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Dysphoria is associated with reduced cardiac vagal withdrawal during the imagery of pleasant scripts: Evidence for the positive attenuation hypothesis

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ABSTRACT

The present study investigated emotional responding in dysphoric individuals. Dysphoric ($N = 25$) and nondysphoric ($N = 29$) individuals completed an emotional imagery task, including pleasant, neutral and unpleasant emotional-eliciting scripts. Self-reported valence and arousal, and measures of cardiac autonomic activity were collected during the task. Compared to nondysphoric controls, dysphoric individuals showed a reduced heart rate increase to pleasant scripts. Less vagal withdrawal in response to pleasant scripts was also found in dysphoric, but not in nondysphoric, individuals. Conversely, no differences between groups in autonomic responding to unpleasant scripts and in subjective measures were noted. Overall, our data showed that dysphoria is characterized by blunted cardiac autonomic reactivity in response to positive rather than negative emotional stimuli. The present findings also suggest that the lack of vagal suppression may reflect a reduced sensitivity to positive environmental stimuli, which, in turn, has been implicated in the development of major depression in dysphoric individuals.

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1. Introduction

1.1. Emotional responding and depressed mood

Depression is conceptualized as a disorder of emotion (e.g., Clark & Watson, 1991; Gross & Muñoz, 1995; Tomarken & Keener, 1998). The key symptoms of depression are a loss of interest or pleasure in daily activities (i.e., anhedonia) and/or an excessive and persistent negative mood and emotions (e.g., sadness), suggesting that impaired emotional responding plays a crucial role in the development and/or the maintenance of depressive symptoms (e.g., Rottenberg, Gross, & Gotlib, 2005). Although the constructs that underlie mood – diffuse feeling states that are not necessarily elicited by specific events or objects (Watson, 2000) – and emotions – action dispositions that prepare the organism to respond adaptively to meaningful stimuli (Frijda, Kuipers, & ter Schure, 1989; Lang, 1995) – are distinguishable, they have been typically considered as mutually interrelated (e.g., Rosenberg, 1998). Accordingly, a growing number of studies have examined how depressed mood affects emotional responding. As mood is likely to potentiate like-valenced emotions, persistent and/or excessive negative mood has been hypothesized to potentiate reactivity to unpleasant emotions (i.e., the negative potentiation hypothesis), on the one hand, and to attenuate reactivity to pleasant emotions (i.e., the positive attenuation hypothesis), on the other hand (for a review, see Bylsma, Morris, & Rottenberg, 2008).

Consistent with the positive attenuation hypothesis, it has been reported that clinically depressed individuals (i.e., with major depressive disorder, MDD) evaluate pleasant stimuli as less pleasant compared to healthy individuals in the absence of difference for unpleasant stimuli (e.g., Berenbaum & Oltmanns, 1992; Dunn, Dalgleish, Lawrence, Cusack, & Ogilvie, 2004; Sloan, Strauss, & Wisner, 2001). At the behavioral level there is evidence that facial reactivity in response to pleasant, but not to unpleasant, stimuli is reduced in clinically depressed individuals compared to healthy controls (e.g., Berenbaum & Oltmanns, 1992; Sloan et al., 2001).

Alternatively, the negative potentiation hypothesis postulates that depressed mood is associated with increased reactivity to unpleasant emotions. In line with this hypothesis, it has been found that, compared to healthy controls, depressed individuals are characterized by great electrodermal reactivity in response to a

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negative scenario (Sigmon & Nelson-Gray, 1992). Similarly, studies examining emotional reactivity in undergraduates scoring high on depression measures (i.e., with dysphoria) have reported greater skin conductance during the presentation of an aversive stimulus (Lewinsohn, Lobitz, & Wilson, 1973) or in response to a negative feedback about personality characteristics (Golin, Hartman, Klatt, Munz, & Wolfgang, 1977). There is also converging evidence that undergraduates with vs. without dysphoria exhibit greater affective potentiation of the eyeblink startle reflex. That is, the eyeblink startle reflex is greater during the viewing of unpleasant stimuli in participants scoring high relative to low on self-report depression measure (e.g., Cook, Davis, Hawk, & Spence, 1992; Cook, Hawk, Davis, & Stevenson, 1991). However, studies on clinically depressed patients have reported no affective potentiation of the startle reflex when viewing unpleasant stimuli (e.g., Allen, Trinder, & Brennan, 1999; Dichter, Tomarken, Shelton, & Sutton, 2004; Kaviani et al., 2004), suggesting that the negative potentiation hypothesis may not generalize to diagnosed depression (e.g., Gotlib, 1984).

The limited support for the negative potentiation hypothesis has led to the formulation of a third alternative, known as the emotional context insensitivity (ECI), which postulates that depression is characterized by valence-independent deficits in emotional responding (Rottenberg, Gross, et al., 2005). The ECI hypothesis is based on the assumption that depressed mood states reduce or disrupt the natural tendency of the organism to respond to environmental stimuli, thus biasing the organism against action (Nesse, 2000; Nesse & Ellsworth, 2009). As a consequence, depressed mood would result in reduced reactivity to pleasant *and* unpleasant emotional stimuli and therefore in reduced approach- *and* withdrawal-related behaviors, respectively (e.g., Bradley, 2000). Impaired valence modulation of subjective measures in response to pleasant and unpleasant film clips provided support for the ECI hypothesis (Rottenberg, Gross, et al., 2005; Rottenberg, Kasch, Gross, & Gotlib, 2002). Specifically, it has been shown that the levels of self-reported sadness did not vary as a function of the emotional valence of film clips in clinically depressed patients, whereas healthy individuals reported higher levels of sadness in sad rather than neutral and pleasant film clips.

