Biological Psychology 125 (2017) 130–145

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

The combined effects of menstrual cycle phase and acute stress on reward-related processing

Stella Banis[a,*], Monicque M. Lorist[a,b]

[a] Department of Experimental Psychology, University of Groningen, Groningen, The Netherlands
[b] BCN Neuroimaging Center, University of Groningen, Groningen, The Netherlands

ARTICLE INFO

Article history:
Received 4 December 2015
Received in revised form 6 December 2016
Accepted 21 February 2017
Available online 22 February 2017

ABSTRACT

We investigated the combined effects of menstrual cycle phase and acute stress on reward-related processing, employing a monetary incentive delay task in combination with EEG. Females participated during late follicular and late luteal phases, performing in both control and stress conditions. We found evidence for both independent and interaction effects of phase and stress on reward-related brain activity. Phase modulated the sensitivity to feedback valence, with a stronger signaling of negative performance outcomes in the late follicular versus late luteal phase. In contrast, in the control condition, the late luteal versus late follicular phase was associated with a heightened sensitivity to reward condition, with enhanced performance monitoring in potential-reward versus no-reward trials. Stress decreased attentional preparation during reward anticipation, but increased the influence of reward condition on the processing of positive performance outcomes. We found no evidence for an increased sensitivity to stress during the late luteal versus late follicular phase.

1. Introduction

Fluctuations in gonadal hormone levels are thought to play an important role in the development of certain psychiatric disorders in women (Deecher, Andree, Sloan, & Schechter, 2008; Steiner, Dunn, & Born, 2003). For example, the increased vulnerability to depression in women relative to men appears to be most pronounced during the late luteal (i.e. premenstrual) phase, the postpartum period, and the perimenopausal period, all stages in which hormonal fluctuations are steep (Deecher et al., 2008). This association between fluctuating hormones and disorders with sex differences in prevalence rates may be partly based on hormonal modulations of the brain's reward and stress circuitries (Kajantie & Phillips, 2006; Russo & Nestler, 2013). Moreover, activity within reward systems has been shown to be influenced by stress exposure (Dedovic, D'Aguiar, & Pruessner, 2009; Starcke & Brand, 2012). However, only little is known about how hormonal modulations of reward-related processing and stress regulation interact. In the present study, we aimed at examining the combined effects of menstrual cycle phase and acute stress on reward-related processing, using the menstrual cycle as a natural paradigm to examine the effects of changing hormone levels.

The menstrual cycle has a median length of 29.5 days (Becker et al., 2005), which can be divided into the follicular phase, the period from menstruation until ovulation, and the luteal phase, the period between ovulation and menses onset (Chabbert Buffet, Djakoure, Christin Maitre, & Bouchard, 1998). In the early follicular phase, levels of the gonadal hormones estradiol and progesterone are very low. Estradiol levels start rising from the midfollicular phase and peak during the late follicular phase, while progesterone remains low. During the luteal phase, estradiol levels decrease to a moderate level, while progesterone increases, peaking at the midluteal phase. The late luteal phase is marked by a steep decline of both estradiol and progesterone levels (Chabbert Buffet et al., 1998). Animal studies have shown widespread neurophysiological effects of these hormones (Becker, 2009; McEwen, 2002), but their influence on the brain's reward and stress circuitries in women has remained elusive (Dreher et al., 2007).

Preclinical research has yielded substantial evidence that estradiol and progesterone interact with mesolimbic and mesocortical dopamine (DA) systems, which play an important role in reward-related behaviors (Becker, 2009; McEwen). Especially, estradiol appears to potentiate DA activity, whereas progesterone has been hypothesized to oppose this effect (Jackson, Robinson, & Becker, 2006). In humans, subjective responses in women to stimulant

* Corresponding author at: Department of Psychology, Faculty of Behavioral and Social Sciences, University of Groningen, Grote Kruisstraat 2/1, 9712 TS Groningen, The Netherlands.
E-mail address: h.m.banis@rug.nl (S. Banis).

http://dx.doi.org/10.1016/j.biopsycho.2017.02.005
0301-0511/

drugs have been reported to be increased during the follicular compared to the luteal phase (see for review, Terner & De Wit, 2006). Findings from fMRI studies have supported the stimulating influence of estradiol on the brain's reward system. For example, Dreher et al. (2007) found that brain reward areas showed increased activity in the midfollicular relative to the midluteal phase. In addition, Thomas, Météreau, Déchaud, Pugeat, and Dreher (2014), investigating the impact of hormonal treatment (HT) during the menopause transition, scanning women immediately after estradiol therapy and before progesterone administration, found that HT increased responsiveness of reward areas. Furthermore, estradiol and progesterone may interact on the reward system, resulting in decreased reward-related neural activity, as evidenced by Bayer, Bandurski, and Sommer (2013), who found a reduced sensitivity to the magnitude of gains and losses, in the midluteal compared to the early follicular phase.

Importantly, given the high variability of hormone levels across the cycle, differences between the follicular and luteal phases in reward-related processing might well depend on the specific subphases examined. More specifically, it has been hypothesized that the sudden drop in hormone levels during the late luteal phase causes a decline in endogenous DA activity, mimicking a withdrawal state, which in turn may cause enhanced DA release in response to reward cues (see for review, Ossewaarde et al., 2011b). This could, for example, explain the more frequent cravings of women for foods in combination with increases in energy intake in the (late) luteal relative to the follicular phase (Davidsen, Vistisen, & Astrup, 2007; Dye & Blundell, 1997), and the higher liking of alcohol consumption in the late luteal compared to the midfollicular phase (Evans & Levin, 2011). Findings from fMRI studies on this topic have yielded equivocal results. Ossewaarde et al. (2011b) found enhanced ventral striatal responses to reward anticipation during the late luteal as compared to the late follicular phase. In contrast, Macoveanu et al. (2016), employing a sex-steroid hormone manipulation which reduced estradiol and testosterone levels, found *reduced* amygdala responsivity to the magnitude of rewards in the manipulation compared to the placebo condition in the mid- to late follicular phase. In sum, the evidence is mixed with regard to the influence of dropping hormone levels on reward-related brain activity.

