1.60</td>
<td>−1.08</td>
</tr>
<tr>
<td>Tense</td>
<td>−2.46*</td>
<td>−2.72*</td>
<td>10.00*</td>
<td>8.71*</td>
<td>3.04*</td>
<td>4.71*</td>
<td>1.23</td>
<td>−0.80</td>
</tr>
<tr>
<td>Active</td>
<td>−0.70</td>
<td>0.63</td>
<td>−2.32*</td>
<td>−0.88</td>
<td>−2.07*</td>
<td>0.68</td>
<td>−0.91</td>
<td>−1.92</td>
</tr>
<tr>
<td>Positive</td>
<td>0.80</td>
<td>2.37*</td>
<td>−6.66*</td>
<td>−7.61*</td>
<td>−3.86*</td>
<td>−3.08*</td>
<td>0.51</td>
<td>−0.24</td>
</tr>
<tr>
<td>Alert</td>
<td>−1.11</td>
<td>1.30</td>
<td>−4.69*</td>
<td>−3.20*</td>
<td>−2.00*</td>
<td>−0.38</td>
<td>−0.64</td>
<td>−0.93</td>
</tr>
<tr>
<td>Interest</td>
<td>0.12</td>
<td>0.23</td>
<td>−3.92*</td>
<td>−4.62*</td>
<td>−1.38</td>
<td>−0.76</td>
<td>0.85</td>
<td>−0.36</td>
</tr>
</tbody>
</table>

