University of Groningen

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

Banis, Stella; Lorist, Monicque M.

Published in:
Biological Psychology

DOI:
[10.1016/j.biopsycho.2017.02.005](https://doi.org/10.1016/j.biopsycho.2017.02.005)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2017

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Banis, S., & Lorist, M. M. (2017). The combined effects of menstrual cycle phase and acute stress on reward-related processing. *Biological Psychology*, 125, 130-145.
<https://doi.org/10.1016/j.biopsycho.2017.02.005>

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

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.

© 2017 Elsevier B.V. All rights reserved.

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 mid-luteal 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).

drugs have been reported to be increased during the follicular compared to the luteal phase (see for review, [Turner & 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 sub-phases 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

amplitudes and larger increases in theta power following misses compared to hits, in the present study.

In sum, we investigated the combined effects of menstrual cycle phase and acute stress on reward-related brain activity, as reflected in cue-related and feedback-related EEG measures. Based on the literature described above, we expected changes in sensitivity to reward prospect and feedback information across the menstrual cycle, as reflected in phase modulations of cue-related and feedback-related EEG measures. In addition, we expected acute stress to impair reward-related neural processing, as reflected in stress modulations of these measures. Finally, we expected an increased sensitivity to stress during the late luteal relative to the late follicular phase, as reflected in enhanced subjective and physiological stress responses, and in enhanced impairments of reward-related neural processing.

2. Method

2.1. Participants

Due to the novelty of the current design, we could not predict effect sizes in advance. Given the extensive design of the study and the application of strict inclusion and exclusion criteria, we aimed at including as many participants as possible. Our final sample ($n = 17$) permitted the detection of large effects.

Eighteen healthy, non-pregnant, right-handed females (mean age = 20.7 years, range 19–26 years) completed both experimental sessions. None of the women had used hormonal contraceptives within the six months previous to these sessions, and all had regular menstrual cycling with normal mean cycle length (mean = 29 days, range 26–34 days). They had no history of psychiatric disorders including (Premenstrual Dysphoric Disorder (PMDD), as determined with the Mini International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998) and the Premenstrual Symptoms Screening Tool (PSST; Steiner, Macdougall, & Brown, 2003). None of the participants had experienced severe physical or emotional trauma. Furthermore, participants reported no evidence of neurological or endocrine disease; no current use of psychoactive medication or drugs or corticosteroids; no habit of watching violent movies or playing violent video games; and normal or corrected-to-normal vision. Participants did not consume more than three alcoholic beverages per day on average, and did not smoke. In addition, participants were asked not to consume alcohol 24 h prior to the experiment. Participants received either course credits or money for their participation. In addition, they received a monetary bonus depending on their task scores, as described below. The study was approved by the Ethical Committee Psychology of the Psychology Department of the University of Groningen, and all participants gave written informed consent.

2.2. Design and procedure

Participants were tested in a crossover design with the counter-balanced factors menstrual cycle phase (late follicular versus late luteal) and stress induction (stress versus control). Each woman was tested once during the late follicular phase and once during the late luteal phase, performing in both stress induction conditions during each session. During a screening session prior to the actual experiment, candidates completed the PSST (Steiner, Macdougall et al., 2003) and the M.I.N.I. (Sheehan et al., 1998). In addition, all participants received instructions for the ovulation predictor test (see below).

Timing of experimental sessions was determined as follows. Late follicular phase sessions were scheduled between days 8 and 12 with respect to the first day of the menstrual cycle (day

1 = menses onset; mean time point of session: day 10.7, $SD = 1.2$). All late follicular sessions took place in menstrual cycles of normal length ($M = 28.6$, $SD = 2.5$, range 24–33 days). Late luteal phase sessions were planned following the luteinizing hormone (LH) surge, as determined using commercially available ovulation predictor tests (Dutch Diagnostics, Zutphen, The Netherlands). Sessions were scheduled between days 10 and 14 after the surge (day 0 = LH surge; mean time point of session: 3.3 days before menstruation started, $SD = 1.6$). For menstrual cycle phase verification, we measured salivary progesterone levels on both session days. In addition, all participants were asked to report the date of onset of their next menses. These verification measures also allowed us to confirm that no participant was pregnant during the experiment.

On the days of the experimental sessions, participants arrived at the laboratory at 11.30 a.m. After the application of the electrocap and the electrocardiogram (ECG) electrodes, participants practiced the MID task. Then, they provided salivary samples for progesterone determination, after which they had a resting period of 5 min.

All experimental testing took place between 13.00 and 17.00 p.m. to ensure relatively stable and low levels of endogenous cortisol. Participants completed two task blocks (12 min each) of the MID task, in both stress induction conditions (Fig. S1). Immediately before the task blocks, participants were shown highly aversive versus neutral control movie fragments (2:20 or 1:30 min). In addition, halfway through the task blocks (after 6 min), part of the preceding fragment (0:45 min) was shown again. The order of stress induction conditions was counterbalanced across subjects. Both conditions were separated by a break of 75 min. Participants completed the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) and provided salivary samples for cortisol determination, at three time points, in both stress induction conditions: before the first task block (t_1), after the first task block (t_2) and after the second task block (t_3). In addition, participants rated their emotions while watching the movie clips, after the second task block of both conditions.

2.3. Stress induction

To induce a stressful state, highly aversive movie fragments were shown to the participants immediately before the task blocks. In addition, halfway through the task blocks, part of the preceding fragment (0:45 min) was shown again. The four movie clips were selected from a distressing movie [*Irréversible* (2002), Gaspar Noé] and contained scenes with maximally aggressive behavior and violence against men and women. Occasionally, people in the video shouted and cried out in anger, pain, or distress. The effectiveness of these movie clips in inducing stress has been confirmed in previous studies (Henckens et al., 2009; Ossewaarde et al., 2010). For the control condition, neutral fragments from another movie [*Comment j'ai tué mon père* (2001), Anne Fontaine] were shown. Stressful and neutral movie clips were comparable in amount of speech, human presence, luminance, and language. Participants were instructed to view the movie clips (2:20, 1:30, 1:30, 1:30 min, respectively) attentively, imagining being an eyewitness of the events. Additionally, they were asked to watch constantly, not to look away from the screen.

2.4. Monetary incentive delay task

The task was a modified version of the MID task as developed by Knutson et al. (2000). Each task block consisted of 80 potentially rewarding trials and 80 nonrewarding trials. Participants completed two task blocks per stress induction condition, resulting in 160 potentially rewarding trials and 160 nonrewarding trials per condition.

Each trial (Fig. S2) started with the presentation of a fixation cross, for a randomly varying interval of 800–1200 ms. Then, a cue was presented for 250 ms signaling potential reward (a plus sign within a circle) or no reward (a times sign within a circle), starting the anticipation phase. Following a second presentation of a fixation cross (800–1200 ms), a brief target (a white square) appeared on the screen with a start duration of 200 ms. Participants were instructed to push a button as fast as possible upon detection of the target, irrespective of the cue type. Following a third presentation of a fixation cross (800–1200 ms), there was an outcome phase in which feedback was presented for 1000 ms. Feedback informed participants whether they had pushed the button within the presentation time of the target (“hit!” or “miss!”), and whether they had won money in that trial (“+€10” or “+€0”). In potentially rewarding trials only, hits were rewarded with €10. At the end of each task block, participants received additional feedback indicating the amount of money earned during the previous block. They were told that they would earn a percentage of their cumulative total win, after both experimental sessions, but were not told the exact percentage. To equalize total gain across conditions and participants, the presentation time of the target was adapted on a trial by trial basis per reward condition. Target duration was shortened by 20 ms when the previous target was hit; it was lengthened by 10 ms when the previous target was missed (Ossewaarde et al., 2011b). In addition, target duration was set to never exceed 100–1000 ms boundaries.

2.5. Progesterone sampling and analysis

To measure progesterone levels, single saliva samples (3 ml) were collected during both experimental sessions, using saliva tubes (Greiner Bio One, Alphen aan de Rijn, Netherlands). Participants were requested not to brush or floss their teeth, and to abstain from eating and drinking anything but water, for 3 h prior to saliva sampling. All samples were stored at a maximum temperature of -20°C until analysis. Thawed samples were prepared for biochemical analysis by centrifuging them for 10 min at 2000g. Progesterone concentrations were determined in duplicate samples employing an in house radioimmunoassay, with a sensitivity of 37 pmol/L (Radboud University Nijmegen Medical Centre, Nijmegen, Netherlands).

After progesterone determination, one participant was excluded from further analysis, because her salivary progesterone level in the follicular phase (371.0 pmol/L) deviated more than three standard deviations from the group mean ($M = 79.1$ pmol/L, $SD = 74.9$). Salivary progesterone levels from 17 participants were analyzed using a paired *t*-test.

