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Reference


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Not Everyone’s Heart Contracts to Reward: 
Insensitivity to Varying Levels of Reward in Dysphoria

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Abstract

Reward insensitivity in depression and dysphoria has been demonstrated by self-report, behavioral, and neuroscience data. These findings show less anticipated and experienced pleasure to rewarding stimuli, no behavioral adaptation in anticipation of rewards, and altered functioning in reward-related brain areas. The present study expands previous research by using cardiovascular reactivity to three levels of reward as an indicator of effort mobilization. Undergraduates with low versus high depression scores worked on a cognitive task in anticipation of no, versus a small, versus a significant amount of money for successful task performance. Results of pre-ejection period and heart rate reactivity confirmed the expected linear increase as a function of reward value in nondysphoric participants and the expected blunted response across all reward levels in dysphoric participants. The present findings thus show that dysphoric individuals have a motivational deficit in terms of reduced effort-related cardiac reactivity when anticipating a monetary reward.

Keywords: depression, dysphoria, effort mobilization, cardiovascular reactivity, monetary reward, reward insensitivity
1. Introduction

1.1 Overview

There is ample evidence that the activity of the cardiovascular system during goal-directed actions sensitively responds to changes in hedonic consequences (Richter, 2012). For instance, it has been shown that higher reward value is associated with increases in heart rate in healthy individuals (e.g., Fowles, Fisher, & Tranel, 1982; Tranel, Fisher, & Fowles, 1982). More recently, a linear relationship between increasing reward value and increases in measures of sympathetic impact on the heart has been demonstrated in a healthy sample (e.g., Richter & Gendolla, 2009). On the behavioral level, people usually develop a response bias in favor of the rewarded or more frequently reinforced stimulus (e.g., Henriques & Davidson, 2000; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2009; Pizzagalli, Jahn, & O'Shea, 2005). Finally, during anticipation and receipt of rewards, healthy individuals show specific activation patterns in cortical and subcortical regions implicated in the neural reward circuit (e.g., Elliott, Newman, Longe, & Deakin, 2003; Nestler & Carlezon, 2006).

On the other hand, there is a vast literature on reward insensitivity in certain populations, especially in the case of depression (for a review see Eshel & Roiser, 2010). Most of the evidence regarding depressed individuals’ insensitivity to rewards stems from self-report, behavioral, and neuroscientific data that suggest less anticipated and experienced pleasure (e.g., MacPhillamy & Lewinsohn, 1974), no behavioral adaptation (e.g., Henriques & Davidson, 2000), and dysfunction in reward- or approach-related brain areas (e.g., Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Smoski et al., 2009).

Two recent studies on effort mobilization have linked dysphoria (i.e., subclinical depression) to effort-related cardiovascular responding during reward anticipation and have found attenuated cardiovascular reactivity of dysphoric compared to nondysphoric individuals (Brinkmann, Schüpbach, Ancel Joye, & Gendolla, 2009). However, as these studies only compared a reward to a no-reward condition, the question remains open as to whether dysphoric individuals show blunted cardiovascular response across several levels of reward magnitude. The present study thus aimed at expanding previous evidence of a motivational deficit in terms of reward insensitivity in dysphoria: Complementing self-report, behavioral, and neuroscience data we tested dysphoric and nondysphoric individual’s cardiovascular reactivity as an indicator of effort mobilization. Moreover, we compared a no-reward condition to conditions with a small and with a significant monetary reward.
1.2 Reward insensitivity in depression and dysphoria

Since the introduction of the term “anhedonia” by Ribot (1896) to denote a loss of interest or pleasure, evidence for reward insensitivity not only in clinical depression but also in subclinical dysphoria has accumulated. Depressed and dysphoric individuals anticipate and experience less pleasure concerning a variety of activities and hedonic consequences, attribute lower value to rewards, and report weaker approach motivation (e.g., Dickson & MacLeod, 2006; Kasch, Rottenberg, Arnow, & Gotlib, 2002; MacPhillamy & Lewinsohn, 1974). On a behavioral level, depressed and dysphoric individuals fail to develop a response bias toward the rewarded or more frequently reinforced stimulus, fail to choose the options maximizing their winnings, and show deficits in reward-based decision making (e.g., Forbes, Shaw, & Dahl, 2007; Henriques & Davidson, 2000; Kunisato et al., 2012; Liu et al., 2011; Pizzagalli, Iosifescu, et al., 2009; Pizzagalli et al., 2005). Finally, a number of recent studies demonstrated reduced activity in cortical and subcortical components of the neural reward circuit of depressed and dysphoric individuals, both during anticipation and outcome phases of reward processing (e.g., Forbes et al., 2009; Knutson et al., 2008; Pizzagalli, Holmes, et al., 2009; Smoski et al., 2009; Steele, Kumar, & Ebmeier, 2007). Most of these brain imaging studies concerned patients with major depression but also recovered depressed (McCabe, Cowen, & Harmer, 2009) and healthy populations at risk (Gotlib et al., 2010). In a similar vein, there is evidence for electrocortical hypoactivation of left prefrontal areas in depressed and dysphoric individuals—in the resting state but also during reward anticipation (e.g., Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Gotlib, Ranganath, & Rosenfeld, 1998; Harmon-Jones et al., 2002; Shankman, Klein, Tenke, & Bruder, 2007; Thibodeau, Jorgensen, & Kim, 2006; Tomarken & Keener, 1998).

These studies suggest that both clinical depression and subclinical dysphoria are characterized by impairments in approach-related motivation. Such deficits might be more pronounced in the motivational anticipatory phase of reward processing than in the consummatory outcome phase (Sherdell, Waugh, & Gotlib, 2011), which suggests more impairments in reward “wanting” than in reward “liking” (cf. Berridge & Robinson, 2003). However, measures investigating this motivational deficit directly from an effort-mobilization point of view have not been considered yet. We thus do not know whether depressed or dysphoric individuals indeed mobilize less effort than a control group when anticipating a rewarding consequence after successful goal pursuit.
1.3 Effort mobilization and cardiovascular reactivity

Effort mobilization refers to the resources a person is mobilizing at a certain point in time in order to carry out a certain behavior (Gendolla & Wright, 2009). In his integrative model, Wright (1996) proposed that effort mobilization can be operationalized by a person’s cardiovascular response and, more specifically, by cardiovascular parameters that are influenced by the activation of the sympathetic nervous system. These propositions are based on motivational intensity theory (Brehm & Self, 1989) and the active coping approach (Obrist, 1981).

According to motivational intensity theory, reward value determines success importance and thus the maximum effort a person is willing to invest—the more important success is for the individual, the more effort she or he is willing to potentially invest for goal attainment. However, the actual mobilization of effort at a given point in time is supposed to be directly determined by success importance only when there is no clear or predefined performance standard (i.e., an *unclear* or *unfixed* task difficulty in the terminology of motivational intensity theory). In contrast, when there is a clear performance standard (i.e., a *fixed* task difficulty), people are expected to adjust their effort mobilization as a function of perceived task difficulty—as long as effort mobilization is possible and justified by the upper limit of success importance (Brehm & Self, 1989). In the context of the present study, only tasks with an *unclear* performance standard will be considered, that is, conditions where reward value (i.e., success importance) directly determines actual effort mobilization because there is no other information about, for instance, the performance standard.

Wright (1996) proposed that in an active coping context (i.e., when the individual can actively influence the outcome of a situation or task) effort mobilization can be operationalized by assessing cardiovascular parameters that are influenced by sympathetic activation. This is based on Obrist’s (1981) observation that in an active coping context the sympathetic impact on the heart is proportional to task engagement. Wright further specified that systolic blood pressure (SBP) in particular should reliably follow the pattern proposed by motivational intensity theory (Brehm & Self, 1989) because sympathetic activation potentiates myocardial contractility, which, together with peripheral resistance, determines SBP. Diastolic blood pressure (DBP), in contrast, is mainly influenced by vascular resistance and thus not considered a reliable indicator of effort mobilization. Heart rate (HR) is determined by both sympathetic and parasympathetic influences. It can be expected to reflect the hypothesized pattern but only to the extent that sympathetic activation is not masked by parallel increases in parasympathetic activity and that an increase in HR is not caused by a withdrawal of vagal restraint (see Berntson, Cacioppo, & Quigley, 1993; Brownley, Hurwitz,
Past research on motivational intensity theory has indeed found SBP and, less consistently, HR and DBP to follow the predictions (for reviews see Gendolla & Brinkmann, 2005; Gendolla, Brinkmann, & Silvestrini, 2012; Wright & Kirby, 2001).

Recent research in the framework of motivational intensity theory has also used cardiac pre-ejection period (PEP; the time interval from the onset of ventricular depolarization to the onset of ventricular ejection). PEP is determined by myocardial contractility and can thus be considered a reliable and valid indicator of beta-adrenergic sympathetic impact on the heart (see also Kelsey, 2012; Sherwood et al., 1990). These recent studies have corroborated motivational intensity theory’s predictions (e.g., Annis, Wright, & Williams, 2001; Richter, Friedrich, & Gendolla, 2008) and, most importantly, demonstrated the linear increase in PEP reactivity across three levels of monetary reward (Richter & Gendolla, 2009).

1.4 Reduced cardiovascular response to reward in dysphoria

Based on past research on effort mobilization for obtaining rewards (Richter & Gendolla, 2006, 2007, 2009; see also Wright, Killebrew, & Pimpalapure, 2002) and on evidence for reduced reward responsiveness in depression and dysphoria (for a review see Eshel & Roiser, 2010), two recent studies have addressed the question of reduced effort mobilization for obtaining a monetary reward in a subclinical sample of dysphoric students (Brinkmann et al., 2009). The core assumption of these studies—and of the present study—is that dysphoria is associated with an insensitivity to the promised reward, which leads to a lower level of success importance and thus maximally justified effort for the task at hand. Results indeed revealed that nondysphoric participants had higher PEP and SBP reactivity when they could earn 10 Swiss Francs (about 10 USD) after successful task performance compared to a neutral condition without hedonic consequence. In contrast, dysphoric participants showed no increase in cardiovascular reactivity in the reward condition compared to the no-reward condition but a generally low reactivity similar to nondysphoric participants in the no-reward condition. These first two studies lend support to the hypothesis of dysphorics’ reduced effort mobilization for obtaining a reward after successful performance of a cognitive task with unclear task difficulty. However, as these studies only compared a reward to a no-reward condition, they leave open the question whether depressed or dysphoric individuals are not sensitive at all to differences in reward value and thus do not modulate their level of maximally justified effort as a function of reward value.

The present study therefore aims at answering these questions in a subclinical sample of dysphoric and nondysphoric students. We chose a subclinical sample with low versus high
self-reported depression scores based on a dimensional approach of psychopathology (e.g., Beach & Amir, 2003; Ruscio & Ruscio, 2002) and on the assumption that research with analogue, subclinical samples can give important insights in the psychological mechanisms of depression (Vredenburg, Flett, & Krames, 1993). The present study complements behavioral and neuroscientific research on reward insensitivity by looking at dysphoric individuals’ motivational deficit using cardiovascular measures of effort mobilization during the performance of a cognitive task. Moreover, we manipulated monetary reward at three levels—no reward, a small amount of money, and a significant amount of money. Based on previous research on effort mobilization as a function of reward value (Richter & Gendolla, 2009), we hypothesized that nondysphoric participant’s cardiovascular reactivity would linearly increase from the no-reward to the high-reward condition. In contrast, we expected dysphoric participant’s cardiovascular response to be low and independent of reward value (Brinkmann et al., 2009; Eshel & Roiser, 2010). In accordance with Wright’s (1996) integrative model we expected especially PEP reactivity and, to a lesser extent, SBP reactivity to show this pattern. Moreover, we hypothesized that self-reported reward attractiveness and success importance would mirror the cardiovascular response pattern, that is, a linear increase from the no-reward to the high-reward condition for nondysphoric individuals and low evaluations independent of reward value for dysphoric individuals.

2. Method

2.1 Participants and design

This study was run in a 2 (dysphoric vs. nondysphoric) x 3 (0 vs. 5 vs. 15 Swiss Francs) between-persons design. After having obtained approval of the protocol by the appropriate Institutional Review Board of the University of Geneva, we recruited participants from an introductory psychology class of 206 university students who had participated in the questionnaire sessions. Out of this sample, we randomly selected participants who scored in the lower quartile (< 11 for men and women) or the upper quartile (> 16 for men, >19 for women; all above the recommended cut-off score of 16) of the Center for Epidemiologic Studies – Depression Scale (CES-D; Radloff, 1977). One month later, these students were invited via an anonymous code to participate in the experiment in exchange for course credit and were randomly assigned to the three reward conditions. From the 96 participants, data of seven participants could not be used for analyses because of bad signal quality of our primary dependent variable PEP. Moreover, two participants had missing blood pressure data. However, as blood pressure was not our primary dependent variable we kept those participants for all analyses excepting blood pressure. Finally, one participant in the
dysphoric-15-Swiss-Francs cell showed extremely high reactivity scores on all four cardiovascular measures (more than 3 SDs above overall means). We thus excluded this participant, even though this had no impact on the results.

The final sample consisted of 88 students (78 women and 10 men with a mean age of 21.39 years, SD = 4.83). Forty-one participants were located in the upper quartile of the CES-D score distribution (M = 29.61, SD=8.08) and were referred to as dysphoric. Forty-seven participants were situated in the lower quartile of the CES-D (M = 7.34, SD=2.89) and were referred to as nondysphoric. Unfortunately, the minimal male participants were not evenly distributed across the six cells (no man in the nondysphoric-5-Swiss-Francs cell, one man in the dysphoric-5-Swiss-Francs-cell, three men in the nondysphoric-15-Swiss-Francs cell, and two men in the remaining three cells). Given this fact, we repeated all of the analyses described below by considering women only. As the results were virtually the same for all analyses, we report the results for the whole sample without considering participants’ gender. Moreover, we also repeated all of the analyses described below by removing those participants whose CES-D scores at the second measurement time (experimental session) did not stay within the limits defined for recruitment at the first measurement time (questionnaire session). As the results were virtually the same except for SBP baseline, we report the results of the whole sample.

2.2 Cardiovascular measures

Cardiovascular measures were assessed during habituation and task performance and directly transferred to and stored on a computer drive so that both experimenter and participants were ignorant of these values. SBP and DBP (in millimeters of mercury [mmHg]) were measured noninvasively with a Vasotrac® APM205A monitor (MEDWAVE®, St. Paul, MN) that uses applanation tonometry (for a validation study see Belani et al., 1999). A pressure sensor was placed on the wrist on top of the radial artery of the participant’s nondominant arm. The device yields one blood pressure measure every 12 to 15 heart beats (i.e., 4 to 6 measures per minute). HR and PEP were measured continuously and noninvasively with electrocardiogram (ECG) and impedance cardiogram (ICG) signals using a Cardioscreen® 1000 (medis, Ilmenau, Germany) haemodynamic monitoring-system (for a validation study see Scherhag, Kaden, Kentschke, Sueselbeck, & Borggrefe, 2005). Four dual gel-pad sensors (medis-ZTECT™) were placed on each side of the base of the participant’s neck and on each side of the thorax along the middle axillary line at the level of the xiphoid. Data were sampled at 1000 Hz.
2.3 Self-report measures

In order to measure dysphoria, we used the CES-D, a self-report depression scale for community samples. The French version by Fuhrer and Rouillon (1989) consists of 20 items. Participants had to indicate the frequency of depressive symptom occurrence during the past week on 4-point scales from 0 (never, very seldom) to 3 (frequently, always). The total score is calculated by summing all negative and reverse-scored positive items and varies from 0 to 60. CES-D scores at both measurement points (i.e., at the questionnaire session and after participation in the experiment) were correlated, $r(88) = .72, p < .001$, and showed high internal consistency (Cronbach’s $\alpha > .92$).

Participant’s momentary mood was assessed by a French version of the positive and negative hedonic tone scales of the UWIST mood adjective checklist (Matthews, Jones, & Chamberlain, 1990). Participants had to indicate their momentary feeling state by scoring four positive and four negative adjectives on 7-point scales ranging from 1 (not at all) to 7 (very much). A mood index was calculated by summing all positive and reverse-scored negative items, so that higher scores indicate more a positive mood (Cronbach’s $\alpha = .91$).

In order to assess the impact of our reward manipulation and to ensure that reward value was salient (see Richter, 2010), we asked all participants to “answer five questions about the upcoming task” on 7-point scales ranging from 1 (not at all) to 7 (very much). Two questions assessed participants’ perception of success importance (“How important is it for you to succeed in the task?”, “How satisfied will you be after successful task performance?”). Three questions evaluated reward attractiveness (“How attractive does the reward seem to you?”, “How valuable is the reward for you?”,”How interesting is it for you to get the reward?”). The first two questions were positively correlated, $r(83) = .65, p < .001$, and were merged to a sum score of success importance. The latter three questions were also positively inter-correlated, $.74 < r(57) < .85, ps < .001$, and were merged to a sum score of reward attractiveness.$^2$

2.4 Procedure and experimental task

The study was run in individual sessions, which took about 30 minutes each, and were computerized using a personal computer and experimental software (Inquisit 3.0, Millisecond Software, Seattle, WA) for all instructions and stimuli presentation. The experimenter first welcomed the participant, asked her or him to take a seat in front of the computer monitor, to answer some biographical questions, and to sign an informed consent form. Then, the experimenter applied the blood pressure sensor and the gel-pad sensors for ECG and ICG signals. After that, she left the room and monitored the experiment from an outside control
Participants read introductory information and answered questions about their momentary feeling state on the UWIST scale. Then, participants watched a 7.5-min excerpt of a hedonically neutral documentary film, which served as a habituation period to determine cardiovascular baseline measures.

After the habituation period, participants received instructions for a modified version of the Sternberg task (Sternberg, 1966). During each trial, a series of black capital letters appeared one after the other in the center of the screen for a duration of 1 s per letter (e.g., Z, U, D, R, A). At the end of each series, which varied in length, a red capital target letter (e.g., Z) appeared for a maximum of 2 s. Participants had to indicate whether or not this target letter had been part of the series of black letters presented before by pressing one of two keys. If participants had not answered after 2 s, a message asking to answer more quickly appeared for 1 s. If participants had answered during the 2 s, a message confirming answer recording appeared for the remaining time plus 1 s so that each presentation of the target letter and the response time window took exactly 3 s. Accordingly, each trial lasted 6 to 12 s—depending on the length of the series of black letters—and was followed by a 2-s pause. Instructions explained that the length of the series of black letters would vary in a random manner. In order to keep task difficulty unclear, participants did not receive any information about the total time of the task or about the difficulty (i.e., length) of the trials. In fact, 29 trials of varying length (from 3 to 9 black letters) were presented during the 5-min performance period.

After task instructions participants received reward information. They learned that at the end of the experiment they would get to know the performance standard of the task by choosing one out of three cards: Participants had succeeded in the task when they had at least as many correct trials as indicated on the chosen card. This manipulation further helped to keep task difficulty unclear because participants did not know in advance the specific standard that would be used to evaluate their performance. There was no further information for participants in the 0-Swiss-Francs condition. Participants assigned to the 5- and 15-Swiss-Francs conditions received the additional information that, if they succeeded in the task, they would receive 5 or 15 Swiss Francs, respectively.

At this point, the experimenter entered the room to ensure that participants had understood all instructions. For participants in the 5- and 15-Swiss-Francs conditions, she put the money on the table next to the computer monitor to make the reward more concrete and attractive. Next, participants answered the five questions evaluating reward attractiveness and success importance. Then the 5-min performance period started during which cardiovascular activity was assessed. At the end of the task, the individual performance score appeared on the
screen. The experimenter re-entered the room and let participants choose one of the three cards. Depending on the condition, the individual task performance, and the performance standard indicated on the chosen card, the experimenter handed out the promised money. She then removed the blood pressure sensor and the electrodes and asked participants to complete the CES-D in an adjacent room, ostensibly for an unrelated questionnaire validation study. Finally, participants were debriefed, thanked, and given their course credit.