**Self-report variables**

Heart period: −2.16*, −1.04, −8.41*, −5.95*, −1.09, −2.62*, −3.11*, 1.21

P-wave amplitude: 1.95, 1.94, 3.77*, 1.60, 1.64, 2.28*, 1.24, −0.12

T-wave amplitude: −0.96, −2.25*, −7.54*, −3.41*, 0.76, −1.42, −4.35*, 1.54

ST-segment: 1.74, −1.64, −6.72*, −5.41*, 0.14, −1.58, −3.00*, 1.16

PQ-time: 0.84, 1.44, −6.25*, −6.13*, 0.22, 0.15, −1.08, 0.03

Relative Q-T time: 1.81, 1.91, 3.66*, 1.79, 0.25, 2.23*, 1.89, −1.62

LVET: −1.27, −1.02, −5.05*, −4.22*, 1.18, −2.00*, −0.43, 2.32*, 0.93

PEP: −1.06, −1.06, −8.20*, −4.30*, −1.70, 1.18, −2.70*, −2.07*

Ejection speed: 2.85*, 3.28*, 2.94*, 1.37, −0.48, −1.33, 1.46, 0.61

R-Z time: −0.72, −1.14, −8.06*, −6.59*, −0.44, −0.71, −2.71*, 0.17

Heather index: 2.22*, 2.76*, 5.73*, 4.36*, −0.19, −0.76, 2.53*, 0.37

SV: 0.97, 1.38, −2.48*, −2.41*, 0.41, −1.60, 0.18, 1.55

CO: 2.27*, 1.96, 4.86, 2.33*, 1.05, −0.45, 3.04*, 1.05

SBP: −2.20*, −0.38, 7.07*, 6.48*, 1.97, 2.95*, 1.55, −0.77

DBP: −1.03, −0.06, 2.08*, 3.35*, 0.45, 3.22†, −1.03, −2.09†

TPR: −3.17*, −2.05*, −4.58*, −0.54, −0.03, 1.64, −2.93†, −1.18

PVA: −2.02, 0.29, 5.00*, −3.92*, −1.89, 1.39, −1.47, 0.14

PTT: −2.08*, 0.47, −9.05*, −6.96*, −1.64, −1.00, −2.17*, −0.05

SCR-No: 1.95, 0.74, 8.34*, 5.25*, 0.81, 3.05*, 3.78*, −1.65

SCR-amplitude: 2.88*, 2.18*, 3.20*, 0.96, 1.40, 0.54, 1.53, 0.77

SCL: −2.19*, −0.91, 4.17*, 4.24*, 0.70, 1.85, 0.51, −0.97

TMP-forehead: −4.74*, −1.23, −0.48, 2.43*, 0.63, 2.93*, −2.08*, −1.84

TMP-finger: 1.13, 1.18, −0.41, −1.61, 1.85, 0.10, 0.92, 1.22

Respiration rate: 1.07, −1.39, −0.42, 0.23, 0.16, −0.40, 1.64, 0.39

Sensory feedback: 2.26*, 2.32*, 0.87, 0.18, 0.24, −0.20, 0.51, 0.31

EMG corrugator: 0.94, 1.07, 1.78, −2.10*, 0.20, 0.51, 2.74*, −0.22

EMG zygomaticus: −0.52, 2.85*, 2.80*, 0.63, 1.19, −0.35, 1.41, 1.06

EMG extensor: −1.48, −1.39, −0.15, 2.05*, 0.05, 1.53, −1.71†, −1.21

**Somatovisceral variables**

Notes. rl = real-life context. im = imagination context. LVET = left-ventricular ejection time. PEP = pre-ejection period. SV = stroke volume. CO = cardiac output. SBP = systolic blood pressure. DBP = diastolic blood pressure. TPR = total peripheral resistance. PVA = pulse volume amplitude. PTT = pulse transit time. SCR = skin conductance response. SCL = skin conductance level. TMP = skin temperature. EMG = electromyogram.

df = 37 (self-report variables)/35 (somatovisceral variables).  df = 37/156. df = 74/72. df = 151/149.

α < .05 (two-tailed). α < .05 (one-tailed).

3.98, p < .01 (fear > anger), and specific profile patterns, F(28,26) = 2.16, p < .01. Thirteen of the 29 somatovisceral variables were differentially affected by fear and anger inductions (see “Specific response rl”, Table 1). During fear, we found shorter heart period, PEP, R-Z time, and PTT; reduced T-wave amplitude and ST-segment; larger Heather index, CO, SCR-No. and EMG corrugator activity. Means of specific variables are shown in Figure 3. Note that control group means in many variables were very similar, which attests to the efficiency of the experimental control group procedure and the adjustment of group means by analysis of covariance. In contrast, treatment group means varied widely across treatment groups. Of the two expected fear-specific responses, one (CO, d = 0.97) but not the other (skin temperature at the finger) was obtained. During anger, we found larger TPR, skin temperature at the forehead, and EMG extensor activity, two of which were expected (TPR, d = −0.93; EMG extensor, d = −0.54). However, the third expected specific response, DBP, was not obtained.

**Imagination context.** Again, profiles of fear and anger emotion self-report responses had different patterns, F(10,145) = 2.31, p < .05, but essentially equal levels. Fear and anger inductions differed in the feelings of fear (d = 0.53) and anger (d = −0.95), and only in these target emotions. Neither somatovisceral profile levels nor patterns were specific. Thus, only the a priori expected, stronger
increase of DBP ($d = -0.67$) during anger than during fear can be reported (see “Specific response im”, Table 1; see also Figures 1 and 2).

Discussion

As noted in the introduction, many students of emotion have concluded that emotion specificity does not exist at the somatovisceral level. As an alternative to this nonspecificity view, we have offered the model of “context-deviation specificity.” We have argued that comparing emotion inductions with control periods or comparing emotion inductions across studies could easily capture context plus emotion effects instead of emotion effects alone. In this way, both false-positive and false-negative “emotion” specificity results could have been created.

To disentangle context–emotion confounds and to increase the power of the experiment in general, the present study took several methodological steps. First, control conditions were almost identical to treatment conditions. However, control groups were pre-informed about the nature of the forthcoming induction. Second, the use of ANCOVA ruled out random, preexisting differences among groups in the response variables entering into the crucial emotion response and specific response contrasts. Third, fear and anger were each induced in two different contexts, real-life and imagination. Fourth, within each emotion induction, we presented three or four episodes. Episodes with homogeneous responses were aggregated to obtain more reliable response means. Fifth, sample size was large enough to ensure high statistical power. Finally, profile analysis was used to detect profile level and pattern differences between context and emotion and to guard against the inflation of the alpha error given multiple dependent variables.