2.6. Measurements of stress and data reduction

2.6.1. Subjective measurements of stress

Mood was assessed using the PANAS (Watson et al., 1988), at three time points in each stress induction condition: before the first task block (t1), after the first task block (t2), and after the second task block (t3; Fig. S1). In addition, after the second task block of each stress induction condition, participants rated their emotions while watching the movie clips, on a 10-point scale (1 = not at all, 10 = very much so). Those emotions included anger, fear, sadness, happiness, disgust, and surprise.

2.6.2. Physiological measurements of stress

To measure the hypothalamic-pituitary-adrenal (HPA) axis stress response as reflected in cortisol levels, saliva samples (2 ml) were collected using saliva tubes, at three time points in each stress induction condition: before the first task block (t1), after the first task block (t2), and after the second task block (t3; Fig.

S1). All samples were stored at a maximum temperature of -20°C until analysis. Thawed samples were prepared for biochemical analysis by centrifuging them for 10 min at 2000g. Cortisol concentrations were determined in duplicate samples using an in house radioimmunoassay, with a sensitivity of 0.30 nmol/L (Radboud University Nijmegen Medical Centre, Nijmegen, Netherlands). Baseline-corrected cortisol levels were determined by subtracting baseline cortisol levels at t1 from cortisol levels at t2 and t3 in each stress induction condition. This baseline correction was applied to account for the typical decline in cortisol levels over the course of the day (Edwards, Clow, Evans, & Hucklebridge, 2001).

To measure the sympathetic nervous system stress response as reflected in heart rate (HR) and heart rate variability (HRV), we recorded the ECG during the movie clips. The ECG was registered using three Sn electrodes, which were placed on the sternum (common electrode) and on the left and right sides of the body, between the two lower ribs. R-peaks in the ECG signal were detected online, with an accuracy of 2 ms, using Portilab (Twente Medical Systems International). These R-peaks were used to create inter-beat interval (IBI) time series. IBI's were visually inspected and manually corrected, upon which mean HR and mean power of HRV in the mid-frequency band (0.07–0.14 Hz) were calculated, using the CARSPAN spectral analysis program (Mulder, 1992). Heart rate variability, especially variability in the 0.10 Hz band, is suppressed during mental effort (e.g., Mulder, De Waard, & Brookhuis, 2005). Power spectral data were Ln-transformed to reduce inter-individual differences in range and to normalize the data (Van Roon, 1998).

2.7. Electrophysiological recordings and data reduction

EEG was measured using 28 Sn electrodes attached to an electrocap (ElectroCap International Inc., Eaton, Ohio, USA), positioned according to the 10–10 system. Recordings were taken from channels FP1, FP2, AFz, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, PO7, O1, Oz, O2 and PO8. Horizontal electro-oculogram (EOG) was recorded bipolarly using two electrodes placed at the outer canthi of both eyes. Vertical EOG was measured using two electrodes placed above and below the left eye. All electrode impedances were kept below 10 k Ω , besides the two reference electrodes on both mastoids which were kept below 5 k Ω . EEG and EOG signals were filtered with a 0.16-Hz high-pass filter and a 200-Hz low-pass filter, and recorded with a 500-Hz sample rate.

Off-line, EEG and EOG data were re-referenced to the computed average of both mastoids. Data were down-sampled to 256 Hz, after additional filtering: for the ERP analysis, with a low-pass filter of 30 Hz and a slope of 48 dB/oct; for the TFR analysis, with a low pass filter of 55 Hz and a slope of 48 dB/oct.

For the ERP analyses of cue-related and feedback-related segments, data were segmented in 1150-ms epochs, starting 100 ms before cue or feedback onset, respectively. For the TFR analysis, segments covered 3000 ms, starting 1000 ms before cue/feedback onset. Epochs with too rapidly changing activity (maximal allowed voltage step ± 60 μV and ± 75 μV for the ERP and TFR analyses, respectively) were rejected. After removal of these artifacts, EEG was corrected for eye movements and blinks using the regression procedure of Gratton, Coles, and Donchin (1983). Then, for the ERP analyses only, epochs which contained EEG voltage differences exceeding 200 μV , or EEG amplitudes exceeding ± 100 μV , were eliminated. Furthermore, ERP/TFR segments were visually inspected for edge artifacts and other remaining artifacts. After these ocular correction and artifact rejection procedures, EEG was averaged relative to a 100 ms pre-cue/feedback baseline. For the ERP analysis of cue segments, separate averages were calculated for each combination of phase (late follicular versus late luteal), stress induction (stress versus control), and reward condition (potential-

reward versus no-reward), resulting in eight average waveforms for each electrode and participant. For the ERP analysis of feedback segments, separate averages were calculated for each combination of phase, stress induction, feedback valence (hit versus miss), and reward condition, resulting in sixteen average waveforms for each electrode and participant.

Time-frequency analyses were performed with the Matlab-based FieldTrip toolbox (Oostenveld, Fries, Maris, & Schoffelen, 2011). To study the oscillatory dynamics of the EEG, single-trial cue/feedback-locked data were convolved with a family of complex Morlet wavelets. These wavelets contained a fixed number of cycles of sinusoidal oscillations for each frequency band (4–7 Hz, 5 cycles; 8–12 Hz, 6 cycles; 13–20 Hz, 7 cycles; 21–30 Hz, 7 cycles). This analysis produced raw power estimates for each time point between 400 ms pre-cue/feedback and 1050/1000 ms post-cue/feedback (in 10-ms steps) at frequencies of 4–30 Hz (in 0.5-Hz steps). Subsequently, a condition-specific, relative baseline correction was applied. First, we calculated average spectral power across trials per condition per participant. Then, we divided the average power at each time point by the average power of the pertaining frequency in the –400––200 ms pre-cue/feedback interval.

2.8. Data analysis

2.8.1. Measurements of stress

Negative affect ratings and baseline-corrected cortisol levels were subjected to repeated measures analyses of variance (rANOVAs) with the within-subjects (WS) factors phase, stress induction and time (negative affect: t1, t2, t3; cortisol: t2, t3). Emotion ratings were subjected to rANOVAs with the WS factors phase, stress induction and emotion (six emotions). HR and HRV values were subjected to rANOVAs with the WS factors phase, stress induction and clip (clip 1, clip 2).

Treatment of missing data. One cortisol sample from one participant was missed due to researcher error (forgetting to sample), and two HR as well as HRV measurements from another participant were missed due to technical problems during the experiment. Excluding participants because of missing data possibly affects the representativeness of findings and reduces statistical power (Graham, 2009). Therefore, we used the multiple imputation method (Multiple Imputation module of SPSS Version 21.0: imputation method automatic, linear regression) to predict the values of these missing data, as described by Van Buuren (2007).

2.8.2. Behavioral measures

Reaction time data of responses during the MID task were first filtered by removing values below 100 ms (Hsu, 2005; Ulrich & Miller, 1994). Subsequently, outliers relative to participants' condition-specific (phase by stress induction by reward condition) means were eliminated, using the outlier removal algorithm outlined by Van Selst and Jolicoeur (1994). The resulting mean reaction times were subjected to rANOVAs with the WS factors phase, stress induction and reward condition.

2.8.3. ERPs

For the ERP analyses, electrodes and time windows were selected on the basis of previous studies and visual inspection of ERP waveforms and topographic maps collapsed across conditions and participants.

Cue-related ERPs. In line with previous findings, we found that the CNV was already detectable around 400 ms post-cue, and that its topography shifted from anterior to posterior sites (Fig. 1; Grent-'t-Jong & Woldorff, 2007; Van den Berg et al., 2014).¹ We quantified

the CNV as the mean amplitude in three consecutive windows, at three different electrodes: between 400 and 470 ms at Fz, between 550 and 800 ms at FCz, and between 800 and 1050 ms at Cz. The resulting CNV measures were analyzed using rANOVAs with the WS factors phase, stress induction and reward condition.

Feedback-related ERPs. In our previous studies, we found that FRN results were dependent on the method of measuring FRN amplitude (Banis et al., 2014; Banis & Lorist, 2012). Therefore, the FRN was measured in two ways. First, the FRN was quantified as the mean amplitude (MA) between 250 and 325 ms post-feedback at FCz (see Fig. 2; Di Bernardi Luft, Nolte, & Bhattacharya, 2013; Gehring & Willoughby, 2002). Second, the FRN was measured as the difference in voltage at FCz between the 250–325 ms mean amplitude and the average of the mean amplitudes of the preceding (P200: 160–220 ms window) and following (P300: 350–410 ms window) peaks (MAC = mean amplitude corrected for surrounding peaks; Banis & Lorist, 2012; Yeung & Sanfey, 2004). The resulting FRN measures were analyzed using rANOVAs with the WS factors phase, stress induction, feedback valence and reward condition.

We added the MAC measure to account for possible overlap between the FRN and other ERP components, most notably the P300. In our most recent study including oscillatory power analyses (Banis et al., 2014), we found that the results of the MAC measure best matched the results of feedback-related theta power, a measure which is more directly related to neurophysiological phenomena. Recent studies have further supported the idea that correction for surrounding peaks approaches may yield more reliable results than the mean amplitudes approach, and that studies should include several measuring methods to demonstrate the reliability of reported findings (Mushtaq, Stoet, Bland, & Schaefer, 2013; Pfabigan, Sailer, & Lamm, 2015). In order to gain more insight into the possible role of overlapping components in the present study, we performed rANOVAs on the peaks surrounding the FRN, as well.

2.8.4. Oscillatory power

For the time-frequency analyses, electrodes and time windows were selected on the basis of previous studies, and visual inspection of average topographical plots and average power plots across conditions and participants (see Fig. 3, Fig. 4; Cohen, 2014).

Cue-related power. Cue-related alpha (8–12 Hz) was quantified as the mean activity between 400 and 1050 ms post-cue, at Oz (Capotosto et al., 2009; Thut et al., 2006; Worden et al., 2000). For exploratory purposes, cue-related theta (4–7 Hz) was quantified as the mean activity at Fz, between 200 and 500 ms post-cue. The resulting power values were analyzed using rANOVAs with the WS factors phase, stress induction and reward condition.

Feedback-related power. Feedback-related theta power (4–7 Hz) was quantified as the mean activity at Fz, between 300 and 600 ms post-feedback. The resulting power values were analyzed using rANOVAs with the WS factors phase, stress induction, feedback valence and reward condition.

¹ In the present study, participants were instructed to react as quickly as possible upon detection of the target, and were thus stimulated to prepare instantly following cue-onset. Cue-target intervals were very short, ranging from 1050 to 1450 ms. This experimental set-up is similar to the set-ups by Van den Berg et al. (2014) and Grent-'t-Jong and Woldorff (2007), who employed short cue-target intervals as well (700 or 1300 ms, and 900 or 1900 ms, respectively). These short cue-target intervals appear to stimulate fast attentional orientation, as reflected in the early onset of a sustained negative polarity, around 400 ms after cue-onset, in the latter two and the present studies. According to Grent-'t-Jong and Woldorff (2007) attentional orienting is initiated by the medial frontal cortex, which then engages medial parietal areas. Furthermore, from around 400 ms, our ERPs show activity overlapping with the supposedly early CNV. We presume that this activity reflects sensory-evoked activity caused by the visual offset of our cue, at 250 ms (Luck, 2014). A similar pattern is visible in the ERPs reported by Van den Berg et al. (2014) employing a cue duration of 400 ms, somewhat later in the cue-related interval.

¹ In the present study, participants were instructed to react as quickly as possible upon detection of the target, and were thus stimulated to prepare instantly follow-

Fig. 1. Cue-related topographical voltage maps and ERPs. (A) Topographical voltage maps (400–470 ms, 550–800 ms, 800–1050 ms post-cue) of the difference between potential-reward and no-reward trials, showing the shift from frontal to more posterior sites. (B) ERPs from Fz, FCz and Cz: thick lines represent potential-reward trials; thin lines represent no-reward trials; blue lines represent the late follicular phase; red lines represent the late luteal phase. The CNV was more negative following potential-reward relative to no-reward cues. The reward condition effect did not significantly differ between late luteal and late follicular phases. (C) Topographical voltage map (400–470 ms) of the difference between control condition and stress condition trials. (D) ERPs from Fz: solid lines represent the control condition; dotted lines represent the stress condition; blue lines represent the late follicular phase; red lines represent the late luteal phase. The CNV was smaller in the stress relative to the control condition, during the early interval (400–470 ms), independent of phase.

2.8.5. Specifications statistical analyses

For all rANOVAs in this study, the univariate results are reported, with Greenhouse-Geisser corrected p-values for non-sphericity being reported when appropriate. Reported p-values are two-tailed unless specified as one-tailed. Effect sizes are reported using partial eta-squared (η_p^2), which is the proportion of variance explained by

a given variable of the variance remaining after excluding variance explained by other variables (Richardson, 2011). Values of 0.01, 0.06, and 0.14 are considered to reflect small, medium, and large effects, respectively (Cohen, 1969, as cited in Richardson, 2011). As power was limited in this study due to the small sample size, we also reported effects showing p-values approaching significance

Fig. 2. Feedback-related topographical voltage maps and ERPs. (A) Topographical voltage maps (250–325 ms post-feedback) of the difference between miss and hit trials (left) and between no-reward and potential-reward trials (right). (B) ERPs from FCz as a function of feedback valence and reward condition: solid lines represent hit trials; dashed lines represent miss trials; thick lines represent potential-reward trials; thin lines represent no-reward trials. The FRN was larger in response to misses relative to hits, and in no-reward compared to potential-reward trials. The feedback valence effect on the FRN was most pronounced in potential-reward trials. (C) ERPs from FCz as a function of feedback valence and menstrual cycle phase: solid lines represent hit trials; dashed lines represent miss trials; blue lines represent the late follicular phase; red lines represent the late luteal phase. The effect of feedback valence on the FRN (as quantified by the MAC) was more pronounced in the late follicular relative to the late luteal phase. (D) ERPs from FCz as a function of reward condition and stress induction, for hits and misses separately. Thick lines represent potential-reward trials; thin lines represent no-reward trials; black lines represent the control condition; red lines represent the stress condition. The effect of reward condition on the processing of feedback valence was more pronounced in the stress relative to the control condition, especially due to differential processing of hits. This result only applied to the FRN as quantified by the MAC.

(between 0.05 and 0.10). These effects should be interpreted with caution. For analyses following up on significant interactions, we applied the Bonferroni method in order to adjust for multiple comparisons.

3. Results

3.1. Progesterone levels

Salivary progesterone levels were higher in the late luteal phase ($M = 197.2$ pmol/L, $SD = 128.0$) than in the late follicular phase ($M = 61.9$ pmol/L, $SD = 18.0$, $t(16) = 4.47$, $p < .001$), confirming that participants were on average tested during the intended menstrual cycle phase. Late luteal progesterone levels varied from < 37 pmol/L (level not measurable by assay) to 491.0 pmol/L. Fifteen participants showed the highest levels during the late luteal phase. One participant showed a slightly lower level during the late luteal (47.4 pmol/L) compared to the late follicular phase (60.7 pmol/L),

while another participant showed similar levels during both phases (< 37 pmol/L). The latter two participants had their menses onset shortly after their luteal sessions, that is on the same date.

3.2. Measurements of stress

3.2.1. Subjective measurements of stress

Participants reported having experienced more anger, fear, sadness, disgust and surprise, and less happiness, while watching the aversive relative to the neutral movie clips (stress induction: $F(1, 16) = 67.96$, $p < .001$, $\eta_p^2 = .81$; stress induction by emotion: $F(5, 80) = 62.97$, $p < .001$, $\eta_p^2 = .80$; Table 1). In addition, the effect of stress induction depended on the combination of emotion and phase ($F(5, 80) = 2.55$, $p = .034$, $\eta_p^2 = .14$). Especially, the stress-related increase in disgust seemed to be more pronounced in the late follicular relative to the late luteal phase, but the stress induction by phase interaction did not reach significance ($F(1, 16) = 5.92$, $p = .027$, $\eta_p^2 = .27$).

A Cue-related relative power averaged over all conditions, at Oz

B Cue-related relative alpha power averaged over all conditions, at Oz

Fig. 3. Time-frequency and line plots of cue-related relative power, averaged over all conditions. (A) Time-frequency representation of cue-related relative power at Oz, averaged over all conditions. Only for time-frequency plots, relative power averages were converted to a decibel (dB) scale, enabling comparison between different frequencies. (B) Line plot of cue-related relative alpha (8–12 Hz) power at Oz, averaged over all conditions.

A Feedback-related relative power averaged over all conditions, at Fz

B Feedback-related relative theta power averaged over all conditions, at Fz

Fig. 4. Time-frequency and line plots of feedback-related relative power, averaged over all conditions. (A) Time-frequency representation of feedback-related relative power at Fz, averaged over all conditions. Only for time-frequency plots, relative power averages were converted to a decibel (dB) scale. (B) Line plot of feedback-related relative theta (4–7 Hz) power at Fz, averaged over all conditions.

Table 1

Mean ratings of emotions experienced during movie clips, as a function of menstrual cycle phase and stress induction (standard deviations in parentheses).

Phase	Follicular		Luteal	
	Stress	Control	Stress	Control
Anger	5.9 (1.9)	1.4 (0.9)	5.8 (2.5)	1.7 (1.2)
Fear	5.7 (1.7)	1.9 (1.1)	5.8 (1.8)	2.7 (2.0)
Sadness	4.0 (1.8)	2.2 (1.2)	4.5 (2.2)	2.0 (0.9)
Disgust	8.0 (1.0)	1.2 (0.6)	7.4 (1.8)	1.5 (1.2)
Surprise	6.1 (1.7)	3.5 (2.1)	5.0 (2.3)	3.8 (1.8)
Happiness	1.4 (0.9)	4.3 (1.8)	1.4 (0.7)	3.7 (2.0)

In addition, participants reported higher negative affect in the stress relative to the control condition (stress induction: $F(1, 16) = 6.34, p = .023, \eta_p^2 = .28$; Fig. 5). This stress induction effect was modulated by time (stress induction by time: $F(1.50, 23.95) = 4.35, p = .034, \eta_p^2 = .21$). At baseline, there was no significant difference in negative affect between both stress induction conditions ($F(1, 16) = 1.40, n.s., \eta_p^2 = .08$), while at t2 and t3 participants did report higher negative affect in the stress compared to the control condition (t2: $F(1, 16) = 5.21, p = .036, \eta_p^2 = .25$; t3: $F(1, 16) = 7.27,$

$p = .016, \eta_p^2 = .31$). Importantly, phase did not affect negative affect ($F < 1, n.s.$).

3.2.2. Physiological measurements of stress

HR was higher during the aversive movie clips than during the neutral movie clips (stress induction: $F(1, 16) = 3.36, p = .043$, one-tailed, $\eta_p^2 = .17$; Fig. 5). Notably, overall HR during the movie clips was higher during the late luteal phase ($M = 66.3, SD = 10.9$) than during the late follicular phase ($M = 61.4, SD = 8.6$; phase: $F(1, 16) = 5.22, p = .036, \eta_p^2 = .25$). In addition, HRV was lower during the aversive relative to the neutral movie clips ($F(1, 16) = 8.94, p = .009, \eta_p^2 = .36$; Fig. 5). Phase did not affect HRV significantly.

Furthermore, baseline-corrected cortisol levels were higher in the stress relative to the control condition (stress: $M = +0.53 \text{ nmol/L}, SD = 1.57$, control: $M = -.29 \text{ nmol/L}, SD = 0.51$; stress induction: $F(1, 16) = 3.56, p = .039$, one-tailed, $\eta_p^2 = .18$; Fig. 5). The observed pattern suggests that the effect of stress increased with time, but the stress induction by time interaction did not reach significance ($F(1, 16) = 4.19, p = .057, \eta_p^2 = .21$). Phase did not affect these baseline-corrected cortisol levels. Notably, phase did affect cortisol levels at baseline, that is immediately before the first task block in both stress induction conditions, with higher levels during the late follic-

Fig. 5. Effects of stress induction on subjective and physiological stress measures. Mean negative affect, baseline-corrected salivary cortisol, heart rate and heart rate variability (0.07–0.14 Hz) as a function of time, stress induction and menstrual cycle phase. Error bars represent standard errors. Solid lines represent the control condition; dotted lines represent the stress condition; blue lines represent the late follicular phase; red lines represent the late luteal phase. Participants reported higher negative affect in the stress relative to the control condition, at t2 and t3 (top left). Heart rate was higher and heart rate variability was lower during the aversive compared to the neutral movie clips (bottom). Baseline-corrected cortisol levels were higher in the stress relative to the control condition (top right). Furthermore, phase affected physiological measures independent of stress induction: baseline cortisol levels were higher in the late follicular relative to the late luteal phase, whereas overall HR during the movie clips was higher during the late luteal versus late follicular phase. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

ular ($M = 3.48$, $SD = 1.73$) relative to the late luteal phase ($M = 2.85$, $SD = 1.53$; phase: $F(1, 16) = 8.53$, $p = .010$, $\eta_p^2 = .35$).

In summary, the results from both subjective and physiological stress measurements confirmed that our stress induction procedure yielded mild to moderate stress responses. These stress responses were not significantly modulated by phase. Further-

more, phase affected physiological measures independent of stress induction. Baseline cortisol levels were higher in the late follicular relative to the late luteal phase, whereas overall HR during the movie clips was higher during the late luteal versus late follicular phase.

A Potential reward minus no reward

Fig. 6. Cue-related topographical map and line plot of alpha relative power (8–12 Hz). (A) Topographical map of the difference between potential-reward and no-reward trials (400–1050 ms post-cue). (B) Line plot of alpha relative power at Oz as a function of reward condition and phase. Thick lines represent potential-reward trials; thin lines represent no-reward trials; blue lines represent the late follicular phase; red lines represent the late luteal phase. Alpha power reductions were larger following potential-reward relative to no-reward cues, independent of phase. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

3.3. Behavioral results

Responses to targets were faster during potential-reward trials ($M = 158$ ms, $SD = 9$) than during no-reward trials ($M = 163$ ms, $SD = 9$; reward condition: $F(1, 16) = 29.52$, $p < .001$, $\eta_p^2 = .65$). Neither phase nor stress induction modulated RTs. The observed mean percentage of hits was slightly higher for potential-reward trials ($M = 37.9\%$, $SD = 1.4$) than for no-reward trials ($M = 37.1\%$, $SD = 1.3$; $t(16) = 2.89$, $p = .011$). All participants won approximately the same amount of money ($M = 24.12$ euros, $SD = 0.82$).

3.4. ERP results

3.4.1. Cue-related activity

The CNV was quantified in three successive post-cue time windows at Fz, FCz and Cz, respectively. During all three intervals, the CNV was larger, that is more negative, following potential-reward

compared to no-reward cues (Fig. 1; Table 2). In the early interval (400–470 ms), the CNV was affected by stress, with smaller amplitudes in the stress relative to the control condition. Phase did not influence this stress induction effect on the CNV (stress induction by phase: $F < 1$, n.s.).

3.4.2. Feedback-related activity

In general, the FRN was larger, that is more negative, in response to misses relative to hits, and in no-reward compared to potential-reward trials (Table 3; Fig. 2). These feedback valence and reward condition effects were significant for both FRN measures. Feedback valence effects were dependent on reward condition. For both FRN measures, the effect of feedback valence was more pronounced in potential-reward (MA: $F(1, 16) = 99.52$, $p < .001$, $\eta_p^2 = .86$; MAC: $F(1, 16) = 46.96$, $p < .001$, $\eta_p^2 = .75$) than in no-reward trials (MA: $F(1, 16) = 45.94$, $p < .001$, $\eta_p^2 = .74$; MAC: $F(1, 16) = 28.15$, $p < .001$, $\eta_p^2 = .64$). In addition, separate analyses per feedback valence showed that the effect of reward condition was stronger in hit (MA: $F(1, 16) = 39.84$, $p < .001$, $\eta_p^2 = .71$; MAC: $F(1, 16) = 21.65$, $p < .001$, $\eta_p^2 = .58$) compared to miss trials (MA: $F(1, 16) = 7.91$, $p = .013$, $\eta_p^2 = .33$; MAC: $F(1, 16) = 2.38$, n.s., $\eta_p^2 = .13$).

The effects of feedback valence and reward condition on peaks surrounding the FRN, that is the P200 and P300, were similar to their effects on the FRN (Table 4). In general, the P200 and P300 were larger in response to hits relative to misses, and in potential-reward versus no-reward trials (Fig. 2). For both the P200 and P300, the effect of feedback valence was larger in potential-reward (P200: $F(1, 16) = 26.68$, $p < .001$, $\eta_p^2 = .63$; P300: $F(1, 16) = 30.07$, $p < .001$, $\eta_p^2 = .65$) relative to no-reward trials (P200: $F(1, 16) = 16.33$, $p = .001$, $\eta_p^2 = .51$; P300: $F(1, 16) = 11.06$, $p = .004$, $\eta_p^2 = .41$). Additionally, the effect of reward condition was larger in hit (P200: $F(1, 16) = 8.54$, $p = .010$, $\eta_p^2 = .35$; P300: $F(1, 16) = 41.25$, $p < .001$, $\eta_p^2 = .72$) relative to miss trials (P200: $F(1, 16) = 3.31$, $p = .088$, $\eta_p^2 = .17$; P300: $F(1, 16) = 10.52$, $p = .005$, $\eta_p^2 = .40$). Measuring the FRN while correcting for overlap with these surrounding peaks (MAC) resulted in smaller, but still large, main and interaction effects of feedback valence and reward condition, relative to the MA measure.

Stress induction modulated the feedback valence by reward condition interaction on the FRN, but only as quantified by the MAC (Table 3). The effect of reward condition on the processing of feedback valence was more pronounced in the stress ($F(1, 16) = 17.85$, $p = .001$, $\eta_p^2 = .53$) relative to the control condition ($F(1, 16) = 5.50$, $p = .032$, $\eta_p^2 = .26$; Fig. 2). Separate analyses per feedback valence (see above) showed that the significant effect of reward condition on the processing of hits was stronger in the stress relative to the control condition (reward condition by stress induction: $F(1, 16) = 6.61$, $p = .021$, $\eta_p^2 = .29$), whereas the nonsignificant effect of reward condition on the processing of misses was not modulated by stress induction (reward condition by stress induction: $F < 1$, n.s.; Fig. 2).

With regard to FRN-surrounding peaks, stress induction modulated the feedback valence by reward condition interaction on the P300, not on the P200 (Table 4). Reward condition only had a significant effect on the processing of feedback valence in the control condition ($F(1, 16) = 33.02$, $p < .001$, $\eta_p^2 = .67$), not in the stress condition ($F(1, 16) = 4.31$, $p = .054$, $\eta_p^2 = .21$; Fig. 2). Furthermore, separate analyses per feedback valence condition showed, opposite to the effects on the MAC measure of the FRN, that the effect of reward condition on the processing of hits was stronger in the control relative to the stress condition (reward condition by stress induction: $F(1, 16) = 7.64$, $p = .014$, $\eta_p^2 = .32$), whereas stress induction did not modulate the effect of reward condition on the processing of misses (reward condition by stress induction: $F < 1$, n.s.; Fig. 2).

Table 2 Summary of effects on cue-related EEG measures. The values of $F(1, 16)$, p and η_p^2 are reported.^a

EEG measure	CNV – Fz 400–470 ms			CNV – FCz 550–800 ms			CNV – Cz 800–1050 ms			Alpha – Oz 8–12 Hz 400–1050 ms		
	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
Reward condition	17.57	.001	.52	11.56	.004	.42	18.23	.001	.53	7.17	.017	.31
Reward condition by phase	3.85	.067	.19									
Stress induction	4.72	.045	.23									

^a Effects are only included if p -value $< .10$ for at least one measure. Entries with an F -value < 1 are omitted.**Table 3**Summary of effects on feedback-related EEG measures. The values of $F(1, 16)$, p and η_p^2 are reported.^a

EEG measure	FRN – FCz Mean amplitude (MA) 250–325 ms			FRN – FCz MA corrected for surrounding peaks ¹			Theta – Fz 4–7 Hz 300–600 ms		
	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
Feedback valence	85.50	$< .001$	.84	42.64	$< .001$	.73	2.26	.153	.12
Reward condition	31.25	$< .001$	.66	8.36	.011	.34	9.05	.008	.36
Feedback valence by reward condition	33.40	$< .001$	.68	15.71	.001	.50	2.90	.108	.15
Feedback valence by reward condition by stress induction				9.51	.007	.37			
Feedback valence by phase	1.40	.253	.08	6.62	.020	.29	1.50	.238	.09
Feedback valence by stress induction by phase	1.55	.230	.09				3.74	.071	.19
Reward condition by stress induction by phase							6.55	.021	.29

^a Effects are only included if p -value $< .10$ for at least one measure. Entries with an F -value < 1 are omitted.¹ Mean amplitude 250–325 ms post-feedback minus average of mean amplitudes preceding (160–220 ms window) and following (350–410 ms window) peaks (MAC).**Table 4**Summary of effects on feedback-related P200 and P300 at FCz. The values of $F(1, 16)$, p and η_p^2 are reported.^a

EEG measure	P200 160–220 ms			P300 350–410 ms		
	F	p	η_p^2	F	p	η_p^2
Feedback valence	32.89	$< .001$	.67	23.42	$< .001$	.59
Reward condition	7.41	.015	.32	28.15	$< .001$	.64
Feedback valence by reward condition	5.89	.027	.27	17.18	.001	.52
Stress induction	3.41	.083	.18			
Reward condition by stress induction				4.54	.049	.22
Feedback valence by reward condition by stress induction				6.22	.024	.28

^a Effects are only included if p -value $< .10$ for at least one measure including the FRN measures. Entries with an F -value < 1 are omitted.

Furthermore, phase modulated the effect of feedback valence on the FRN as quantified by the MAC (Table 3). Feedback valence had a significant effect in both phases, but more pronounced in the late follicular ($F(1, 16) = 37.38$, $p < .001$, $\eta_p^2 = .70$) relative to the late luteal phase ($F(1, 16) = 34.98$, $p < .001$, $\eta_p^2 = .69$; Fig. 2). Separate analyses per feedback valence suggested that this phase effect especially concerned the processing of misses ($F(1, 16) = 4.85$, $p = .043$, $\eta_p^2 = .23$), with a larger FRN in the late follicular compared to the late luteal phase, and not of hits ($F < 1$, n.s.), although the former effect as such was not significant after correction for multiple comparisons. Phase had no significant effect on the FRN-surrounding peaks (i.e., P200 or P300).

3.5. Oscillatory power results

3.5.1. Cue-related power

Alpha power reductions were larger following potential-reward compared to no-reward cues (Table 2, Fig. 6; Fig. S3). Phase did not modulate this reward condition effect on alpha power. In addition, stress induction did not affect alpha power.

Exploratory analysis. Cue-related theta power increases were larger following no-reward relative to potential-reward cues ($F(1, 16) = 13.95$, $p = .002$, $\eta_p^2 = .47$), in contrast with feedback-related theta power increases (see below).

3.5.2. Feedback-related power

Visual inspection of Fig. 7 suggests the presence of a feedback valence effect in the late follicular phase on theta power, at least during the control condition, and the absence of a feedback valence effect in the late luteal phase, but the pertaining feedback valence by stress induction by phase interaction did not reach significance (Table 3).

In contrast with feedback valence, reward condition did have a significant effect on theta power, with larger increases in potential-reward versus no-reward trials (Table 3, Fig. 7; Fig. S4). However, this reward condition effect depended on the combination of phase and stress induction. In the late follicular phase (reward condition: $F(1, 16) = 4.85$, $p = .043$, $\eta_p^2 = .23$; reward condition by stress: $F(1, 16) = 6.23$, $p = .024$, $\eta_p^2 = .28$), reward condition had a significant effect during the stress ($F(1, 16) = 9.28$, $p = .008$, $\eta_p^2 = .37$), but not during the control condition ($F < 1$, n.s.). In the late luteal phase, reward condition had an effect in both stress induction conditions (reward condition: $F(1, 16) = 9.08$, $p = .008$, $\eta_p^2 = .36$; reward condition by stress induction: $F < 1$, n.s.).

4. Discussion

Aim of the present study was to investigate the combined effects of menstrual cycle phase and acute stress on reward-related pro-

Fig. 7. Feedback-related topographical maps and line plots of theta relative power (4–7 Hz). (A) Topographical map of the difference between miss and hit trials (300–600 ms post-feedback). (B) Line plot of theta relative power at Fz as a function of feedback valence and stress induction, for the late follicular (top) and late luteal phases (bottom). Solid lines represent hit trials; dashed lines represent miss trials; black lines represent the control condition; red lines represent the stress condition. Theta power increases were not significantly larger following misses compared to hits. The feedback valence by stress induction by phase interaction did not reach significance. (C) Topographical map of the difference between potential-reward and no-reward trials (300–600 ms post-feedback). (D) Line plots of theta relative power at Fz as a function of reward condition and stress induction, for the late follicular (top) and late luteal phases (bottom). Thick lines represent potential-reward trials; thin lines represent no-reward trials; black lines represent the control condition; red lines represent the stress condition. In general, theta power increases were larger in potential-reward relative to no-reward trials, but this effect depended on the combination of phase and stress induction condition. In the late follicular phase, reward condition had a significant effect in the stress condition only, whereas in the late luteal phase, reward condition had a significant effect in both stress and control conditions.

cessing. Participants were tested during the late follicular and late luteal phases, as verified by salivary progesterone determination, and performed in both control and stress conditions. The stress induction procedure yielded mild to moderate stress responses, which did not significantly differ between menstrual cycle phases. During the MID task, participants responded faster to targets in potential-reward relative to no-reward trials, confirming that the task was successful in eliciting motivated behavior. We found evi-

dence for both independent and interaction effects of menstrual cycle phase and stress induction on reward-related brain activity. In this section, we will first discuss our findings with regard to phase effects during reward anticipation and feedback. Then, we will discuss our findings concerning acute stress effects in late follicular and late luteal phases. Finally, we will discuss limitations of the present study.

4.1. Effects of phase during reward anticipation and feedback

4.1.1. Late follicular phase: heightened sensitivity to valence of feedback

Phase modulated brain activity during the stage of feedback. More specifically, the effect of feedback valence on the FRN as quantified by the MAC was more pronounced in the late follicular relative to the late luteal phase. The FRN is thought to reflect the signaling of unfavorable outcomes and a need for increased cognitive control (Cohen et al., 2011; Van de Vijver et al., 2011). In accordance with this notion, we found larger FRN amplitudes following misses relative to hits. The additional finding that this valence effect was more pronounced in the late follicular relative to the late luteal phase, suggests that the signaling of unfavorable (versus favorable) outcomes was stronger in the late follicular phase.

Our findings with regard to the processing of feedback valence are in accordance with fMRI studies supporting a potentiating influence of estradiol on the brain's reward system in the presence of low progesterone levels, during reward delivery (Dreher et al., 2007; Thomas et al., 2014). In addition, our findings are in line with findings from a study employing a hormone manipulation reducing estradiol levels in women, to some extent mimicking the late luteal phase, resulting in a decreased responsivity to the magnitude of rewards (Macoveanu et al., 2016).

4.1.2. Late luteal phase: heightened sensitivity to reward prospect

In contrast with the larger sensitivity to the valence of feedback in the late follicular compared to the luteal phase, we found that the late luteal relative to the late follicular phase is associated with an increased sensitivity to reward prospect, although not under stress. Theta oscillations in the frontal network are thought to play an important role in signaling negative outcomes and implementing behavioral adaptations (Van de Vijver et al., 2011), and previous studies have indeed reported larger feedback-related theta power increases after negative relative to positive outcomes (e.g., Banis et al., 2014; Cohen et al., 2007). In the present study employing the MID task, we did not find a significant effect of feedback valence. We did find larger theta power increases in potential-reward compared to no-reward trials, suggesting that the level of communication in the frontal network following the reception of feedback is increased when a reward is at stake. These findings indicate that reward condition had a greater influence on feedback-related theta power than feedback valence, in the present study.

This reward condition effect on feedback-related theta power depended on the combination of phase and stress induction condition. Potential-reward-related increases in performance monitoring during the late luteal phase were present in both stress induction conditions. In the late follicular phase, this effect was limited to the stress condition. These findings indicate that the late luteal relative to the late follicular phase is associated with a heightened sensitivity to reward condition, under control conditions. Similarly, Ossewaarde et al. (2011b) reported enhanced ventral striatal responses in the late luteal compared to the late follicular phase, during reward anticipation. These authors proposed that the enhanced sensitivity to reward prospect might be related to the late luteal drop in hormone levels, decreasing endogenous DA activity, causing increased DA release following reward cues. However, our findings are in contrast with the earlier mentioned studies supporting a potentiating influence of estradiol on the brain's reward system, during reward anticipation as well (Dreher et al., 2007; Thomas et al., 2014).

4.1.3. Conclusion

Our findings provide evidence that female gonadal hormone levels influence reward-related processing, and that these effects may differ between specific psychological components of reward-

related processing. Whereas the late follicular phase seems to be associated with an increased sensitivity to the valence of feedback, the late luteal phase appears to be related to a heightened sensitivity to the prospect of reward. As Berridge, Robinson, and Aldridge (2009) pointed out, reward-related processing can be dissected into anticipatory ("wanting"), consummatory ("liking"), and learning components, which are associated with distinct neurobiological substrates. The factor reward condition in the current study might be linked to the "wanting" component; the factor feedback valence might be related to the "liking" component; and both factors might be related to the learning component. The neurobiological substrates underlying these different components may be differentially affected by gonadal hormone levels. Consequently, the steeply declining estradiol levels in the late luteal phase might cause an increase in wanting, whereas the high estradiol combined with low progesterone levels in the late follicular phase might cause an increase in liking. The reported increase in depression risk in women, during stages of steep decline in hormonal levels (Deecher et al., 2008), may be related to a loss of distinction between positive and negative stimuli. Consequently, this might result in a lower appreciation of normally rewarding stimuli, that is anhedonia, which is a core symptom of depression (Russo & Nestler, 2013).

4.2. Effects of acute stress in late luteal and late follicular phases

4.2.1. Subjective and physiological stress responses: no support for an increased stress sensitivity in the late luteal phase

In contrast with our hypothesis, we did not find significant differences in subjective and physiological stress responses between the late luteal and late follicular phases. Therefore, we cannot confirm that the high estradiol levels in the late follicular phase attenuate stress reactivity relative to the dropping levels in the late luteal phase. Previous studies did report increased *psychophysiological* reactivity to laboratory stressors in the luteal relative to the follicular phase (Kirschbaum et al., 1999; Lustyk et al., 2010; Terman et al., 1991). Notably, the latter studies compared approximately *midluteal* and *midfollicular* phases, while we compared *late luteal* and *late follicular* phases. Given the evidence that progesterone may stimulate HPA axis activity (Roca et al., 2003), the enhanced stress response in the midluteal phase might be explained by the peaking levels of progesterone, during this phase. These differential findings indicate that stress-sensitivity may fluctuate across the menstrual cycle, but that phases of heightened sensitivity are confined to specific subphases, characterized by specific hormonal conditions.

An enhanced stress sensitivity in the late luteal phase might be limited to women with PMDD (Epperson et al., 2007). This is in line with a recent review concluding that clear evidence for a specific premenstrual mood syndrome in *healthy* women is lacking (Romans et al., 2012).

4.2.2. Acute stress affects attentional preparation during reward anticipation

We found that stress affected brain activity during reward anticipation, which is in line with previous studies (Dedovic et al., 2009; Ossewaarde et al., 2011a; Starcke & Brand, 2012). However, in contrast with our expectations of an increased sensitivity to stress during the late luteal compared to the late follicular phase (e.g., Kirschbaum et al., 1999; Lustyk et al., 2010), the results indicated no significant phase differences in the way stress affected attentional preparation to upcoming targets, as reflected in the CNV.

Stress decreased CNV amplitudes in this early interval, indicating impaired attentional orienting to subsequent targets under stress (Grent-'t-Jong & Woldorff, 2007). This is in accordance with the notion that stress especially impairs higher-order functions,

such as top-down attentional control (Arnstén & Goldman-Rakic, 1998). However, stress did not affect RTs, whereas reward condition did, which is possibly related to the difference in the respective effect sizes on the CNV ($\eta_p^2 = .23$ versus $\eta_p^2 = .52$).

4.2.3. Acute stress increases impact of reward condition on feedback processing

In addition to the effect of acute stress on attentional preparation during reward anticipation, acute stress modulated brain activity during the processing of feedback information. FRN amplitudes following hits were larger in no-reward trials than in potential-reward trials, especially in the stress compared to the control condition. Notably, this finding only applied to the FRN as quantified by the MAC measure, taking into account surrounding peaks, and not to the MA measure. Unfortunately, it is impossible to precisely discriminate between overlapping components using the EEG technique (Luck, 2014), and processes underlying the FRN might already start and continue in earlier and later time windows, respectively. In the present study, the MAC measure showed a result pattern which was opposite to that of the frontocentral P300, the latter showing a larger effect of reward condition on the processing of hits in the *control* relative to the stress condition. One cannot be sure whether the observed interactions on the MAC were caused by FRN-related activity or by P300-related activity. However, the P300 was maximal at parietal electrodes, indicating that the FRN and P300 reflect different processes.

Although acute stress impaired attentional preparation during reward anticipation, it seemed to enhance the impact of reward condition on the processing of hits. Nevertheless, stress did not influence *performance* monitoring per se, that is, monitoring whether targets were hit or not. Participants seemed to be more sensitive to the actual delivery of reward following hits rather than being more focused on hitting versus missing targets, when exposed to stress. This interpretation seems to be in accordance with behavioral evidence showing increased consumption of rewarding substances under stressful circumstances (e.g., Koob, 2008; Rutters et al., 2009; Uhart & Wand, 2009).

As proposed by Maier, Makwana, and Hare (2015), exposure to acute stress may impair self-controlled decisions in favor of actions leading to immediate reward, by increasing the influence of immediately rewarding attributes and decreasing the potency of regions promoting goal-directed behaviors. The stress-related increase in sensitivity to reward prospect during the processing of hits, in the present study, is in line with this theory. Nevertheless, we did not find evidence for stress-related impairments of performance monitoring.

Previous neuroimaging/EEG studies, however, have found a decreased sensitivity to feedback information in stress versus control conditions (Banis et al., 2014; Banis & Lorist, 2012; Porcelli et al., 2012). In two preceding studies, we examined the impact of acute noise stress on feedback-related EEG measures, employing a simple gambling task (Banis et al., 2014; Banis & Lorist, 2012). In both studies, we found evidence for modulation of the FRN by acute stress exposure, either by decreasing feedback valence and magnitude effects on the FRN (Banis & Lorist, 2012) or by a general decrease in FRN amplitude (Banis et al., 2014). In the latter study, we also investigated feedback-related theta power and found smaller increases in the stress relative to the control condition. These stress-related modulations of the FRN and feedback-related theta power were not replicated in the present study. This discrepancy might be explained by the employment of different tasks, which provided different contexts in which feedback was processed. The presence of the factor reward condition and/or the absence of loss trials in the MID task seem to have had a strong influence on brain activity, both during reward anticipation and feedback stages, as reflected in effects of reward condition on both cue-

related and feedback-related theta power, and the absence of a significant effect of feedback valence on feedback-related theta power. These findings suggest that during the MID task, evaluation in terms of positive or negative prospects already takes place during the stage of reward anticipation. However, our exploratory analysis of cue-related theta power did not show stress-related modulations either, which indicates that stress did not impair evaluation of prospects.

4.3. Limitations

An important limitation of the present study was the small number of participants, which was sufficient to detect large effect sizes only and limits the reliability of our findings. Therefore, our conclusions should be interpreted with care and require replication. Two other limitations concern the measurement of hormone levels, for menstrual cycle phase verification. First, in order to measure progesterone levels, we collected *single* saliva samples during both experimental sessions, while it is preferable to sample more often, as salivary hormone levels undergo strong fluctuations. Second, we did not measure estradiol levels, which is needed for a more precise estimation of the timing of sessions within the menstrual cycle. Finally, although our stress induction procedure was successful in eliciting stress, it did not result in the high levels of stress as induced by motivated performance tasks combining uncontrollability and social evaluative threat (Dickerson & Kemeny, 2004). The employment of stronger stressors might reveal phase-specific stress effects.

4.4. Conclusion

To summarize, we found evidence for both independent and interaction effects of menstrual cycle phase and stress induction on reward-related brain activity. Phase modulated the sensitivity to feedback valence, with a stronger signaling of unfavorable performance outcomes in the late follicular relative to the late luteal phase. In contrast, the late luteal relative to the late follicular phase was associated with an increased sensitivity to reward condition, with enhanced performance monitoring in potential-reward relative to no-reward trials, in the control condition. Stress impaired attentional preparation during reward anticipation, but increased the influence of reward condition on the processing of favorable performance outcomes. We found no evidence for an increased sensitivity to stress during the late luteal relative to the late follicular phase.

Acknowledgments

This research was supported by an NWO grant (400-07-001). We thank Dr. E. J. Hermans, at the Radboud University Nijmegen, for supplying us with the movie clips. We thank Dr. A. M. van Roon, at the University Medical Center Groningen, for his assistance with the cardiovascular analyses.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.biopsycho.2017.02.005>.

References

- Arnstén, A. F. T., & Goldman-Rakic, P. (1998). Noise stress impairs prefrontal cortical cognitive function in monkeys: Evidence for a hyperdopaminergic mechanism. *Archives of General Psychiatry*, 55(4), 362–368. <http://dx.doi.org/10.1001/archpsyc.55.4.362>

- Banis, S., & Lorist, M. M. (2012). Acute noise stress impairs feedback processing. *Biological Psychology*, 91(2), 163–171. <http://dx.doi.org/10.1016/j.biopsycho.2012.06.009>
- Banis, S., Geerligs, L., & Lorist, M. M. (2014). Acute stress modulates feedback processing in men and women: Differential effects on the feedback-related negativity and theta and beta power. *Public Library Of Science*, 9(4), e95690. <http://dx.doi.org/10.1371/journal.pone.0095690>
- Bayer, J., Bandurski, P., & Sommer, T. (2013). Differential modulation of activity related to the anticipation of monetary gains and losses across the menstrual cycle. *European Journal of Neuroscience*, 38, 3519–3526.
- Becker, J. B., Arnold, A. P., Berkley, K. J., Blaustein, J. D., Eckel, L. A., Hampson, E., ... & Young, E. (2005). Strategies and methods for research on sex differences in brain and behavior. *Endocrinology*, 146(4), 1650–1673.
- Becker, J. B. (2009). Sexual differentiation of motivation: A novel mechanism? *Hormones and Behavior*, 55(5), 646–654.
- Berridge, K. C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: 'liking', 'wanting', and learning. *Current Opinion in Pharmacology*, 9(1), 65–73. <http://dx.doi.org/10.1016/j.coph.2008.12.014>
- Broyd, S. J., Richards, H. J., Helps, S. K., Chronaki, G., Bamford, S., & Sonuga-Barke, E. (2012). An electrophysiological monetary incentive delay (e-MID) task: A way to decompose the different components of neural response to positive and negative monetary reinforcement. *Journal of Neuroscience Methods*, 209(1), 40–49. <http://dx.doi.org/10.1016/j.jneumeth.2012.05.015>
- Capotosto, P., Babiloni, C., Romani, G. L., & Corbetta, M. (2009). Frontoparietal cortex controls spatial attention through modulation of anticipatory alpha rhythms. *The Journal of Neuroscience*, 29(18), 5863–5872.
- Chabbert Buffet, N., Djakoure, C., Christin Maitre, S., & Bouchard, P. (1998). Regulation of the human menstrual cycle. *Frontiers in Neuroendocrinology*, 19(3), 151–186.
- Cohen, M. X., Elger, C. E., & Ranganath, C. (2007). Reward expectation modulates feedback-related negativity and EEG spectra. *Neuroimage*, 35(2), 968–978. <http://dx.doi.org/10.1016/j.neuroimage.2006.11.056>
- Cohen, M. X., Wilmes, K., & Van de Vijver, I. (2011). Cortical electrophysiological network dynamics of feedback learning. *Trends in Cognitive Sciences*, 15(12), 558–566. <http://dx.doi.org/10.1016/j.tics.2011.10.004>
- Cohen, M. X. (2014). *Analyzing neural time series data: theory and practice*. MIT Press: Cambridge, MA.
- Davidson, L., Vistisen, B., & Astrup, A. (2007). Impact of the menstrual cycle on determinants of energy balance: A putative role in weight loss attempts. *International Journal of Obesity*, 31(12), 1777–1785.
- Dedovic, K., D'Aguiar, C., & Pruessner, J. C. (2009). What stress does to your brain: A review of neuroimaging studies. *Canadian Journal of Psychiatry*, 54(1), 6–15.
- Deecher, D., Andree, T. H., Sloan, D., & Schechter, L. E. (2008). From menarche to menopause: Exploring the underlying biology of depression in women experiencing hormonal changes. *Psychoneuroendocrinology*, 33(1), 3–17.
- Di Bernardi Luft, C., Nolte, G., & Bhattacharya, J. (2013). High-learners present larger mid-frontal theta power and connectivity in response to incorrect performance feedback. *The Journal of Neuroscience*, 33(5), 2029–2038. <http://dx.doi.org/10.1523/JNEUROSCI.2565-12.2013>
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355–391.
- Dreher, J., Schmidt, P. J., Kohn, P., Furman, D., Rubinow, D., & Berman, K. F. (2007). Menstrual cycle phase modulates reward-related neural function in women. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 104(7), 2465–2470.
- Dye, L., & Blundell, J. E. (1997). Menstrual cycle and appetite control: Implications for weight regulation. *Human Reproduction (Oxford, England)*, 12(6), 1142–1151.
- Edwards, S., Clow, A., Evans, P., & Hucklebridge, F. (2001). Exploration of the awakening cortisol response in relation to diurnal cortisol secretory activity. *Life Sciences*, 68(18), 2093–2103.
- Epperson, C. N., Pittman, B., Czarkowski, K. A., Stiklus, S., Krystal, J. H., & Grillon, C. (2007). Luteal-phase accentuation of acoustic startle response in women with premenstrual dysphoric disorder. *Neuropsychopharmacology*, 32, 2190–2198.
- Evans, S. M., & Levin, F. R. (2011). Response to alcohol in women: Role of the menstrual cycle and a family history of alcoholism. *Drug and Alcohol Dependence*, 114(1), 18–30.
- Gehring, W. J., & Willoughby, A. R. (2002). The medial frontal cortex and the rapid processing of monetary gains and losses. *Science*, 295(5563), 2279–2282. <http://dx.doi.org/10.1126/science.1066893>
- Goldstein, J. M., Jerram, M., Abbs, B., Whitfield-Gabrieli, S., & Makris, N. (2010). Sex differences in stress response reactivity activation dependent on female hormonal cycle. *The Journal of Neuroscience*, 30(2), 431–438.
- Graham, J. W. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology*, 60, 549–576.
- Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography & Clinical Neurophysiology*, 55(4), 468–484. [http://dx.doi.org/10.1016/0013-4694\(83\)90135-9](http://dx.doi.org/10.1016/0013-4694(83)90135-9)
- Grent-t-jong, T., & Woldorff, M. G. (2007). Timing and sequence of brain activity in top-down control of visual-spatial attention. *Plos Biology*, 5(1), e12.
- Henckens, M. J. A. G., Hermans, E. J., Pu, Z., Joëls, M., & Fernández, G. (2009). Stressed memories: How acute stress affects memory formation in humans. *The Journal of Neuroscience*, 29(32), 10111–10119.
- Hsu, Y. (2005). On measuring the minimum detection time: A simple reaction time study in the time estimation paradigm. *British Journal of Mathematical and Statistical Psychology*, 58(2), 259–284.
- Jackson, L., Robinson, T. E., & Becker, J. B. (2006). Sex differences and hormonal influences on acquisition of cocaine self-administration in rats. *Neuropsychopharmacology*, 31(1), 129–138.
- Jacobs, E. G., Holsen, L. M., Lancaster, K., Makris, N., Whitfield-Gabrieli, S., Remington, A., ... & Goldstein, J. M. (2015). 17 β -Estradiol differentially regulates stress circuitry activity in healthy and depressed women. *Neuropsychopharmacology*, 40, 566–576.
- Kajantie, E., & Phillips, D. I. W. (2006). The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology*, 31, 151–178.
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamic-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154–162.
- Knutson, B., Westdorp, A., Kaiser, E., & Hommer, D. (2000). fMRI visualization of brain activity during a monetary incentive delay task. *Neuroimage*, 12(1), 20–27.
- Koob, G. F. (2008). A role for brain stress systems in addiction. *Neuron*, 59(1), 11–34. <http://dx.doi.org/10.1016/j.neuron.2008.06.012>
- Luck, S. J. (2014). *An Introduction to the Event-Related Potential Technique* (2nd ed.). Cambridge, MA: MIT Press.
- Lustyk, M. K., Olson, K. C., Gerrish, W. G., Holder, A., & Widman, L. (2010). Psychophysiological and neuroendocrine responses to laboratory stressors in women: implications of menstrual cycle phase and stressor type. *Biological Psychology*, 83(2), 84–92.
- Macoveanu, J., Henningson, S., Pinborg, A., Jensen, P., Knudsen, G. M., Frokjaer, V. G., & Siebner, H. R. (2016). Sex-steroid hormone manipulation reduces brain response to reward. *Neuropsychopharmacology*, 41, 1057–1065.
- Maier, S. U., Makwana, A. B., & Hare, T. A. (2015). Acute stress impairs self-control in goal-directed choice by altering multiple functional connections within the brain's decision circuits. *Neuron*, 87, 621–631.
- McEwen, B. (2002). Estrogen actions throughout the brain. *Recent Progress in Hormone Research*, 57, 357–384.
- Mulder, L. J. M., De Waard, D., & Brookhuis, K. A. (2005). Estimating mental effort using heart rate and heart rate variability. In N. Stanton, A. Hedge, K. A. Brookhuis, E. Salas, & H. Hendrick (Eds.), *Handbook of human factors and ergonomics methods*. Boca Raton: CRC Press [pp. 20–1–20–8].
- Mulder, L. J. (1992). Measurement and analysis methods of heart rate and respiration for use in applied environments. *Biological Psychology*, 34(2–3), 205–236.
- Mushtaq, F., Stoet, G., Bland, A. R., & Schaefer, A. (2013). Relative changes from prior reward contingencies can constrain brain correlates of outcome monitoring. *Public Library Of Science*, 8(6), e66350. <http://dx.doi.org/10.1371/journal.pone.0066350>
- Oostenveld, R., Fries, P., Maris, E., & Schoffelen, J. (2011). FieldTrip: Open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Computational Intelligence and Neuroscience*, 2011(156869) <http://dx.doi.org/10.1155/2011/156869156869>
- Ossewaarde, L., Hermans, E. J., Van Wingen, G. A., Kooijman, S. C., Johansson, I., Bäckström, T., & Fernández, G. (2010). Neural mechanisms underlying changes in stress-sensitivity across the menstrual cycle. *Psychoneuroendocrinology*, 35(1), 47–55.
- Ossewaarde, L., Qin, S., Van Marle, H. J. F., Van Wingen, G. A., Fernández, G., & Hermans, E. J. (2011). Stress-induced reduction in reward-related prefrontal cortex function. *Neuroimage*, 55(1), 345–352.
- Ossewaarde, L., Van Wingen, G. A., Kooijman, S. C., Bäckström, T., Fernández, G., & Hermans, E. J. (2011). Changes in functioning of mesolimbic incentive processing circuits during the premenstrual phase. *Social Cognitive and Affective Neuroscience*, 6(5), 612–620. <http://dx.doi.org/10.1093/scan/nsq071>
- Pfabigan, D. M., Sailer, U., & Lamm, C. (2015). Size does matter! Perceptual stimulus properties affect event-related potentials during feedback processing. *Psychophysiology*, 52(9), 1238–1247. <http://dx.doi.org/10.1111/psyp.12458>
- Porcelli, A. J., Lewis, A. H., & Delgado, M. R. (2012). Acute stress influences neural circuits of reward processing. *Frontiers in Neuroscience*, 6, 157. <http://dx.doi.org/10.3389/fnins.2012.00157>
- Protopopescu, X., Pan, H., Altemus, M., Tiescher, O., Polanczyk, M., McEwen, B., ... & Stern, E. (2005). Orbitofrontal cortex activity related to emotional processing changes across the menstrual cycle. *Proceedings of the National Academy of Sciences of the United States of America*, 102(44), 16060–16065.
- Richardson, J. T. E. (2011). Eta squared and partial eta squared as measures of effect size in educational research. *Educational Research Review*, 6, 135–147.
- Roca, C. A., Schmidt, P. J., Altemus, M., Deuster, P., Danaceau, M. A., Putnam, K., & Rubinow, D. R. (2003). Differential menstrual cycle regulation of hypothalamic-pituitary-adrenal axis in women with premenstrual syndrome and controls. *The Journal of Clinical Endocrinology and Metabolism*, 88(7), 3057–3063.
- Romans, S. E., Kreindler, D., Asllani, E., Einstein, G., Laredo, S., Levitt, A., ... & Stewart, D. E. (2012). Mood and the menstrual cycle. *Psychotherapy and Psychosomatics*, 82(1), 53–60.
- Russo, S. J., & Nestler, E. J. (2013). The brain reward circuitry in mood disorders. *Nature Reviews Neuroscience*, 14(9), 609–625.