Besides changes in reward-related processing, the menstrual cycle has been associated with changes in stress-sensitivity. Stress-related cardiovascular reactivity and cortisol levels have been shown to increase in the luteal relative to the follicular phase (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Lustyk, Olson, Gerrish, Holder, & Widman, 2010; Tersman, Collins, & Eneroth, 1991). In addition, neuroimaging studies have shown that neural responses in the stress response circuitry to emotional stimuli vary across the cycle (Goldstein, Jerram, Abbs, Whitfield-Gabrieli, & Makris, 2010; Ossewaarde et al., 2010; Protopopescu et al., 2005). Given that the brain's stress circuit is densely populated with estradiol receptors, and elevated estradiol levels during the late follicular phase have been associated with an attenuation of stress-related brain activity (Jacobs et al., 2015), these cycle-related fluctuations in stress-sensitivity may be related to gonadal hormone fluctuations, as well.

In addition to the menstrual cycle-related variability in reward-related processing and stress-sensitivity, both effects might be interrelated, as exposure to stress has been shown to modulate reward-related behaviors. For example, acute stress enhances eating in the absence of hunger (Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2009), and stress stimulates the transition to and maintenance of alcohol and drug dependence (Koob, 2008; Uhart & Wand, 2009). Neuroimaging studies have shown that stress may reduce potential-reward-related activity in the medial prefrontal cortex during reward anticipation (Ossewaarde et al., 2011a) and decrease sensitivity to the valence of monetary outcomes in the dorsal striatum and orbitofrontal cortex (Porcelli, Lewis, & Delgado, 2012). Furthermore, in two previous electroencephalography (EEG) studies (Banis, Geerligs, & Lorist, 2014; Banis & Lorist, 2012), we found evidence for impaired processing of monetary outcomes, under acute stress.

Aim of the present study was to investigate the combined effects of menstrual cycle phase and acute stress on reward-related processing. We compared the late luteal phase, characterized by a steep decline in hormone levels, and the late follicular phase, marked by high estradiol and low progesterone levels. Stress was induced by exposing participants to highly aversive (versus neutral) movie fragments in combination with a self-referencing instruction, immediately before the task blocks (e.g., Henckens, Hermans, Pu, Joëls, & Fernández, 2009). To validate the procedure, we measured heart rate, heart rate variability, and subjective emotions, during the movie clips; and salivary cortisol and subjective negative affect, prior to and after the task blocks.

To examine reward-related processing, we used a modified version of the monetary incentive delay (MID) task (Knutson, Westdorp, Kaiser, & Hommer, 2000). The task consists of potentially rewarding and nonrewarding trials, indicated by a cue. Following this cue, participants are presented with a target upon which they have to react as quickly as possible, by pressing a button. Feedback informs them whether they have reacted within the presentation time of the target and whether they have won money in that trial. During task performance, we applied EEG. Employment of the MID task in combination with the high temporal resolution of the EEG recordings enables the examination of successive stages of reward-related brain activity, related to reward anticipation and feedback (Broyd et al., 2012).

So far, EEG studies of reward-related processing have mainly focused on the processing of feedback, whereas the stage of reward anticipation has received less attention. Recent research suggests that the prospect of reward may enhance attentional preparation to upcoming stimuli (Van den Berg, Krebs, Lorist, & Woldorff, 2014). In the EEG time domain, cues signaling the impending presentation of a stimulus requiring a response, elicit the contingent negative variation (CNV; Walter, Cooper, Aldridge, McCallum, & Winter, 1964). The CNV has been shown to reflect the orienting to and anticipation of the imperative stimulus, and response preparation (Grent-'t-Jong & Woldorff, 2007; Van Boxtel & Böcker, 2004). In the frequency domain, attentional preparation to upcoming stimuli has been associated with cue-related alpha power reductions over occipital regions representing the attended location, which are thought to reflect an increase in cortical excitability facilitating the processing of upcoming stimuli (Thut, Nietzel, Brandt, & Pascual-Leone, 2006; Worden, Foxe, Wang, & Simpson, 2000). Top-down control signals from the fronto-parietal attentional network are thought to be the source of these attention-related modulations (Capotosto, Babiloni, Romani, & Corbetta, 2009). As reward prospect may amplify attentional preparation (Van den Berg et al., 2014), we expected potential-reward-related enhancements of the CNV and reductions in alpha power, in the current study.

With regard to the processing of feedback, the feedback-related negativity (FRN) is a well-known ERP component, which is elicited in response to external feedback and is larger in amplitude following negative compared to positive outcomes (e.g., Gehring & Willoughby, 2002). In the frequency domain, increases in theta power over frontocentral scalp sites have been shown to be larger after negative relative to positive outcomes (e.g., Cohen, Elger, & Ranganath, 2007). Both the FRN and feedback-related theta oscillations are thought to reflect the signaling of unfavorable outcomes (Cohen, Wilmes, & Van de Vijver, 2011; Van de Vijver, Ridderinkhof, & Cohen, 2011). Based on these findings, we expected larger FRN