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Dysphoria and Mobilization of Mental Effort: Effects on Cardiovascular Reactivity

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Abstract Two studies examined the influence of dysphoria on motivational intensity in a student sample. Participants worked on a memory task (Study 1) or a mental concentration task (Study 2) without fixed performance standard ("do your best"). Based on their scores on the Center for Epidemiologic Studies – Depression Scale (L. S. Radloff, 1977), dysphoric and nondysphoric students were compared with regard to their effort-related cardiovascular reactivity during task performance. As predicted on the basis of the mood-behavior-model (G. H. E. Gendolla, 2000) and motivational intensity theory (J. W. Brehm & E. A. Self, 1989), dysphoric participants showed stronger cardiovascular reactivity while working on the cognitive tasks than nondysphoric participants. In Study 1, nondysphoric participants performed better on the memory task than dysphoric participants. Theoretical implications are discussed.

Keywords Dysphoria · Negative affect · Effort mobilization · Cardiovascular reactivity · Motivational intensity

People suffering from depression or dysphoria are said not only to experience long lasting negative mood and to have emotional, functional, and cognitive deficits, but also to lack motivation (Heckhausen, 1991). A motivational deficit in depression has been considered under different perspectives—amongst others with respect to behavioral approach and inhibition systems (Gray, 1982), self-regulation (e.g., Strauman, 2002), motivational influences on cognitive deficits (e.g., Hertel, 2000), and mood-congruent negative cognitions (e.g., Scott & Ingram, 1998).

Our research addresses the question if dysphoria and depression are associated with motivational deficits with regard to the *intensity* of motivation. Specifically, we are interested in the mobilization of mental effort during performance of instrumental tasks as an indicator of the intensity of motivation at a certain point in time. Based on the notion that dysphoria is largely characterized by a depressed mood we posit that moods play an important role in determining how dysphoric individuals react to behavioral challenges when they need to self-regulate the mobilization of resources for performance. Based on the research reported above, the mood-behavior-model (MBM; Gendolla, 2000), and motivational intensity theory (Brehm & Self, 1989) we develop and test predictions for the impact of dysphoria on motivational intensity. Effort mobilization is quantified as the reactivity of the cardiovascular system, especially the reactivity of systolic blood pressure (Gendolla & Brinkmann, 2005; Wright, 1996).

Dysphoria, motivation, and self-regulation

Before describing theory and hypotheses in detail we highlight some findings in depression and dysphoria research—stemming from clinical patient as well as subclinical community or student samples—which address motivational and self-regulation processes. First, there is evidence based on behavioral, self-report, and neurophysiological measures, that depression is associated with weak approach-motivation, as indicated by a relative hypoactivation of the left frontal cortex and lower responsiveness to rewards (e.g., Davidson, Pizzagalli, & Nitschke, 2002; Fowles, 1994; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 2000; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Tomarken

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& Keener, 1998). Findings with respect to a hyperactivation of behavioral inhibition in depression or dysphoria are existent but less consistent (e.g., Dickson & MacLeod, 2004; Kasch et al., 2002; Pinto-Meza et al., 2006).

In light of this evidence, Strauman (2002) postulated an incorporation of the self into the brain-behavior system in order to better describe and understand human approach and avoidance motivation. Accordingly, depressive individuals suffer from impairments of self-regulation resulting in affective and motivational deficits. The importance of self-regulation has also been stressed by others who point to the fact that the cognitive deficits commonly observed in depressive individuals and patients (see Burt, Zembar, & Niederehe, 1995; Rogers et al., 2004, for reviews) might partially be caused by motivational deficits. There is, for instance, evidence that aspects of self-regulation problems, such as rumination (Hertel, 1998; Watkins & Brown, 2002), unrealistic intentions (Kuhl & Helle, 1986), and deficits in attention control and initiative (Hertel, 2000), play an important role in the association between depression or dysphoria and cognitive deficits. Likewise, Abramson, Alloy, and Rosoff (1981) concluded that depressed individuals suffer from a motivational rather than from a cognitive-associative deficit.

Furthermore, studies on the mood-congruent memory bias in depression and dysphoria point at reduced self- and affect-regulation abilities. Josephson, Singer, and Salovey (1996) found that depressed individuals were not able to self-regulate their negative affect by activating positive memories (mood-incongruent recall). Likewise, Gilboa and Gotlib (1997) showed that previously dysphoric individuals better recalled negative stimuli and stayed longer in a negative mood after a negative mood induction than did never-dysphoric individuals. Greenberg and Pyszczynski emphasized the role of the depressive self-focusing style as a mediator for attributions after success or failure that leads to a self-serving bias in nondepressed but not in depressed individuals (e.g., Greenberg, Pyszczynski, Burling, & Tibbs, 1992; Pyszczynski & Greenberg, 1987).

Finally, there is evidence that depression and dysphoria can—besides other negative cognitive biases—influence judgments and evaluations (e.g., Scott & Ingram, 1998). Lyubomirsky, Tucker, Caldwell, and Berg (1999) reported that dysphoric individuals rated the probability of solving a personal problem lower than did nondysphoric individuals. The authors concluded that dysphoric rumination depletes energy and motivation and interferes with efforts to problem solving.

Mood and effort mobilization

A number of recent studies on the effects of transient mood states on the mobilization of mental effort have supported the predictions of the MBM (Gendolla, 2000). This model posits that moods per se do not have motivational implications, but that they influence behavior in the context of behavioral challenges, such as instrumental tasks. One of the two processes proposed to influence behavior is the *informational mood impact*, which states that moods influence the intensity and persistence of behavior. This impact presumes that moods may influence behavior via mood-congruency effects on behavior-related judgments and evaluations. Accordingly, people in a negative mood tend to appraise a given task as more difficult and their own capacities as lower than do people in a positive mood. Gendolla and colleagues have found clear support for these assumptions (see Gendolla & Brinkmann, 2005; Richter, Gendolla, & Krüsken, 2006, for reviews).

The authors manipulated their participants' mood and subsequently asked them to do their best (i.e., to self-regulate resources for performance) on a mental task for which no performance standard was provided.[1] By this means, they could show that participants in a negative mood actually mobilized more effort than participants in a positive mood as indicated by stronger systolic blood pressure reactivity of negative mood participants (e.g., Gendolla, Abele, & Krüsken, 2001; Gendolla & Krüsken, 2002a, Experiment 2). The replicated finding from studies administrating different mood induction procedures and different types of cognitive demands was that cardiovascular activity did not differ between positive and negative mood groups before and after the mood induction. It was only at the time of task performance that negative mood led to stronger cardiovascular reactivity than positive mood. This effect did not occur because of different importance attributed to the task, but because of subjectively higher task demand in a negative mood (e.g., Gendolla & Krüsken, 2002a,b). Similar results have been found by Wright and colleagues in samples with high vs. low perceived ability (e.g., Wright & Dill, 1993; Wright, Murray, Storey, & Williams, 1997). The mood effect on difficulty appraisals and systolic blood pressure reactivity during performance diminished when the significant value of mood as information was reduced (Gendolla & Krüsken, 2002c).

These studies also support the notion that the intensity of motivation can be reliably operationalized by cardiovascular reactivity as proposed by Wright's integration of motivational intensity theory (Brehm & Self, 1989) and Obrist's (1981) active coping approach (see Wright, 1996; Wright & Kirby, 2001, for reviews). The active coping approach

[1] Motivational intensity theory distinguishes between tasks with and without fixed performance standards, that is, with or without a certain performance level to attain. Tasks without fixed performance standards are labeled "unfixed" difficulty tasks; the difficulty level can be determined by the participants themselves who are simply asked to do their best (Brehm & Self, 1989).

builds on evidence that β-adrenergic influences exerted by the sympathetic nervous system on the cardiovascular system are proportional to effort and task engagement (Obrist, 1981). Therefore, assessing the reactivity to a behavioral challenge of cardiovascular indices reflecting β-adrenergic sympathetic activation provides an effective means of operationalizing task engagement. This line of research has mainly focused on systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) (Wright, 1996; Wright & Kirby, 2001), even though other more direct measures as for instance pre-ejection period are also conceivable.

However, especially SBP has been proven to be a reliable index of effort mobilization, which makes sense with respect to sympathetic and parasympathetic influences on the heart: SBP mainly varies with the contractility strength of the heart muscle. Myocardial contractility is known to directly increase with sympathetic discharge, which is related to activation. Consequently, an increase in SBP activity is regarded as a reliable indicator of increased task engagement. In contrast, DBP is predominantly determined by the overall flow resistance of the blood vessels in the body. Total peripheral resistance, however, is not systematically influenced by sympathetic activation, and therefore DBP reactivity constitutes a less reliable index of effort mobilization. Finally, HR is independently determined by both sympathetic (i.e., HR accelerating) and parasympathetic (i.e., HR decelerating) influences. An increase in HR can therefore represent both sympathetic activation and parasympathetic withdrawal. Consequently, HR can be considered an indicator of sympathetic activation to the extent that its influence is not masked by parasympathetic activity. In summary, SBP is a reliable and sensitive indicator of effort mobilization, even though increases in sympathetic activation may also result in simultaneous elevations of SBP, DBP, and HR (see Brownley, Hurwitz, & Schneiderman, 2000; Obrist, 1981; Papillo & Shapiro, 1990; Smith, Allred, Morrison, & Carlson, 1989, for more details).

The present studies

The present studies tested the central hypothesis that dysphoric individuals would show stronger cardiovascular reactivity—especially SBP—while performing a cognitive task without fixed performance standard than nondysphoric individuals. The main advantage of operationalizing motivational intensity as cardiovascular reactivity is that it is less susceptible to social presentation biases than self-report measures and less influenced by individual capacity and performance strategies than performance measures. Therefore, no directed hypotheses were formulated with regard to performance on the cognitive tasks. On the one hand, performance has been shown to be positively related to effort mobilization (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002a,b,c). But on the other hand, there is evidence that depressed and dysphoric individuals perform worse in cognitive tasks than normal controls (e.g., Burt et al., 1995; Rogers et al., 2004). Thus, in the present context of dysphoria effects on motivational intensity, performance was assessed exploratively.

We chose a student sample with extreme scores on a self-report depression scale for our studies. We did not expect that students with high scores were suffering from manifest clinical depression but assumed that they would be dysphoric, show subclinical symptoms, and have higher vulnerability for depression. Therefore, we also assessed participants' current mood state. Moreover, there are good reasons to choose a student sample for depression research because of many environmental stressors, high prevalence of depressive symptoms, and lower probability of other (psycho-) pathologies and concurrent treatments in such a sample, as well as the homogeneity of the group (Vredenburg, Flett, & Krames, 1993). Results concerning gender differences in student samples with respect to depressive symptoms are mixed: The usual gender differences between women and men in both clinical and subclinical populations are not consistently found in student samples (Nolen-Hoeksema, 1987, 2001). Thus, we decided to include only women in our sample with the objective of reducing variability in group assignment due to gender biases in the self-report of depressive symptoms. Finally, we chose cognitive tasks that demanded a minimum of motor movement from the participants to ensure that cardiovascular changes could be attributed to the mobilization of mental resources rather than to metabolic movement effects.