2.5 Data scoring and analysis

Heart rate (in beats per min [bpm]) was determined by means of a LabVIEW-based software (National Instruments, Austin, TX) developed in our laboratory (Richter, 2009) that detects and counts R-peaks in the ECG signal. Data were also visually inspected and edited for ectopic heart beats. For PEP measures (in ms), the ICG dZ/dt signal (first derivative) was ensemble-averaged over 60-s time intervals. Only valid cycles were included in ensemble averages. The ECG R-onset and the ICG B-point were automatically detected by the same software, visually inspected by two independent raters, and modified if necessary (see Sherwood et al., 1990). PEP was then calculated as the interval between ECG R-onset and ICG B-point (Berntson, Lozano, Chen, & Cacioppo, 2004). As inter-rater reliability was very high, ICC(2,1) = .98 (Shrout & Fleiss, 1979), the averaged PEP values from both raters were used for analyses.

Cardiovascular baseline scores were determined by averaging the last 4 min of the habituation period (Cronbach’s αs > .98). Task scores were determined by averaging the 5 min of the performance period. We then calculated cardiovascular reactivity scores by subtracting baseline scores from task scores (Cronbach’s αs > .94) (see Kelsey, Ornduff, & Alpert, 2007; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991).

Task performance measures consisted of the number of correctly and incorrectly answered trials on the Sternberg task. Additionally, we calculated sensitivity and response bias on the basis of signal detection theory. Following the recommendations of Snodgrass and Corwin (1988), sensitivity was calculated as the difference of corrected hit rate minus corrected false alarm rate. Response bias was calculated as false alarm rate divided by 1 minus sensitivity.

We submitted cardiovascular baseline, self-report, and performance measures to 2 (dysphoric vs. nondysphoric) x 3 (0 vs. 5 vs. 15 Swiss Francs) between-persons ANOVAs. For the specific analysis of SBP, DBP, HR, and PEP reactivity during task performance and of self-reported reward attractiveness, we calculated a priori contrasts, the most appropriate test for our predicted pattern (see Rosenthal & Rosnow, 1985). Contrast weights were -1 for
dysphorics in all three conditions and for nondysphorics in the 0-Swiss-Francs condition, +1 for nondysphorics in the 5-Swiss-Francs condition, and +3 for nondysphorics in the 15-Swiss-Francs condition. These contrast weights allowed us to simultaneously test the expected linear increase in the nondysphoric group as well as the hypothesized blunted response across all three reward conditions in the dysphoric group. As suggested by an anonymous reviewer, we conducted additional contrasts to further explore whether there were any differences among the conditions. These tests involved a set of three Helmert contrasts to test for differences between the four conditions that should show low reactivity as well as a final contrast comparing the nondysphoric/5-Swiss-Francs and the nondysphoric/15-Swiss-Francs conditions.

3. Results
3.1 Cardiovascular baselines

Means and standard errors of cardiovascular baseline values can be found in Table 1. Results of 2 (dysphoria) x 3 (reward) ANOVAs revealed no significant main or interaction effects on PEP, HR, and DBP baseline measures, $F$s < 2.79, $p$s > .09. For SBP there was a significant dysphoria x reward interaction, $F(2, 80) = 3.13$, $p = .05$, $\eta^2_p = .07$, in absence of significant main effects, $F$s < 1.81, $p$s > .17.

3.2 Cardiovascular reactivity
3.2.1 PEP reactivity

The a priori contrast specified above proved to be reliable for PEP reactivity, $F(1, 82) = 13.88$, $p < .001$, $\eta^2_p = .14$. As can be seen in Figure 1, PEP reactivity of nondysphoric participants increased from the 0-Swiss-Francs to the 5-Swiss-Francs and to the 15-Swiss-Francs conditions. In contrast, dysphoric participants’ PEP reactivity did not. This corroborates our main hypothesis that nondysphoric participants would show increasing PEP reactivity with increasing reward value, whereas dysphoric participants’ reactivity would be rather low across all reward levels. The additional contrasts revealed no significant differences among the three dysphoria and the nondysphoric/0-Swiss-Francs conditions, $F$s < 3.19, $p$s > .07. As expected, PEP reactivity of nondysphoric participants in the 5-Swiss-Francs and 15-Swiss-Francs conditions differed significantly, $F(1, 82) = 6.10$, $p = .02$, $\eta^2_p = .07$ (see Table 2).
3.2.2 HR reactivity

Analyses of HR reactivity also showed the significant a priori contrast, $F(1, 82) = 7.65, p = .01, \eta^2_p = .09$. The pattern of results mirrored that of PEP reactivity (see Figure 2) and thus lends further support to our hypothesis. HR reactivity of nondysphoric participants increased from the 0-Swiss-Francs to the 5-Swiss-Francs and to the 15-Swiss-Francs conditions. In contrast, dysphoric participants’ HR reactivity was rather low. The additional contrasts revealed a significant difference between dysphoric participants’ reactivity in the 5-Swiss-Francs and the 15-Swiss-Francs condition, $F(1, 82) = 4.10, p = .05, \eta^2_p = .05$. All other contrasts were not reliable, $F_s < 2.03, p_s > .15$ (see Table 2).