In addition to the subjective emotional experience, the ECI hypothesis has been tested using measures of emotion-expressive behavior (e.g., zygomatic or corrugator electromyography, EMG), the eyeblink startle reflex and psychophysiological reactivity. Consistent with the ECI hypothesis, patients with MDD are likely to be characterized by reduced facial EMG modulation during an affective imagery task compared to healthy controls (Gehricke & Shapiro, 2000; Greden, Genero, Price, Feinberg, & Levine, 1986), although mixed results exist (Rottenberg, Gross, et al., 2005). Likewise, there is evidence of reduced affective modulation of the startle reflex during the passive viewing of affective pictures in clinically depressed patients (Allen et al., 1999; Dichter et al., 2004; Kaviani et al., 2004) and nonclinical depressed individuals (Mneimne, McDermut, & Powers, 2008; but see also Sloan & Sandt, 2010) compared to healthy controls.

1.2. Autonomic emotional responding and depressed mood

In the attempt to better understand how depression influences emotional responding, Bylsma et al. (2008) have conducted a meta-analysis of studies examining emotional reactivity in patients diagnosed with MDD. Overall, the results of the meta-analysis revealed that patients with MDD showed reduced reactivity to unpleasant and pleasant stimuli. However, the reduction was greater for positive (Cohen's $d = -0.53$) than negative (Cohen's $d = -0.25$) stimuli. Although these results appear to support the positive attenuation and the ECI hypotheses, findings were inconsistent when each response domain (experiential/subjective, behavioral/expressive, and peripheral physiology) was analyzed separately. With respect to peripheral physiology, the meta-analysis included only four studies and did not find evidence for the positive attenuation hypothesis in MDD patients. In addition, although Bylsma et al. (2008) reported a small effect size for the reduction of physiological reactivity in response to negative emotion (Cohen's $d = -0.22$), this effect size had significant heterogeneity, creating challenges in data interpretation. In particular, in contrast to the ECI hypothesis, heart rate and skin conductance level, which are the most frequently used measures of autonomic nervous system (ANS) arousal in emotional responding (for a review, see Kreibig, 2010), were not reduced in emotionally laden contexts in clinically depressed patients compared to healthy controls (Rottenberg, Gross, et al., 2005; Rottenberg et al., 2002). Similarly, Sloan and Sandt (2010) did not find any association between dysphoric symptoms and emotional modulation of autonomic arousal, as evaluated with heart rate and skin conductance level.

A possible explanation for these inconsistent findings is that the physiological measures used so far to examine ANS reactivity to emotional stimuli may have been partially inadequate. Specifically, given that skin conductance level reflects the activity of sympathetic cholinergic neurons at the level of eccrine dermal sweat glands (Venables & Christie, 1980), it is not informative regarding the parasympathetic branch of the ANS. Conversely, the heart is dually innervated by the sympathetic and the parasympathetic nervous systems, which accelerate and decelerate heart rate, respectively, and vary coactively, independently or reciprocally in response to different stimuli (Berntson, Cacioppo, & Quigley, 1991, 1993). It is possible therefore that emotional responding involves a coactivation of the cardiac sympathetic and parasympathetic branches, which, in turn, may explain the null effect observed for heart rate reactivity.

To date, however, studies examining cardiac autonomic emotional responding have mainly focused on healthy individuals and reported heart rate acceleration due to cardiac vagal withdrawal rather than increased β-adrenergic sympathetic activity in response to specific positive emotions (Kreibig, 2010). Specifically, it has been shown that autonomic response to positive emotions such as happiness or imagined anticipatory pleasure includes increased heart rate and vagal suppression, as reflected by reduced heart rate variability (HRV; e.g., Marci, Glick, Loh, & Dougherty, 2007; Rainville, Bechara, Naqvi, & Damasio, 2006; for a review, see Kreibig, 2010). Likewise, there is preliminary evidence that sadness is also associated with increased heart rate and cardiac vagal withdrawal, as reflected by reduced root mean square of successive RR differences (rMSSD) of HRV, in healthy participants during personalized recall of sad vs. neutral episodes (Rainville et al., 2006).

1.2.1. Cardiac vagal fluctuation and depressed mood

The predominance of vagal control on heart rate response to emotional stimuli in healthy individuals is consistent with previous studies suggesting that the parasympathetic branch of the ANS activates specific organs with rapid, phasic effects as a function of environmental conditions (e.g., Porges, 1995; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). Although parasympathetic control works to "brake" energy expenditure during resting conditions, it can be rapidly and actively withdrawn – which we refer to vagal fluctuation (Rottenberg, Clift, Bolden, & Salomon, 2007) – in response to demanding environmental conditions that require information processing or increased attention (Suess, Porges, & Plude, 1994), coping with positive or negative emotions (Beauchaine, 2001; Friedman & Thayer, 1998; Thayer, Friedman, & Borkovec, 1996), and threats to life (George et al., 1989). Therefore, vagal withdrawal found in response to emotionally arousing scripts

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