Study 1

This study compared two groups of university students (dysphoric vs. nondysphoric) with regard to their cardiovascular reactivity during performance of a memory task. We expected performance-related cardiovascular reactivity—especially SBP—to be more pronounced in dysphoric than in nondysphoric participants.

Method

Participants

One hundred forty-seven students in an introductory psychology course participated in questionnaire sessions in exchange for course credit. Out of this sample 35 women (mean age 23 years) who scored in the lower or the upper quartile of the Center for Epidemiologic Studies – Depression Scale (CES-D; Radloff, 1977) were randomly selected for participation in

an ostensibly unrelated experiment via a personal code which guaranteed anonymity. Seventeen participants were situated in the upper quartile of the CES-D ($M = 33.47$, $SE = 2.15$) and therefore referred to as dysphoric. Eighteen participants were located in the lower quartile of the CES-D ($M = 6.61$, $SE = 0.66$) and therefore referred to as nondysphoric.

Physiological apparatus

Cardiovascular measures were obtained noninvasively with a computer-aided, multichannel monitor (Par Electronics Physioport III) that uses oscillometry to determine SBP (millimeters of mercury [mmHg]), DBP (millimeters of mercury [mmHg]), and HR (beats per minute [bpm]). A blood pressure cuff (Boso) was placed over the brachial arteria above the elbow of participants' nondominant arm and inflated automatically every 2 min during 2 measurement periods—habituation (baseline) and task performance. Each single measure took less than 1 min. Obtained values were stored on a computer disk so that both experimenter and participants were ignorant of all physiological values measured during the experiment.

Self-report data

In order to survey depressive symptomatology, the French version of the CES-D (Fuhrer & Rouillon, 1989) was administered in the mentioned questionnaire sessions about 2 weeks prior to the experiment. The CES-D is a short self-report scale especially developed for use in community samples and consists of 20 items asking for frequency of depressive symptom experience on a scale from 0 (*never, very seldom*) to 3 (*frequently, always*). Radloff (1977) proposed a cut-off score of 16 to distinguish depressed from nondepressed individuals, while Fuhrer and Rouillon (1989) favored cut-off scores of 17 and 23 for men and women, respectively. The CES-D has proven its excellent validity in depression research and is reported to have better scale discriminability than the Beck Depression Inventory (see Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). It also showed high internal consistency ($\alpha = .96$).[2]

At the beginning of the experimental session, we administered the positive (i.e., "happy," "joyful," "contented," "cheerful") and negative (i.e., "sad," "frustrated," "depressed," "dissatisfied") hedonic tone scales of the UWIST mood adjective checklist (Matthews, Jones, & Chamberlain, 1990) in order to assess participants' momentary mood state. Participants indicated the extent to which each adjective reflected their momentary feeling state on 7-point scales ranging from 1 (*not at all*) to 7 (*very much*).

Procedure

The study was conducted with the help of the psychological software E-Prime (version 1.1, Psychology Software Tools) by an experimenter who was hired and unaware of both the hypotheses and participants' depression status. The experimental session took about 30 min and was described as an investigation of physiological activity during periods of relaxation and demand. Participants, who attended the session individually, were greeted and seated in front of a computer. After signed consent was obtained, the experimenter attached the blood pressure cuff and gave instructions for computer use. Then she left the room and monitored the experiment from an outside control room. Computer-generated beeps informed her about the beginning of each experimental period without the need for seeing the participants and their entries on the computer. The experiment started with general instructions and assessment of biographical data and participants' momentary mood state with the UWIST scale.

For the following habituation period, the experimenter reentered the room, handed a magazine to the participant, and informed her that she was allowed to read for a period of about 9 min while cardiovascular measures would be taken. Then the experimenter left the room and started the blood pressure monitor. After assessment of the five cardiovascular baseline values, the experimenter reentered the room, took the magazine, and instructed the participant to continue working on the computer.

The experiment continued with instructions concerning the memory task. Participants were instructed to memorize within 5 min as many letter series (e.g., EPQZ) as possible out of a list of 15 senseless letter series and to note the recalled series afterwards. That means that we simply asked them to do their best instead of providing them with a fixed performance standard (i.e., a fixed number of letter series to memorize). A preview of the list of letter series was then presented for 1 s on the computer screen to give participants an impression of the task. Then the 5 min performance period started. Meanwhile, the 15 letter series were simultaneously presented on the computer screen and 3 cardiovascular measures were taken 15 s, 135 s, and 255 s after task onset. After having noted the letter series they could recall, participants learned that the experiment was over. The experimenter reentered the room, removed the blood pressure cuff, and thanked the participant. Finally, participants were carefully debriefed, interviewed with regard to suspicion, and received their course credit.

[2] We also included the BDI-II (Beck, Steer, & Brown, 1996) in the initial questionnaire session. Participants' scores on the BDI-II were highly correlated with their CES-D scores, $r(35) = .96$, $p < .001$. Group assignment did not change when considering the BDI-II instead of the CES-D for the selection process.