3.2.3 Blood pressure reactivity

Our specified a priori contrast was, however, not reliable for SBP and DBP reactivity, $F(1, 80) = 1.19, p = .28$, and $F(1, 80) = 0.63, p = .43$, respectively. As can be seen from the means and standard errors in Table 2, SBP reactivity did not mirror the pattern of PEP reactivity. The 0- and 5-Swiss-Francs conditions roughly depicted the expected pattern with nondysphoric participants in the 5-Swiss-Francs condition having slightly higher SBP reactivity than dysphoric participants and nondysphoric participants without reward. However, SBP reactivity in the 15-Swiss-Francs condition did not show the expected pattern. The additional contrasts revealed a significant difference between dysphoric participants’ DBP reactivity in the 5-Swiss-Francs and 15-Swiss-Francs conditions, $F(1, 80) = 5.12, p = .03, \eta^2_p = .06$. All other contrasts for SBP and DBP were not reliable, $F_s < 2.02, p_s > .16$.

3.3 Self-report and task performance

3.3.1 UWIST mood measure

A 2 (dysphoria) x 3 (reward) ANOVA of the UWIST scale revealed the expected significant dysphoria main effect, $F(1, 74) = 33.63, p < .001, \eta^2_p = .31$, in absence of other main or interaction effects, $F_s < 1.58, p_s > .21$. This result confirms that the dysphoric students ($M = 37.81, SE = 1.42$) who had been selected by means of their CES-D scores from the questionnaire session were indeed in a less positive mood when participating in the experiment than the nondysphoric group ($M = 46.89, SE = 0.82$). Participants’ mood did not significantly correlate with cardiovascular reactivity, $r_s < |.15|, p_s > .20$.

3.3.2 Success importance and reward attractiveness

Means and standard errors of success importance and reward attractiveness are presented in Table 3. A 2 (dysphoria) x 3 (reward) ANOVA of success importance revealed a
significant reward main effect, \( F(2, 77) = 4.79, p = .01, \eta^2_p = .11 \), in absence of other main or interaction effects, \( Fs < 3.01, ps > .08 \). A 2 (dysphoria) x 2 (5 versus 15 Swiss Francs) ANOVA of reward attractiveness also revealed a significant reward main effect, \( F(1, 53) = 8.64, p = .01, \eta^2_p = .14 \), in absence of other main or interaction effects, \( Fs < 1, ps > .51 \). The two a priori contrasts were not significant, \( Fs < 2.46, ps > .12 \). Contrast weights for success importance were identical to cardiovascular reactivity analyses. Contrast weights for reward attractiveness were 0 for the two 0-Swiss-Francs conditions, -1 for the remaining two dysphoria conditions, +0.5 for nondysphorics in the 5-Swiss-Francs condition, and +1.5 for nondysphorics in the 15-Swiss-Francs condition. Both indices did not significantly correlate with cardiovascular reactivity, \( rs < |.17|, ps > .21 \).

3.3.3 Task performance

Finally, we analyzed the sensitivity and response bias indices as well as global reaction time and number of correctly and incorrectly answered trials by means of 2 (dysphoria) x 3 (reward) ANOVAs. Results revealed no significant main or interaction effects on any of the performance measures, \( Fs < 2.29, ps > .10 \). Performance scores did not significantly correlate with cardiovascular reactivity either, \( rs < |.15|, ps > .19 \). Overall means and standard errors were \( M = 0.56 \) and \( SE = 0.02 \) for the sensitivity index, \( M = 0.20 \) and \( SE = 0.01 \) for the response bias index, \( M = 1074.30 \) and \( SE = 17.55 \) for global reaction time in milliseconds, \( M = 22.71 \) and \( SE = 0.28 \) for the number of correctly answered trials, and \( M = 5.28 \) and \( SE = 0.26 \) for the number of incorrectly answered trials.

4. Discussion

The aim of the present study was twofold. First, we aimed at expanding previous evidence on reward insensitivity in depressed and dysphoric individuals. By using cardiovascular measures—especially PEP—as indicators of effort mobilization in an active coping context (Kelsey, 2012; Wright, 1996), we sought to show that dysphoric individuals indeed mobilize less effort than nondysphoric individuals when anticipating a monetary gain. Second, as previous research on cardiovascular reactivity has manipulated only the presence versus absence of reward (Brinkmann et al., 2009), we aimed at further testing dysphorics’ motivational deficit in the framework of motivational intensity theory (Brehm & Self, 1989). We thus sought to demonstrate that dysphoric individuals show reward insensitivity across several levels of reward value.

Results corroborate our main hypothesis. PEP reactivity, our primary measure of beta-adrenergic sympathetic impact on the heart, showed the expected pattern. There was an
increase in PEP reactivity from the 0- to the 5- and 15-Swiss-Francs conditions for nondysphoric participants, whereas dysphoric participants’ reactivity was rather low and similar in all three conditions. The same pattern emerged for HR reactivity, which corresponds to earlier research that has also found HR effects (e.g., Fowles et al., 1982; Richter & Gendolla, 2007; Wright et al., 2002). However, SBP and DBP reactivity did not mirror the reactivity of these cardiac parameters. As DBP is strongly influenced by peripheral resistance, the absence of a DBP effect is not surprising, and past research in the context of motivational intensity theory (Brehm & Self, 1989) has not consistently found DBP effects. The absence of an effect on SBP is more surprising but not problematic. SBP has very often been shown to correspond to predictions derived from motivational intensity theory (for reviews see Gendolla & Brinkmann, 2005; Gendolla et al., 2012; Wright & Kirby, 2001). However, there are also several important exceptions that found evidence for motivational intensity theory’s predictions on cardiac measures but not on SBP (e.g., Annis et al., 2001; Freydefont, Gendolla, & Silvestrini, 2012; Richter, 2010).

It is important to note that the effects on PEP reactivity may be driven not only by beta-adrenergic sympathetic impact on the myocardium but also by cardiac preload (i.e., left ventricular filling) or cardiac afterload (i.e., aortic diastolic pressure). Therefore, Obrist, Light, James, and Strogatz (1987) pointed out that a shortening of PEP should only be interpreted in the presence of accompanying stable or increasing HR and DBP, as rough approximations for preload and afterload, respectively (see also Sherwood et al., 1990). As can been seen from the descriptive data, cells with strong shortening of PEP (e.g., the nondysphoric-15-Swiss-Francs cell) are also characterized by increases in HR and DBP. Without beta-adrenergic sympathetic activation of the heart, such increases in HR and DBP should have been associated with a lengthening of PEP. Thus, based on the accompanying increases in HR and DBP in the cells with strong shortening of PEP, we conclude that PEP reactivity in our study reflects changes in sympathetically driven myocardial contractility.

Both the results of self-reported depressive symptoms on the CES-D scale several weeks before and directly after the experimental session as well as the results of self-reported momentary mood on the UWIST scale at the beginning of the experimental session confirmed that our dysphoric and nondysphoric participants clearly differed with respect to momentary and dispositional mood. We are thus confident that the cardiovascular pattern of reward responsiveness reflects a rather stable disposition to be generally more or less attracted by hedonic incentives and, as a consequence, to mobilize more or less effort in order to attain them.
Self-reported reward attractiveness and success importance did not show the expected effects of increasing attractiveness and importance in nondysphorics and low attractiveness and importance in dysphorics. In this context, it should be noted that the five questions did not stem from an established questionnaire. It is thus conceivable that we were not successful in creating these questions for measuring reward attractiveness and success importance. Moreover, the programming error (see Footnote 2) reduces the validity of participants’ responses in the 0-Swiss-Francs condition. On the other hand, it should be kept in mind that self-report questions are susceptible to self-presentation tendencies (Pyszczynski & Greenberg, 1983; Rhodewalt & Fairfield, 1991) and that we cannot exclude the possibility that participants were reluctant to admit their true evaluation of reward attractiveness and success importance.

Concerning performance outcomes, the different indices derived from the modified Sternberg task were not influenced by the dysphoria groups or the reward manipulation and they did not correlate with the cardiovascular indices of effort mobilization. Here again, two things have to be considered. First, the task had been modified and adapted to our experiment with the primary purpose of creating a cognitive task with a fixed difficulty standard that was, however, unknown to the participants. It was not the main focus of the experiment to select a task that is sensitive to performance effects. Second, it is of note that effort mobilization and performance measures can, but do not necessarily have to be positively associated because performance is not only determined by effort but also by task-related ability and chosen strategies (Locke & Latham, 1990). Moreover, task performance can only reflect the effectiveness of behavior but not its efficiency (Eysenck, Derakshan, Santos, & Calvo, 2007), and effort can have a compensatory function in order to maintain performance (Hockey, 1997). Therefore, effects on effort mobilization in the absence of accompanying effects on performance measures are quite possible.

Taken together, the present study demonstrates reduced cardiac—PEP and HR—responding across three levels of monetary reward in subclinical dysphoria compared to a nondysphoric control group. These results complement previous evidence for reduced self-reported, behavioral, and neural reward responsiveness in depression and dysphoria and underline the importance of motivational deficits in reward salience in these populations. Our experimental procedure allowed the assessment of effort mobilization in the expectation of three different consequences. Therefore, the present study is exclusively concerned with the motivational anticipatory phase of reward processing—in contrast to the emotional outcome phase—and demonstrates attenuated reward “wanting” in dysphoria.
From a neuroscience perspective, there is evidence that clinically depressed, recovered depressed, and individuals at risk for depression show abnormal neural functioning during both reward anticipation and outcome phases (e.g., Forbes et al., 2009; Gotlib et al., 2010; Knutson et al., 2008; McCabe et al., 2009; Pizzagalli, Holmes, et al., 2009; Smoski et al., 2009; Steele et al., 2007). However, reward anticipation in relation with differences in incentive salience and dopamine functioning seems to be of particular importance (Berridge, 2007; Dichter, 2010; Nestler & Carlezon, 2006; Sherdell et al., 2011). From a therapeutic point of view, the disentangling of motivational and consummatory components and the understanding of the different mechanisms involved has important implications. For instance, promising psychotherapeutic and pharmacological treatments for motivational symptoms of anhedonia include behavioral activation therapy and dopamine-active pharmacotherapies (Treadway & Zald, 2011).

Finally, the question of reward insensitivity across several levels of reward value in depression and dysphoria deserves further discussion. For instance, it is possible that 5 Swiss Francs are not sufficiently different from the condition without reward to elicit an effect in dysphoric individuals but larger amounts might be. In this respect, it is of note that 15 Swiss Francs do not represent an extremely high but nevertheless considerable amount of money for the undergraduate participants of this study. Past research has often used rather small amounts of money on a trial-by-trial basis that can accumulate to a moderate gain (e.g., Pizzagalli, Iosifescu, et al., 2009), whereas the monetary reward in the present study was delivered on an all-or-nothing basis. Based on the findings of the present study and of previous research, we cannot affirm that depressed individuals are characterized by a general insensitivity to hedonic contingencies. It remains conceivable that they just have a higher threshold for positive responding to incentives. Therefore, future research needs to further expand the range of possible positive and negative incentives. Moreover, future research should address the effects of other primary (e.g., food) or social rewards (Forbes, 2009) and of negative consequences. An enhanced understanding of the mechanisms and processes involved in dysphoric and depressed individuals’ responding to hedonic consequences has important implications for both pharmacological and psychotherapeutic treatments. For instance, hedonic consequences might be individually tailored to motivate patients to mobilize effort.

Following a dimensional conception of psychopathology, our study was based on subclinical participants with high self-reported depression scores. Previous research on reward insensitivity has often found similar results in clinical and subclinical samples (e.g., Liu et al., 2011). Therefore, we are confident that the present findings can be generalized to patients with major depression. Nevertheless, future research needs to expand the present
results to a clinical sample. Such research should also take into account the fact that antidepressant medication might restore reward sensitivity in patients with major depression (e.g., Stoy et al., 2012). A second limitation of our sample concerns the limited number of male participants. To the best of our knowledge, none of the behavioral or neuroimaging studies on reward insensitivity in dysphoria or depression has reported gender differences. Therefore, we think that there is no reason to believe that the specific pattern of cardiovascular reactivity in our study would be significantly different for men. Nevertheless, future research needs to confirm that the present results also apply to men. A third limitation pertains to the fact that we did not assess health variables (e.g., smoking status, physical activity) that exert an impact on the cardiovascular system and that might have differed between the two dysphoria groups. Therefore, future research should assess and control for those potentially confounding health variables. Finally, as mentioned above, future research needs to expand the range of positive and negative incentives.

In conclusion, the present study contributes to the understanding of reward insensitivity in dysphoria and depression. It demonstrates that dysphoria is characterized by reduced cardiac reactivity during the performance of a cognitive task with an unclear performance standard. These results suggest that dysphoric individuals mobilize less effort than nondysphoric individuals when anticipating either a small or a significant amount of money after successful task performance. Dysphoria is thus characterized by a motivational deficit during the anticipatory phase of reward processing. The present study underlines the importance of considering reward salience and effort mobilization for both the understanding and treatment of reward insensitivity in depression and dysphoria.
References


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Footnotes

1 In the literature, the term dysphoria has been used not only to denote an unhappy, tense, and irritated mood (Musalek, Griengl, Hobl, Sachs, & Zoghlami, 2000; Starcevic, 2007) but also to refer to subclinical populations with elevated depression scores not meeting diagnostic criteria for major depression (see Kendall, Hollon, Beck, Hammen, & Ingram, 1987). In line with most of the research reviewed in this article, we are using the term dysphoria in this latter sense. The reported findings have also been obtained for clinical and subclinical populations. We have highlighted whenever they apply only to one or the other group.

2 Due to a programming error, participants in the 0-Swiss-Francs condition answered not only the two questions assessing success importance but also the three questions related to reward attractiveness. However, none of the participants in the 0-Swiss-Francs condition reported being confused about these inapplicable questions. Therefore, we suspect that they did not excessively reflect on them but referred to the promised course credit as the “reward”. We are thus confident that this programming error did not have an undue impact on the experiment as such. For data analyses, we removed the answers that participants in the 0-Swiss-Francs condition gave to the three reward attractiveness questions and analyzed this index in the reduced 2 (dysphoria) x 2 (5 versus 15 Swiss Francs) factorial design.

3 There was an unfortunate loss of the first participants’ data recorded by the experimental software. Therefore, self-report and performance measures are based on data of 80 and 83 participants, respectively.
Table 1

Means, Standard Errors, and Cell Sizes of Cardiovascular Baselines

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
<th>HR</th>
<th>PEP</th>
<th>SBP</th>
<th>DBP</th>
<th>HR</th>
<th>PEP</th>
<th>SBP</th>
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<td>2.62</td>
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</tr>
</tbody>
</table>

Note. SFr = Swiss Francs. SBP and DBP are indicated in millimeters of mercury, HR is indicated in beats per minute, and PEP is indicated in milliseconds.
Table 2

*Means and Standard Errors of Cardiovascular Reactivity*

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<td>2.01</td>
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<td>3.73</td>
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</table>

Note. SFr = Swiss Francs. SBP and DBP are indicated in millimeters of mercury, HR is indicated in beats per minute, and PEP is indicated in milliseconds. For cell sizes see Table 1.
Table 3

Means and Standard Errors of Success Importance and Reward Attractiveness Ratings

<table>
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<th>N</th>
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<td>SI</td>
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<tr>
<td>15 SFr</td>
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<td>11.58</td>
<td>0.52</td>
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</table>

Note. SFr = Swiss Francs. SI = success importance (scores from 2 to 14). RA = reward attractiveness (scores from 3 to 21).
Figure 1. Means and standard errors of cardiac pre-ejection period reactivity in milliseconds.
Figure 2. Means and standard errors of heart rate reactivity in beats per minute.