Were Control Groups Emotionless?

During the real-life condition, fear-control and anger-control groups reported hardly any feelings at all. This result supports our experimental intention that control group responses should reflect task effects but not emotion effects. One could argue that because control groups knew they were controls for emotion induction treatment groups, their lack of reported feelings was instilled by perceived experimental demands. However, their somatovisceral responses were also subdued, and existing differences in EMG zygomaticus and respiration rate did not point to any known differences between fear and anger; thus, these differences were probably task but not emotion related.

Somewhat different, however, were the results regarding fear and anger task effects during imagination. Here, the anger control group reported more anger-related feelings than the fear control group. Recalling the anger provocation, the anger control group obviously did not very effectively activate also the instruction prior to the anger induction, which during the real-life session had successfully inhibited the arousal of anger. Similar effects within the fear control group were not noted. Below, a more extended explanation of these differential effects will be attempted.

Did Contexts Have Diverging Response Profiles?

The two real-life and imagination contexts elicited markedly different somatovisceral profile levels, as demonstrated in the control group responses in Figure 3. The higher physiological activation during real-life was indeed attributable to the context common to the fear and anger tasks, as both the real-life and the imagination fear and anger control groups demonstrated almost identical activation levels and profiles (with the exception of minor real-life task differences). This outcome was welcome because it allowed the study of physiological emotion effects on the background of context-specific somatovisceral activations. In particular, it justified our control group procedure to estimate the magnitude of context effects.

Emotion self-reports exhibited a different pattern of results. In the control group, the fear inductions during real-life and imagination elicited very similar reported feelings. Thus, somatovisceral activation with large, and reported feelings with small differences between contexts were clearly dissociated. This dissociation was even more pronounced for the anger inductions, where higher real-life somatovisceral activation contrasted with higher, anger-related reported feelings during imagination. Going beyond a mere description of dissociations between response systems (Lang, 1968; see also Miller & Kozak, 1993), these data suggest that somatovisceral activation was context bound, whereas emotion self-reports captured meaning structures, which were less constrained by the context. As already noted above, the anger control group during imagination was not as emotionless as during real-life, attesting to the power of anger episodes to be loaded with personal meaning when recalled.
Did the Emotion Inductions Work?

Emotion self-reports suggest successful emotion inductions, as seen both in the negatively valenced emotion responses (i.e., treatment–control group differences) and in the specific increases in reported fear and anger during fear and anger inductions, respectively (see a summary of results in Table 2, panels 1 and 2). This conclusion applies to both the real-life and the imagination context. Somato-visceral responses, however, draw a different picture. Whereas the
real-life fear and anger inductions elicited strong and patterned physiological responses, during imagination, only the anger induction did so, although somewhat dampened (Table 2, panels 3 and 4). Fear imagination evoked practically no physiological emotion response. Does this result warrant the conclusion that the fear recall failed to induce a fear response? If, as we propose, the answer is yes, why were self-reports of fear elevated and specific during fear recall?

One explanation would posit the operation of defensive interpretations of the real-life induction, when, one week later, the participants were asked to recall the real-life episodes. Because participants were debriefed at the end of Session I, fear treatment subjects might not have been able to imagine their real-life fear induction without interfering, fear-reducing thoughts that the threat was not real after all. This explanation could account for the lack of somatovisceral arousal; it would, however, be at variance with...
the elevated self-reports of negative emotions, particularly of fear, which should be the first target of a defensive orientation. A second explanation is more consistent with the data: (a) The intensity of fear during imagination was so low as to be near somatovisceral response thresholds and (b) the self-reports of fear did not reflect a genuine emotion but instead merely the knowledge of a recent fearful experience, or the demand to imagine it. Thus, the fear induction during imagination probably failed.

In contrast, the anger induction during imagination was successful. Anger treatment subjects might have felt the provocation even more strongly knowing, by then, that they had been mistreated intentionally. Anger control subjects, as noted above, felt some anger too, because the contents of the guided recall through the previous anger episodes might have acted like an imagination induction with standard scripts provoking anger to a degree beyond their own experience at the time.

**Specificity in Emotion Self-Reports**
The inductions elicited a broad array of negative feelings, and positive feelings including alertness and interest in the experiment declined. In terms of a dimensional model of mood, both fear and anger elicited displeasure (item “positive-negative”; Russell, Weiss, & Mendelsohn, 1989) and tense arousal (item “tense-relaxed”; Thayer, 1996), whereas energetic arousal (item “active-tired”; Thayer, 1996) was reduced in fear only. However, responses to the target emotions clearly stood out of a general, nonspecific activation of negative feelings. In addition, self-reports of fear and of anger were the only items that distinguished between fear and anger inductions both in the real-life and the imagination context. These data suggest that a dimensional model of emotion reports alone is incomplete. It does not capture the specific quality of the emotional experience in which we were most interested.

**Why Did Somatovisceral Fear and Anger Responses During Real-Life Overlap?**
In spite of significant somatovisceral profile pattern differences between fear and anger during real-life, all of the somatovisceral variables were pushed by the fear and anger inductions into the same direction. Eleven variables showed changes in only one emotion induction; 16 variables responded to both of the emotion inductions with changes into the same direction. The common denominator of fear and anger real-life emotion responses was a

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**Table 2. Summary of Emotion Responses and Specific Responses for Self-Report and Somatovisceral Variables within Contexts**

<table>
<thead>
<tr>
<th>Emotion response fear</th>
<th>Emotion response anger</th>
<th>Specific response</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Shame, Fear, Sadness, Anger, Heartbeat, Tense, Profile level</td>
<td>↑ Shame, Fear, Sadness, Anger, Heartbeat, Tense, Profile level</td>
<td>f↑ Fear</td>
</tr>
<tr>
<td>↓ Happiness, Active, Positive, Alert, Interest Profiles nonparallel</td>
<td>↓ Happiness, Positive, Alert, Interest Profiles nonparallel</td>
<td>a↑ Anger</td>
</tr>
<tr>
<td>↑ Shame, Fear, Sadness, Anger, Heartbeat, Tense, Profile level</td>
<td>↑ Positive Profiles nonparallel</td>
<td>Profiles nonparallel</td>
</tr>
<tr>
<td>↓ Active, Positive, Alert Profiles nonparallel</td>
<td>Profiles nonparallel</td>
<td></td>
</tr>
<tr>
<td>↑ P-wave amplitude, relative Q-T time, Ejection speed, Heart index, CO, SBP, DBP, SCR-No., SCR-amplitude, SCL, Respiration rate, EMG zygomaticus, Profile level</td>
<td>↑ P-wave amplitude, relative Q-T time, Ejection speed, Heart index, CO, SCR-No., SCR-amplitude, SCL, Respiration rate, EMG zygomaticus, Profile level</td>
<td>Profiles nonparallel</td>
</tr>
<tr>
<td>Profiles nonparallel</td>
<td>Profiles nonparallel</td>
<td></td>
</tr>
<tr>
<td>↓ HP-variability Profiles nonparallel</td>
<td>↓ HP-variability Profiles nonparallel</td>
<td>Profiles nonparallel</td>
</tr>
<tr>
<td>↑ P-wave amplitude, relative Q-T time, SBP, DBP, SCR-No., TMP-forehead, Profile level</td>
<td>↑ P-wave amplitude, relative Q-T time, SBP, DBP, SCR-No., TMP-forehead, Profile level</td>
<td>Profiles nonparallel</td>
</tr>
<tr>
<td>Profiles nonparallel</td>
<td>Profiles nonparallel</td>
<td></td>
</tr>
</tbody>
</table>
| Notes. Emotion responses refer to (treatment–control) contrasts. Specific responses refer to (fear–anger)–(treatment–control) tetrad contrast. Up arrows denote scale or function increases; down arrows, decreases. f = fear-specific. a = anger-specific. For abbreviations of variables, see Table 1.
strong cardiovascular activation that comprised tachycardia; left-
ventricular contractility increases leading to shortened preejection 
period and left-ventricular ejection time; reduction of stroke vol-
ume; elevation of cardiac output, systolic, and diastolic blood pres-
sure; heightened electrodermal activity; and finger vasoconstriction. 
Thus, there was a considerable overlap of fear and anger responses 
during real-life. How can this overlap be explained? To be sure, the 
answer to this question is decisive for conclusions about physio-
logical emotion specificity.

