



# Anhedonia and effort mobilization in dysphoria: Reduced cardiovascular response to reward and punishment<sup>☆</sup>

Kerstin Brinkmann<sup>\*</sup>, Laurent Schüpbach, Isabelle Ancel Joye, Guido H.E. Gendolla

University of Geneva, Switzerland

## ARTICLE INFO

### Article history:

Received 21 July 2009

Received in revised form 1 September 2009

Accepted 18 September 2009

Available online 4 October 2009

### Keywords:

Depression

Anhedonia

Reward

Punishment

Effort mobilization

Cardiovascular reactivity

## ABSTRACT

Instigated by evidence for reduced responsiveness to reward in depression, the present two studies addressed the question if such anhedonic behavior would also become evident in reduced mobilization of mental effort in terms of cardiovascular reactivity. Undergraduates completed the Center for Epidemiologic Studies-Depression Scale (CES-D) and worked on mental tasks, expecting either no consequence, a performance-contingent reward, or a performance-contingent punishment. Study 1 revealed that participants with low CES-D scores showed high systolic blood pressure reactivity in the punishment condition, whereas participants with high CES-D scores showed low systolic reactivity. Study 2 corroborated this finding for reward: Nondysphoric participants expecting a reward showed higher reactivity of systolic blood pressure and pre-ejection period than participants in the neutral condition or than dysphoric participants. Together, the studies demonstrate that reward insensitivity in (subclinical) depression is also found in cardiovascular reactivity. Furthermore, dysphoric individuals do not respond to punishment either, suggesting a general insensitivity to hedonic consequences.

© 2009 Elsevier B.V. All rights reserved.

## 1. Introduction

Depression has commonly been associated with motivational deficits and anhedonia, which is the inability to experience pleasure. One of the core symptoms of a major depressive episode is the loss of interest and pleasure in almost all activities enjoyed before (Diagnostic and Statistical Manual [DSM-IV]; *American Psychiatric Association, 1994*). This also relates to depressed individuals' lack of responsiveness to rewards. A loss of reinforcer effectiveness in depression has already been described by *Costello (1972)*, and *Meehl (1975)* states that "some persons [are] born with more cerebral 'joy-juice' than others" (p. 299). *Strauman (2002)* describes depression as a loss of the motivation to respond to rewarding stimuli, which manifests itself on the neural, cognitive, and behavioral level. Thus, depressed individuals' cognition and behavior seem to be guided by the core conviction that striving for reward and avoiding punishment are futile (*Fowles, 1994*). This phenomenon of insensitivity to rewards in depression has inspired

intense research, from self-report and behavioral to neurophysiological and neurobiological studies.

### 1.2. Reward and punishment responsiveness in depression

With respect to self-report studies, early research on the basis of "expectancy  $\times$  value" theories of motivation came to the conclusion that depressed individuals have reduced reward motivation and in part lower punishment motivation (*Layne et al., 1982, 1983*). The authors suggest that depressed individuals do not care about any outcome, either positive or negative. Similarly, previous research showed that clinical depression but also negative mood are associated with less anticipated and obtained pleasure concerning a variety of activities (*Carson and Adams, 1980; MacPhillamy and Lewinsohn, 1974*) and less engagement in pleasant, rewarding activities (*Lewinsohn and Graf, 1973*). More recent studies based on *Gray's (e.g., 1982, 1990)* theory of a behavioral approach system (BAS) and a behavioral inhibition system (BIS) converge on the fact that depressed individuals report reduced BAS activation and increased BIS activation (e.g., *Kasch et al., 2002*; but see also *Johnson et al., 2003*).