- Rutters, F., Nieuwenhuizen, A. G., Lemmens, S. G. T., Born, J. M., & Westerterp-Plantenga, M. S. (2009). Acute stress-related changes in eating in the absence of hunger. *Obesity*, 17(7), 72–77.
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., . . . & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59, 22–33.
- Starcke, K., & Brand, M. (2012). Decision making under stress: A selective review. *Neuroscience and Biobehavioral Reviews*, 36(4), 1228–1248. <http://dx.doi.org/10.1016/j.neubiorev.2012.02.003>
- Steiner, M., Dunn, E., & Born, L. (2003). Hormones and mood: From menarche to menopause and beyond. *Journal of Affective Disorders*, 74(1), 67–83.
- Steiner, M., Macdougall, M., & Brown, E. (2003). The premenstrual symptoms screening tool (PSST) for clinicians. *Archives of Women's Mental Health*, 6(3), 203–209.
- Terner, J. M., & De Wit, H. (2006). Menstrual cycle phase and responses to drugs of abuse in humans. *Drug and Alcohol Dependence*, 84, 1–13.
- Tersman, Z., Collins, A., & Eneroth, P. (1991). Cardiovascular responses to psychological and physiological stressors during the menstrual cycle. *Psychosomatic Medicine*, 53(2), 185–197.
- Thomas, J., Météreau, E., Déchaud, H., Pugeat, M., & Dreher, J. (2014). Hormonal treatment increases the response of the reward system at the menopause transition: A counterbalanced randomized placebo-controlled fMRI study. *Psychoneuroendocrinology*, 50, 167–180.
- Thut, G., Nietzel, A., Brandt, S. A., & Pascual-Leone, A. (2006). Alpha-band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 26(37), 9494–9502.
- Uhart, M., & Wand, G. S. (2009). Stress, alcohol and drug interaction: An update of human research. *Addiction Biology*, 14(1), 43–64.
- Ulrich, R., & Miller, J. (1994). Effects of truncation on reaction time analysis. *Journal of Experimental Psychology: General*, 123(1), 34–80.
- Van Boxtel, G. J. M., & Böcker, K. B. E. (2004). Cortical measures of anticipation. *Journal of Psychophysiology*, 18(2–3), 61–76.
- Van Buuren, S. (2007). Multiple imputation of discrete and continuous data by fully conditional specification. *Statistical Methods in Medical Research*, 16(3), 219–242.
- Van Roon, A. M. (1998). *Short-term cardiovascular effects of mental tasks*. Groningen: University of Groningen [Doctoral thesis].
- Van Selst, M., & Jolicoeur, P. (1994). A solution to the effect of sample size on outlier elimination. *The Quarterly Journal of Experimental Psychology A: Human Experimental Psychology*, 47A(3), 631–650.
- Van de Vijver, I., Ridderinkhof, K. R., & Cohen, M. X. (2011). Frontal oscillatory dynamics predict feedback learning and action adjustment. *Journal of Cognitive Neuroscience*, 23(12), 4106–4121. <http://dx.doi.org/10.1162/jocn.a.00110>
- Van den Berg, B., Krebs, R. M., Lorist, M. M., & Woldorff, M. G. (2014). Utilization of reward-prospect enhances preparatory attention and reduces stimulus conflict. *Affective & Behavioral Neuroscience*, 14(2), 561–577. <http://dx.doi.org/10.3758/s13415-014-0281-z>
- Walter, W. G., Cooper, R., Aldridge, V. J., McCallum, W. C., & Winter, A. L. (1964). Contingent Negative Variation: an electric sign of sensorimotor association and expectancy in the human brain. *Nature*, 203, 380–384.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54(6), 1063–1070. <http://dx.doi.org/10.1037/0022-3514.54.6.1063>
- Worden, M. S., Foxe, J. J., Wang, N., & Simpson, G. V. (2000). Anticipatory biasing of visuospatial attention indexed by retinotopically specific alpha-band electroencephalography increases over occipital cortex. *The Journal of Neuroscience*, 20(6), RC63.