**Emotion nonspecificity.** A first explanation for the overlap of 
somatovisceral fear and anger responses during real-life is emotion 
nonspecificity. According to this model, fear and anger inductions 
led to one and the same state of general bodily activation, for 
nonspecificity. According to this model, fear and anger inductions 
are discussed.

Overlap, profile patterns were still different. Three arguments will 
be discussed. (a) Perhaps the fear task was more difficult and 
required more effort for its completion than the anger task. How-
ever, fear and anger control groups, which should capture exactly 
such task demands, had equal profile levels. (b) Because low to 
moderate fear may increase the effort to master a task, perhaps the 
fear treatment subjects were more aroused than the anger treatment 
subjects. Under this hypothesis we would expect one or more of 
the following observations: Fear treatment subjects should differ 
from both fear control subjects and anger treatment subjects in 
higher ratings of self-reported interest/motivation, activity/energy, 
and alertness/attentiveness; in higher ratings of the postexperimen-
tal interview item “How important was it for you to master the task 
very well?”; and in faster and louder speech of a short standard 
sentence that for a different research question had been recorded 
right after the induction periods. However, compared to fear con-
trols, fear treatment subjects were less interested, active, or alert; 
they had identical ratings of task importance (between the rating 
categories “somewhat” and “rather”); and speech time and loud-
ness were equal. Compared to anger treatment, fear treatment sub-
jects had identical self-reports of interest, activity, and alertness; 
they also had identical ratings of task importance; and they spoke 
considerably slower and in a lower voice, which conversely re-
acts the energizing effects of anger. Taken together, these data 
clearly contradict a fear-specific effort expenditure hypothesis. (c) 
With growing general bodily activation, perhaps the characteristic 
operation modes of variables diverge more and more. This hypothe-
sis could explain fear and anger profile pattern differences if 
profile levels also differ markedly. However, even though this 
explanation is necessary, it is not sufficient, because other causes 
for pattern differences are conceivable. As already noted above, 
bodily activation seldom is “general.” The mammalian organism is 
capable of orchestrating patterned somatovisceral activations, and 
this has often been demonstrated in the physiological and psycho-
physiological laboratory. Thus, we cannot decide whether charac-
teristic operation modes are the only or even the most likely causes 
of pattern differences.

In sum, weighing all three arguments for the hypothesis of emo-
tion nonspecificity as an explanation of somatovisceral fear and 
anger overlap, we do not find compelling evidence in favor of it.

**Context-dependent common mechanism.** This explanation pos-
ts its that depending on the context, fear and anger may trigger one 
and the same mechanism, which is responsible for the overlap of 
autonomic responses. A well-documented mechanism, the defense 
reflex (Hilton, 1982), is of particular interest here. In the cat and 
in the rat, it can be elicited by L-glutamate microinjections into the 
midbrain periaqueductal gray matter (Bandler, 1982; Bandler & 
Keay, 1996; Hilton & Redfern, 1987). The defense reflex prepares 
“an organism to cope with an emergency and specifically to per-
form the extreme muscular exertion of flight or attack. This is well 
exemplified by the pattern of cardiovascular response which is 
characteristic of the alerting stage of these reactions” (Hilton, 1982, 
p. 159). The cardiovascular manifestations of the defense reflex 
include vasodilatation in skeletal muscle; venoconstriction; vaso-
constriction in the splanchnic area, kidneys, and skin; increases in 
heart rate, in the contractile force of the heart, and in cardiac 
output; finally and most important for the present discussion, in-
creases in both SBP and DBP, which together with the simulta-
neous tachycardia point to a profound inhibition of the baroreceptor 
reflex. The chronotropic, inotropic, and dromotropic response pat-
tern, the marked vasoconstriction in the skin, and the pressor re-
response in both SBP and DBP indeed suggest the elicitation of a 
defense reflex in both fear and anger real-life inductions. During 
anger imagination, signs for a defense reflex were weaker; cardiac 
output and left ventricular contractility increases were missing in 
particular. In sum, both the fear and the anger real-life inductions 
provoked an alerting response, which had a pattern that is very 
similar to the description of the “defense reflex.” During imagi-
nation, this alerting response was largely absent.

**Specificity in Somatovisceral Responses**

Nonparallel fear and anger response patterns suggested that at least 
part of the fear and anger responses did not overlap. The specific 
face response (Table 2) was an even more marked chronotropic 
and inotropic activation, which however lacked an even larger 
pressor response. Thus, the specific fear response was not just a 
stronger defense reflex, but resembled an adrenaline-like response 
pattern (Sibolboro Mezzacappa, Kelsey, & Kaatkin, 1997; Wenger 
et al., 1960). Funkenstein had advanced the adrenaline hypothesis 
of fear years ago (Funkenstein, 1955; see also Wagner, 1989). This 
result found during real-life closely parallels the fear response 
during imagination reported by Sinha et al. (1992), where CO, 
heart rate, and SBP increases as well as TPR, PEP, LVET, and DBP 
decreases were observed. The specific fear response in the present 
study also included more numerous SCR responses and larger 
EMG corrugator activity.

The specific anger response comprised increased skin tempera-
ture at the forehead and larger EMG extensor activity during 
real-life, paralleling the results of Stemmler (1989). During imagi-
nation, these responses were also present (see Figure 3). Perhaps 
due to the slight anger response in anger controls, they missed 
significance when compared to the fear response. A hot face and 
the clenched fist are strong expressive signs of anger, identified 
here in miniature by their somatovisceral manifestations. The al-
leged specific DBP increase during anger in the present and in 
other real-life studies was probably masked by the marked defense 
reflex common to both fear and anger; but it did show up during 
imagination.

**A Component Model of Somatovisceral Response**

This article started out from the notion of emotion—context con-
founders suggesting (a) that emotion and context would each exert 
their unique effects on somatovisceral responses and (b) that the 
control groups would capture most of the context effects. How-
However, detailed inspection of our results revealed the importance of considering three components that shape somatovisceral fear and anger responses. To reflect the conceptual implications of this research, we propose a Component Model of Somatovisceral Response Organization. The first component is constituted by the “nonemotional” induction context, for example, body posture, ambient temperature, ongoing motor activity, or demands by cognitive processes not in the service of emotions. Component I conditions constrain the somatovisceral effects of the other components. In the present investigation, this component was reflected in large part by the control group responses. The second component embraces the effects of organismic, behavioral, and mental demands forced or allowed by a certain context and triggered by the functional goals of an emotion. Thus, when an organism enters an emotional state, this component allows for a flexible organization of bodily resources given the momentary situational circumstances. Examples of this second component are the alerting response of the defense reflex, which is allocated here to the separate legged fight-flight response should be redefined as the alerting response of the defense reflex, which is allocated here to the separate

hypothesized that the function of an emotion signature is to prepare the organism for the emotion-specific, upcoming need to act and to protect itself with a hardwired, fixed somatovisceral adaptation. In the case of fear, it could be preparation for flight. In the case of anger, it could be preparation for fight. But Cannon’s (1929) alleged fight-flight response should be redefined as the alerting response of the defense reflex, which is allocated here to the separate second component of somatovisceral response organization.

In conclusion, this study found evidence for considerable emotion–context confounds and with it for the concept of context–deviation specificity. The proposed Component Model of Somatovisceral Response Organization states that emotion–context confounds may operate in two distinct ways: A first component of response organization is independent of the emotion, whereas a second one is intertwined with the emotion. A third component of the model, the emotion signatures of fear and anger, could at least partially be statistically identified as specific, nonoverlapping emotion responses and thus also be separated from the context-related components. Future research will have to disentangle the context effects of the second component from emotion signatures also experimentally, as has been done here with the first component by use of appropriate control groups.

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