On the neurophysiological level, activation differences in prefrontal cortical areas have received particular attention. Those areas are associated with the representation and maintenance of goals as well as with approach and withdrawal behaviors. A substantial number of studies confirm that depression is related to a relative hypoactivation in left frontal regions, compared to the activation pattern found under normal conditions (e.g., *Davidson and Henriques, 2000; Davidson et al., 2002*; see also *Harmon-Jones et al., 2002; Tomarken and Keener, 1998*). This

<sup>☆</sup> Kerstin Brinkmann, Laurent Schüpbach, Isabelle Ancel Joye, and Guido H. E. Gendolla, Geneva Motivation Lab, University of Geneva, Switzerland. This paper is based on parts of the diploma thesis by Isabelle Ancel Joye. Parts of the present research were presented at the 47th Annual Meeting of the Society for Psychophysiological Research, Savannah, Georgia, October 17–21, 2007, at the 20th Annual Convention of the Association for Psychological Science, Chicago, Illinois, May 22–25, 2008, and at the 29th International Congress of Psychology, Berlin, Germany, July 20–25, 2008.

<sup>\*</sup> Corresponding author. Geneva Motivation Lab, University of Geneva, FPSE, Department of Psychology, 40, Bd. du Pont d'Arve, CH-1211 Geneva 4, Switzerland. Tel.: +41 22 379 92 33; fax: +41 22 379 92 19.

E-mail address: [kerstin.brinkmann@unige.ch](mailto:kerstin.brinkmann@unige.ch) (K. Brinkmann).

evidence is complemented by studies on subcortical structures that are involved in the reward circuit and in the regulation of reward-related behaviors. They show that symptoms of depression such as impaired motivation and reduced reward responsiveness are associated with alterations in mesolimbic dopaminergic areas (e.g., Nestler and Carlezon, 2006). Moreover, recent evidence from event-related potentials has shown that depression is associated with reduced feedback negativity to non-rewards versus rewards (Foti and Hajcak, 2009). On the basis of their results the authors conclude that feedback in general (be it for monetary gains or losses) is less salient to depressed individuals.

Finally, two behavioral studies demonstrated that subclinical (Henriques et al., 1994) as well as clinical depression (Henriques and Davidson, 2000) is related to reduced responsiveness to rewards. The authors analyzed participants' response bias in a signal detection task and found that in the monetary reward condition depressed participants failed to adopt a more liberal response bias—which would maximize their earnings. Results under monetary punishment conditions were ambiguous across the two studies, but suggest that purely depressed individuals are not motivated by punishment. In contrast, subclinical participants and depressed patients with concomitant anxiety were sensitive to signs of punishment and showed a more liberal response bias, maximizing their earnings. Reduced reward responsiveness even proved to be predictive of melancholic symptoms one month later in a nonclinical sample (Pizzagalli et al., 2005).

Taken together, the findings from self-report, behavioral, neurophysiological, and neurobiological studies—investigating clinical as well as subclinical samples—strongly suggest that depressed individuals underestimate rewarding outcomes, do not behaviorally respond to rewards, and have altered cortical and subcortical brain activation and neurotransmission in the respective areas. However, it remains open if this motivational deficit causes depressed individuals to mobilize less effort when they are confronted with challenges that promise rewarding or punishing consequences. Given the reduced responsiveness to reward and punishment reported above, it seems very probable that such anhedonic behavior is also evident in measures of effort mobilization. We therefore hypothesized that clinically depressed but also subclinical (i.e., dysphoric) individuals would be reluctant to mobilize effort in order to obtain a performance-contingent reward or to avoid a performance-contingent punishment.

### 1.3. Effort mobilization and cardiovascular reactivity

To test our hypotheses about effort mobilization, we revert to the concept of effort intensity, which can be defined as the mobilization of resources at a certain point in time in order to carry out a behavior (Brehm and Self, 1989). According to the work of Obrist (1981) and Wright (1996), effort intensity or task engagement can reliably be quantified as the reactivity of the cardiovascular system in the context of task performance. This corresponds in particular to the reactivity of parameters that are systematically influenced by the activation of the sympathetic nervous system (see Brownley et al., 2000; Levick, 2003; Papillo and Shapiro, 1990). The operationalization of effort intensity by a person's cardiovascular response—and especially by systolic blood pressure (SBP) reactivity—has been corroborated by a body of research involving different kinds of mental tasks and different task contexts (for reviews see Gendolla and Brinkmann, 2005; Gendolla et al., 2007; Wright and Kirby, 2001).

