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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.

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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

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dysphoria[1] 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, 2012), 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, & Schneiderman, 2000; Levick, 2003; Papillo & Shapiro, 1990). 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

[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 been obtained for clinical and subclinical populations. We have highlighted whenever they apply only to one or the other group.

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