Results

Self-reported mood

We calculated mood scores by summing the scores of the positive ($\alpha = .93$) and the negative ($\alpha = .88$) hedonic tone adjectives of the UWIST scale. Additionally, we also formed a global mood score by summing the positive and the reversed scored negative adjectives of the UWIST scale ($\alpha = .88$). Results revealed highly significant differences between the two dysphoria groups with regard to their momentary mood state at the beginning of the experiment: Mood scores of dysphoric participants on the positive adjectives ($M = 16.29$, $SE = 1.11$) were significantly lower than those of nondysphoric participants ($M = 20.72$, $SE = 1.17$), $t(33) = 2.73$, $p < .01$. The reverse was found for the mood scores on the negative adjectives (dysphorics: $M = 11.12$, $SE = 1.31$ vs. nondysphorics: $M = 4.67$, $SE = 0.29$), $t(17.57) = 4.79$,[3] $p < .001$. Finally, dysphoric participants ($M = 37.18$, $SE = 1.95$) had lower scores on the global mood score than nondysphoric participants ($M = 48.06$, $SE = 1.25$), $t(33) = 4.75$, $p < .001$. Accordingly, as expected, dysphoric students were in a more negative and a less positive mood when arriving at the laboratory.

Cardiovascular baselines

Cardiovascular baseline scores for SBP, DBP, and HR were created by averaging the last two values obtained during habituation (αs = .91 for SBP baseline, .79 for DBP baseline, and .96 for HR baseline).[4] Means and standard errors are presented in Table 1. According to independent-samples *t*-tests, there were no significant differences between dysphoric and nondysphoric participants with regard to their cardiovascular baseline measures; SBP and HR: *t*s < 1, *p*s > .50; DBP: $t(33) = 1.60$, $p > .13$.

Cardiovascular reactivity

We calculated change (delta) scores for each participant by subtracting the baseline values from the arithmetic means obtained during task performance (see Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). A preliminary analysis revealed that DBP and HR delta scores were not correlated with the respective baseline values, *r*s < .21, *p*s > .23. In contrast,

Table 1 Means (and Standard Errors) of cardiovascular baselines in Study 1

Cardiovascular baselines	Dysphoric		Nondysphoric	
	M	(*SE*)	*M*	(*SE*)
SBP	112.82	(1.88)	112.33	(1.32)
DBP	74.35	(0.84)	72.92	(0.37)
HR	76.79	(2.57)	75.31	(2.21)

Note. $N = 17$ (dysphoric) and $N = 18$ (nondysphoric). SBP and DBP are indicated in millimeter of mercury (mmHg), HR is indicated in beats per minute (bpm).

SBP delta scores were significantly correlated with systolic baseline values, $r(35) = -.38$, $p < .03$. Therefore, we adjusted systolic change scores with respect to SBP baseline values in order to prevent carry-over effects and biases due to the law of initial values (see Llabre et al., 1991).

SBP. We first submitted the baseline-adjusted reactivity scores (delta) to an independent-samples *t*-test. Results revealed that albeit dysphoric participants ($M = 5.92$, $SE = 1.49$) showed stronger SBP reactivity than nondysphoric participants ($M = 3.64$, $SE = 1.07$), the difference did not reach significance, $t(33) = 1.25$, $p = .11$.[5] Subsequently, we analyzed the single reactivity scores for each measurement time (delta 1, delta 2, and delta 3). This procedure is reasonable because of the above mentioned self-regulation difficulties in depression that might implicate difficulties in maintaining self-regulation (and effort mobilization) over a certain period. Therefore, we submitted the single baseline-adjusted reactivity scores to a 2 (dysphoria) × 3 (time) mixed model ANOVA to test if systolic reactivity changed over time. This analysis revealed no main effect of time, $F(2, 64) = 1.64$, $p = .20$, but a significant dysphoria × time interaction, $F(2, 64) = 3.69$, $p = .03$, indicating that dysphorics' reactivity declined over time while nondysphorics' reactivity remained relatively stable (see Fig. 1). To further highlight this effect, we compared dysphoric and nondysphoric individuals with regard to each single baseline-adjusted reactivity score. As depicted in Fig. 1, dysphorics generally showed stronger SBP reactivity than nondysphorics. However, only the difference on the first reactivity measure delta 1 was reliable (dysphorics: $M = 7.31$, $SE = 1.64$ vs. nondysphorics: $M = 3.59$, $SE = 1.15$), $t(33) = 1.88$, $p = .03$.[5]

DBP and HR. There were no differences between dysphoric and nondysphoric participants with regard to their DBP and HR overall reactivity scores (delta), as indicated by

[3] Degrees of freedom are adjusted because of inequality of variances.

[4] The reason for this is that repeated measures ANOVAs revealed a decline of cardiovascular values over the first measures, while the last two measures remained stable (*p*s > .30). This decline is a common finding and due to habituation to the experimental setting and the fact being seated for a while.

[5] Given our directed a priori hypothesis we conducted one-tailed *t*-tests for comparisons between dysphoric and nondysphoric groups with regard to their cardiovascular reactivity.

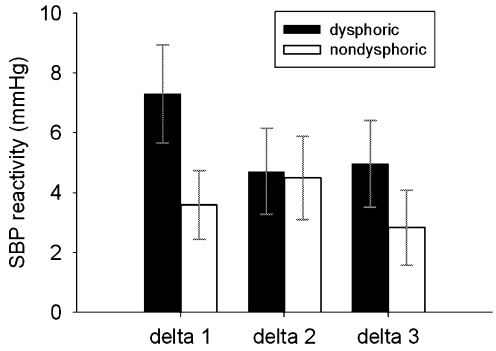

Fig. 1 Cell means and standard errors of systolic blood pressure reactivity for measurement times 1, 2, and 3 in Study 1. Cell means of systolic reactivity scores are baseline-adjusted

independent-samples *t*-tests, $ts < 1$, $ps > .17$.[5] We then calculated single reactivity scores for DBP and HR as we did for SBP and submitted them to 2 (dysphoria) × 3 (time) mixed model ANOVAs. Results revealed neither main effects of time nor dysphoria × time interactions, $Fs < 2.15$, $ps > .13$. Therefore, we did not further compare the DBP and HR single reactivity scores.

Task performance

In order to investigate participants' task performance, we analyzed the total number of recalled letter series, the number of correctly recalled letter series, and the ratio of correctly to totally recalled letter series with independent-samples *t*-tests. Data of three participants were excluded because they differed more than two standard deviations from the respective group mean, so that there were 16 participants per group. With regard to the number of correctly recalled letter series (nondysphorics: $M = 6.56$, $SE = 0.56$ vs. dysphorics: $M = 4.63$, $SE = 0.42$), $t(30) = 2.79$, $p < .01$, as well as the ratio of correctly to totally recalled letter series (nondysphorics: $M = 0.82$, $SE = 0.04$ vs. dysphorics: $M = 0.68$, $SE = 0.05$), $t(30) = 2.16$, $p < .04$, nondysphoric students significantly outperformed dysphoric students. Results regarding the total number of recalled letter series also revealed that nondysphoric students tended to show better performance than dysphoric students (nondysphorics: $M = 7.94$, $SE = 0.45$ vs. dysphorics: $M = 6.81$, $SE = 0.37$), $t(30) = 1.93$, $p = .06$. The three performance indices were not substantially correlated with the baseline-adjusted SBP reactivity scores (delta, delta 1, 2, and 3), $-.28 < r < -.05$, $ps > .12$.

Discussion

The present results confirmed our central prediction that dysphoric participants would show stronger systolic reactivity when faced with a mental challenge without fixed performance standard than nondysphoric participants. As expected and in accordance with the MBM (Gendolla, 2000) statement that mood states per se do not have motivational implications, we found no differences in cardiovascular baseline measures between dysphoric and nondysphoric participants. As outlined earlier, systolic reactivity was our primary index for measuring effort intensity because SBP, which largely depends on myocardial contractility, is systematically influenced by sympathetic discharge to the heart (Brownley et al., 2000; Obrist, 1981; Wright, 1996).

Results actually revealed that dysphoric participants showed stronger SBP reactivity—reflecting the mobilization of more mental effort—than nondysphoric participants. Nevertheless, this difference proved to be significant only for the first of the three measures taken during task performance. For the other two measures as well as for the overall reactivity index results pointed into the same direction. In addition, there was a decline of SBP reactivity over time in the dysphoric group. One could conceive different explications for this finding. For instance, given self-regulation difficulties in depression, it could be that our dysphoric participants mobilized much effort in the beginning but failed to maintain this level of effort over the five-minute period. As Tillema, Cervone, and Scott (2001) pointed out, depressed individuals tend to set higher performance standards than nondepressed individuals. Possibly, participants in the dysphoric group had very high standards and reduced effort mobilization as they realized that they could not meet them.

Interestingly, performance quantified as the number of totally and correctly recalled letter series, as well as the ratio between them did describe the opposite pattern of SBP reactivity during task performance. According to our results, nondysphoric participants clearly outperformed dysphoric participants. This is in accordance with results showing that depressed and dysphoric individuals tend to have cognitive deficits (e.g., Burt et al., 1995; Rogers et al., 2004). Finally, this study revealed highly significant differences between the dysphoric and nondysphoric groups with regard to their momentary mood state. As anticipated, participants who were classified as dysphoric on the basis of the CES-D two weeks prior to the experimental session reported being in a more negative mood than nondysphorics at the beginning of the experiment.

Study 2

This study was designed to replicate and extend the findings of Study 1. First, we replaced the memory task by a mental concentration task in order to address different cognitive processes and thus to facilitate generalization of our findings. In addition, following recommendations by Ingram and Siegle (2002) how to enhance the meaning of findings from research with subclinical populations, we assessed

self-reported dysphoria twice in order to ensure that the scores were stable. Only participants whose CES-D scores were located in the lower or upper third of the distribution at both the first (questionnaire session) and the second (experimental session) measurement time were retained for analyses. Moreover, we assessed participants' momentary mood state not before but after task performance. This was supposed to prevent participants from relying too much on their momentary mood state by being forced to think about it, and thus to show that our findings are independent of being concerned with one's momentary mood state. With some exceptions, we used the same materials and methods as in Study 1. In order to have an even better temporal resolution, cardiovascular values were taken every min.

Method

Participants

One hundred seventy-one students in an introductory psychology course participated in questionnaire sessions in exchange for course credit. Out of this sample we randomly selected women who scored in the lower or upper third of the CES-D for participation in an ostensibly unrelated experiment. As mentioned above, only data of participants whose scores remained within the limits set by the initial distribution (i.e., CES-D score $\leq$10 and $\geq$20, respectively) after the second assessment time were included in the analyses. Thus, data of 25 women (mean age 21 years) were retained and divided into 2 groups. Fourteen participants were referred to as dysphoric (CES-D [t1]: $M = 26.93$, $SE = 0.96$; CES-D [t2]: $M = 25.43$, $SE = 1.08$). Eleven participants were referred to as nondysphoric (CES-D [t1]: $M = 5.64$, $SE = 0.97$; CES-D [t2]: $M = 4.73$, $SE = 0.99$). The CES-D scales showed high internal consistency (αs = .93 at t1 and .94 at t2).[6]

Procedure

This study was conducted by means of the psychological software Inquisit (version 2.0, Millisecond Software) by an experimenter who was hired and unaware of both the hypotheses and participants' depression status. The procedure was identical to Study 1 with the exception of the type of cognitive task, the moment of mood assessment, and the second questionnaire. We administered a computerized task that was adapted from the "d2 mental concentration test" (Brickenkamp, 1981). Participants learned that different symbols would appear on the computer screen and that they had to decide for each stimulus whether it was the letter *d* accompanied by exactly 2 apostrophes, or not (i.e., the letter *d* with more or less than 2 apostrophes or the letter *p* with 1, 2, 3, or 4 apostrophes) by pressing a "yes" or a "no" key. They were advised to work as quickly but also as precisely as possible. That means that we did not provide them with a fixed performance standard (e.g., a fixed presentation time of each stimulus) but simply asked them to do their best. It has also to be acknowledged that for this task participants had to move, i.e., to press a button, in comparison with the quiet sitting in Study 1. Movement artifacts, however, were avoided by attaching the blood pressure cuff to the non-dominant arm. Before participants started the 5 min performance period, they performed 8 test trials to familiarize with the task. Then they worked for 5 min on this task while 5 cardiovascular measures were taken, starting 15 s after task onset. Subsequently, participants' momentary mood state was assessed with the UWIST scale. Finally, participants completed an ostensibly unrelated questionnaire (CES-D, BDI-II), were carefully debriefed, probed for suspicion, and received their course credit.

Results

Self-reported mood

As in Study 1, we calculated three scores based on the positive and negative hedonic tone adjectives of the UWIST scale (αs = .95 for the positive, .87 for the negative, and .93 for the global score). Results revealed that there were significant differences between dysphoric and nondysphoric participants with regard to their momentary mood state immediately after having worked on the cognitive task: Dysphorics ($M = 16.57$, $SE = 1.08$) had lower positive scores than nondysphorics ($M = 21.09$, $SE = 1.16$), $t(23) = 2.83$, $p < .01$. Furthermore, dysphorics ($M = 12.21$, $SE = 1.31$) showed higher negative scores than nondyshporics ($M = 6.18$, $SE = 0.69$), $t(19.18)^3 = 4.07$, $p < .001$. Finally, they also differed significantly on the global sum score (dyshorics: $M = 36.36$, $SE = 2.29$ vs. nondysphorics: $M = 46.91$, $SE = 1.53$), $t(21.60)^3 = 3.83$, $p < .001$.

Cardiovascular baselines

SBP and DBP baseline scores were created by averaging the last three of the eight values obtained during habituation period (αs = .97 for SBP baselines and .96 for DBP baselines).[7] HR baselines were determined by the last of the

[6] As in Study 1, scores of the BDI-II and the CES-D were highly correlated at both assessment times, rs > .83, ps < .001.

[7] The reason for this is that repeated measures ANOVAs revealed a decline of cardiovascular values over the first measures, while the last three measures remained stable (ps > .16).

Table 2 Means (and Standard Errors) of cardiovascular baselines in Study 2

Cardiovascular baselines	Dysphoric		Nondysphoric	
	M	(*SE*)	*M*	(*SE*)
SBP	100.73	(2.12)	103.95	(2.55)
DBP	64.33	(1.47)	67.51	(2.27)
HR	73.18	(0.52)	72.25	(0.41)

Note. $N = 14$ (dysphoric) and $N = 11$ (nondysphoric). SBP and DBP are indicated in millimeter of mercury (mmHg), HR is indicated in beats per minute (bpm).

eight measures.[8] Means and standard errors are presented in Table 2. According to independent-samples *t*-tests, there were no significant differences between the dysphoric and nondysphoric groups with regard to cardiovascular baseline measures ($ts < 1.33$, $ps > .20$).

Cardiovascular reactivity

As in Study 1 we calculated for each participant both an overall change score (delta) and single change scores for each cardiovascular measure taken during task performance (delta 1, delta 2, delta 3, delta 4, and delta 5) (see Llabre et al., 1991). The latter scores were calculated for the sake of comparability of results between Studies 1 and 2. There were no significant correlations between SBP and DBP reactivity scores and their respective baseline scores, $-.18 < r < -.15$, $ps > .40$. In contrast, HR baseline scores were significantly correlated with HR reactivity scores, $r(18) = -.58$, $p < .01$. Therefore, we adjusted HR change scores with respect to HR baseline scores (see Llabre et al., 1991).

SBP. We first compared dysphoric and nondysphoric individuals with regard to their overall reactivity score (delta). As expected and depicted in the left panel of Fig. 2, dysphoric participants ($M = 8.72$, $SE = 2.00$) showed stronger systolic reactivity than nondysphoric participants ($M = 4.20$, $SE = 1.39$), $t(23) = 1.76$, $p < .05$.[5] We then submitted the single reactivity scores to a 2 (dysphoria) × 5 (time) mixed model ANOVA. In contrast to Study 1, there was no significant change over time neither for the total of the participants, nor for either group, $Fs < 1.89$, $ps > .11$. Therefore, we did not further compare the single reactivity scores.

DBP. Comparison of the overall reactivity scores (delta) revealed that dysphoric participants ($M = 6.19$, $SE = 1.11$) tended to have stronger DBP reactivity than nondysphoric participants ($M = 3.70$, $SE = 0.97$), $t(23) = 1.63$, $p < .06$.[5] We then submitted the single reactivity scores to a 2 (dysphoria) × 5 (time) mixed model ANOVA. It revealed a significant decline in DBP reactivity over time in general, $F(4, 92) = 3.98$, $p < .01$, but there was no dysphoria × time interaction, $F(4, 92) = 1.38$, $p > .24$. Hence we did not further compare the single reactivity scores.

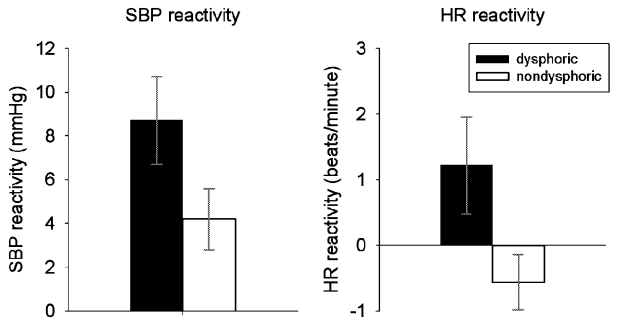

Fig. 2 Cell means and standard errors of overall systolic blood pressure reactivity (left panel) and heart rate reactivity (right panel) in Study 2. Cell means of heart rate reactivity scores are baseline-adjusted

HR. We first submitted the baseline-adjusted reactivity scores (delta) to an independent-samples *t*-test. Results revealed that dysphoric participants ($M = 1.22$, $SE = 0.74$) showed stronger HR reactivity than nondysphoric participants ($M = -0.56$, $SE = 0.42$), $t(17) = 1.90$, $p < .04$[5] (see right panel of Fig. 2). Next we submitted the single baseline-adjusted reactivity scores to a 2 (dysphoria) × 5 (time) mixed model ANOVA. There was neither an effect for the time factor, nor a dysphoria x time interaction, $Fs < 1.62$, $ps > .18$. Therefore, we did not further compare the single reactivity scores.

Task performance

We registered the following indices of participants' task performance: total number of stimuli a participant had performed, number of stimuli a participant had correctly identified, and the ratio between those indices. Moreover, we assessed the average response latency (in ms). Data of two participants were excluded form the analyses because they differed more than two standard deviations from the respective group means. We then submitted the indices to independent-samples *t*-tests. Results revealed that there were no reliable differences, $ts < 1$, $ps > .40$ (average number of stimuli performed: $M = 371.65$, $SE = 8.18$; average number of correctly identified stimuli: $M = 364.04$, $SE = 7.94$; average ratio of correctly to totally performed stimuli: $M = 0.98$, $SE = 0.003$; average response latency: $M = 779.37$, $SE = 19.16$). The four performance indices were not correlated with SBP reactivity (delta), $-.02 < r < .11$, $ps > .50$.

[8] The last three measures of HR baselines were stable ($p > .50$) but the internal consistency was insufficient ($\alpha = .44$). Therefore, we used only the last of the eight measures as HR baseline. In addition, because of problems with the measurement equipment there were some missing HR data, so that HR analyses are based on the data of 19 participants.

Discussion

The present study replicated and extended the findings of Study 1. Again, the dysphoria groups did not differ with regard to their cardiovascular baseline values. But when they were confronted with a cognitive challenge that asked for self-regulation, dysphoric participants showed significantly stronger systolic reactivity as well as marginally stronger diastolic reactivity than nondysphoric participants. Moreover, even if HR reactivity was generally rather low, dysphoric students showed significantly stronger HR reactivity than nondysphoric students. These findings hold true for the overall reactivity scores.

Unlike Study 1, there was no decline in systolic reactivity during performance in Study 2, neither for dysphoric nor for nondysphoric participants. One may argue that differences in task characteristics were responsible for dysphorics' decline of SBP reactivity during performance of the memory task in Study 1 as opposed to the mental concentration task in the present study. The d2 task continuously presented new stimuli on the screen, asking for a reaction and thus creating a new situation for each trial. Consequently, it might have been easier to maintain self-regulation during work on the d2 task because this task was more strongly controlled by the "outside"—that is, the computer program that successively presented stimuli. In contrast, the screen with the 15 letter series in the memory task of Study 1 did not change over the five min performance period and thus the situation rested more or less the same.

With regard to task performance, the differences between both dysphoria groups in the present study were not reliable. As for the pattern of cardiovascular reactivity, we think that task characteristics were responsible for the differences in performance results between Studies 1 and 2. As we argued above, the memory task in Study 1 required more self-regulation skills than the concentration task in Study 2. In consideration of research stating that depressed and dysphoric individuals have self-regulation deficits (e.g., Strauman, 2002; Tomarken & Keener, 1998) and need specific task characteristics in order to control their directed attention (e.g., Hertel, 2000), it is reasonable to assume that the d2 task in the present study provided dysphoric participants with the required hints so that they performed on the same level as nondysphoric participants.

Finally, the mood assessment showed, in accordance with the results of Study 1, that individuals with low and high self-reported dysphoria differed highly significantly with regard to their momentary feeling state, even if the CES-D and the UWIST scales were assessed with a time lag of several weeks. Moreover, because mood was assessed after task performance at the end of the experimental session, the results of Study 2 demonstrate that the effects of dysphoria on cardiovascular reactivity and performance are not due to a high salience of one's own feelings that might have been elicited by prior mood assessment.

General discussion

The main goal of our two studies was to test our prediction based on the MBM (Gendolla, 2000) that dysphoric individuals would show stronger effort-related cardiovascular reactivity than nondysphoric individuals when faced with a cognitive task without fixed performance standard. The results largely support our predictions. As expected, both studies found highly significant differences between dysphoric and nondysphoric participants' momentary mood states—regardless if mood had been assessed before or after completion of the mental task. Undoubtedly, even students selected via a self-report depression scale several weeks before the experimental session differed clearly with regard to their naturally occurring momentary mood.

More important, Study 1 provided first evidence for our central hypothesis insofar as the dysphoric group showed significantly stronger systolic reactivity at the beginning of the memory task, demonstrating that dysphoric participants actually mobilized more mental effort. Study 2 confirmed our hypothesis for the overall reactivity scores not only for SBP, but also for HR, and marginally for DBP. Even if among these indices SBP is the most reliable and sensitive effort-related cardiovascular reactivity parameter, there is evidence that SBP, DBP, and HR can also respond simultaneously in active coping (Brownley et al., 2000; Obrist, 1981; Papillo & Shapiro, 1990; Smith et al., 1989). Accordingly, dysphoric students mobilized more mental effort during the mental concentration task than did nondysphoric students. The fact that this impact of dysphoria on cardiovascular reactivity holds true for two different types of cognitive tasks—a memory and a concentration task—makes us confident that the findings can be generalized.

The present results question the notion that depressed and dysphoric individuals have a motivational deficit with respect to intensity of motivation. According to our findings, motivational intensity is higher in dysphoric than in nondysphoric individuals for tasks without fixed performance standard. Correspondingly, our results replicate and extend the findings by Gendolla and colleagues (see Gendolla & Brinkmann, 2005, for a review), in that not only experimentally manipulated, transient mood states, but also longer lasting affective dispositions that are related to the experience of certain moods can have an impact on effort mobilization. Most important, this effect is not attributable to individual differences regarding cardiovascular baseline values because both dysphoria groups did not differ from one another on these measures.

Furthermore, the results of the present studies are compatible with research in depression and cardiovascular disease. Accordingly, depression and other negative affect dispositions are among the risk factors for the development and worsening of cardiovascular disease (e.g., Frasure-Smith & Lespérance, 2005; Rugulies, 2002; Suls & Bunde, 2005). Our studies demonstrate that dysphoric individuals tend to mobilize more resources in terms of SBP reactivity when they are asked to do their best. A tendency to strong cardiovascular reactivity is in turn associated with and considered as a risk factor for the development of hypertension and cardiovascular disease (e.g., Kibler & Ma, 2004; Light, Dolan, Davis, & Sherwood, 1992). One may argue that among other factors, also affective states play an important role in the linkage between depression and coronary heart disease—for instance because of stronger sympathetic activation induced by subjectively higher task demand in a (dispositionally) more negative mood (see Gendolla & Richter, 2005).

Interestingly, the stronger resource mobilization of dysphorics was not related to performance gains. In Study 1, nondysphoric participants clearly performed better than dysphoric participants. In Study 2, where cardiovascular reactivity differences between both dysphoria groups were more obvious, no performance differences emerged. It seems that the cognitive deficit found in clinically and subclinically depressed individuals (e.g., Burt et al., 1995; Rogers et al., 2004) partly appears also in our dysphoric sample. In contrast to the findings by Gendolla and colleagues (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002a,b,c) who found positive associations between SBP reactivity and performance when mood was experimentally manipulated, the dysphoric participants in our studies did not profit from mobilizing more resources. One can think of several interpretations for this finding.

On the one hand, given the mentioned cognitive deficits in depression and dysphoria, it may be that these individuals generally have to mobilize more resources in order to reach the same results than normal controls (see Hockey, 1997; Wright, 1998). In this light, the dysphoric participants in Study 2 successfully compensated their slight disadvantage by mobilizing more effort, which resulted in a performance that was comparable with those of nondysphorics. However, when dysphoric participants did not maintain effort—as it seems to be the case in Study 1—they attained worse outcomes. On the other hand, given that pronounced cognitive impairments are unlikely to occur in a student sample, the worse performance of dysphoric participants in Study 1 may also reflect their difficulties in self-regulation and attention control (e.g., Hertel, 2000; Kuhl & Helle, 1986; Strauman, 2002). This explication seems plausible when one considers the different task characteristics of the cognitive tasks used in these studies and fits well the above mentioned evidence for motivational deficits being partly responsible for cognitive deficits. One could argue that cognitive deficits especially emerge in connection with a motivational deficit—what may explain the performance differences in Study 1 that did not emerge in Study 2.

The relatively weak association between performance and SBP reactivity in both studies is not surprising. There is evidence that in some circumstances effort may increase performance; previous research regarding the informational mood impact on effort mobilization, for instance, has found positive associations between performance and SBP reactivity (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002a,b,c). Nevertheless, it would be incorrect to equate the two because performance on a task is an *outcome* determined by effort, ability, and strategy use, and different tasks are differentially influenced by these factors (Locke & Latham, 1990).

Finally, some open questions and shortcomings concerning the present research should be mentioned. First, it is an important issue to investigate the mechanisms that are responsible for the stronger cardiovascular reactivity of dysphoric individuals in self-regulation. According to the MBM (Gendolla, 2000), behavior-related judgments and evaluations should mediate between depressed mood and effort mobilization. As outlined above, dysphoria has been shown to have an effect on subjective evaluations and ratings (e.g., Scott & Ingram, 1998). Alternatively, dysphoria might have influenced people's performance standards on this unfixed difficulty task, which led to stronger effort mobilization (see e.g., Tillema et al., 2001; Sherry, Hewitt, Flett, & Harvey, 2003, for research on perfectionistic standards in depression). Even if we are confident—based on prior research on experimentally manipulated mood (e.g., Gendolla & Krüsken, 2002a,b)—that dysphoria affected the subjective difficulty of the task, future research should address these possible mediations between depressed mood and effort mobilization. Second, the issue regarding the here reported disconnection between effort and performance needs to be further addressed. Finally, one shortcoming of our studies is their relatively small sample size and their limitation to women. Future research may benefit from extending the studies to a broader population recruited from the community with more severe depressive symptomatology.

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