The theoretical framework of motivational intensity theory (Brehm and Self, 1989) offers predictions for the influence of a performance-contingent incentive. According to this approach, rewards can have either an *indirect* impact or a *direct* impact on effort intensity. For tasks with fixed difficulty, that is, when people have clear information about an upcoming challenge, rewards are supposed to have an *indirect* impact on effort intensity: Effort intensity is expected to directly vary with task difficulty as long as effort investment seems possible and is justified by success importance. This means that rewards do not influence actual

effort intensity but the maximum people are willing to invest (see Brehm and Self, 1989; Wright, 1996, for details). However, rewards are supposed to have a *direct* impact on effort intensity for tasks with unfixed difficulty (i.e., tasks without performance standard but the instruction to do one's best) and for tasks with unclear difficulty (i.e., when people are ignorant of the existing fixed performance standard). Under these two conditions people are expected to mobilize the maximum effort that seems justified, either because there is no standard or because they lack information about task difficulty that could be used in order to adjust effort mobilization. Therefore, motivational intensity theory predicts that task engagement or effort intensity varies with success importance (e.g., with an incentive value in form of a reward) for tasks with unfixed or unclear difficulty (see Brehm and Self, 1989; Wright, 1996, for details).

Using tasks with unclear difficulty, recent work of Richter and Gendolla (2006, 2007, 2009) has shown that people indeed mobilize more effort in terms of cardiovascular reactivity during task performance when they are offered a reward for successful performance. In their experiments, the authors ensured that participants were not aware of the underlying predefined performance standard of the mental task in order to keep task difficulty unclear. Specifically, participants were instructed to memorize a list of senseless letter strings. However, these letter strings appeared only successively on the computer screen, leaving it open until the end as to how many letter strings were to be memorized and what standard was to be attained. These experiments showed that participants in the monetary and nonmonetary reward conditions had higher reactivity of SBP and in part also of diastolic blood pressure (DBP) and heart rate (HR) (Richter and Gendolla, 2006). Moreover, the authors could show a linear increase in the reactivity of SBP, HR, and cardiac pre-ejection period (PEP) over several reward conditions with increasing monetary incentive (Richter and Gendolla, 2007, 2009).

### 1.4. The present studies

Given the longstanding notion of and the empirical evidence for depressives' insensitivity to reward and in part also to punishment, our studies aimed at testing whether this anhedonic behavior is also evident in reduced effort mobilization. As in prior research, we assessed participants' cardiovascular response to mental tasks with *unclear task difficulty* (see Richter and Gendolla, 2006, 2007). We chose to test our hypotheses in subclinical samples of university students with varying levels of self-reported depression on the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977).

We hypothesized that participants with low CES-D scores would show the "normal" increase in cardiovascular reactivity if they were promised a performance-contingent reward or if they could avoid a performance-contingent loss. We expected furthermore that participants with high CES-D scores would show no increase in cardiovascular reactivity in the reward and punishment conditions and rather have a cardiovascular response similar to a neutral condition without explicit hedonic consequences. Given the body of previous evidence for SBP reactivity as a reliable indicator for effort intensity, our primary variable of interest was participants' SBP response. Recently, also PEP reactivity has been shown to correspond to the predictions of motivational intensity theory (Annis et al., 2001; Richter et al., 2008; Richter and Gendolla, 2009). Therefore, we also assessed PEP as well as DBP and HR, which have been found to correspond to the SBP pattern in several studies (for reviews see Gendolla et al., 2007; Wright and Kirby, 2001).

## 2. Study 1

The first study was a quasi-experiment with the CES-D score as continuous predictor variable and three between-person conditions (hedonic consequences: neutral vs. reward vs. punishment). We predicted an interaction between depression score and condition. Specifically, we expected no association between the depression score

متن کامل مقاله

دریافت فوری ←

**ISI**Articles

مرجع مقالات تخصصی ایران

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان دانلود رایگان ۲ صفحه اول هر مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات