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Short-term food deprivation increases amplitudes of heartbeat-evoked potentials

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Abstract

Nutritional state (i.e., fasting or nonfasting) may affect the processing of interoceptive signals, but mechanisms underlying this effect remain unclear. We investigated 16 healthy women on two separate days: when satiated (standardized food intake) and after an 18-h food deprivation period. On both days, heartbeat-evoked potentials (HEPs) and cardiac and autonomic nervous system activation indices (heart rate, normalized low frequency heart rate variability [nLF HRV]) were assessed. The HEP is an EEG pattern that is considered an index of cortical representation of afferent cardiovascular signals. Average HEP activity (R wave +455–595 ms) was enhanced during food deprivation compared to normal food intake. Cardiac activation did not differ between nutritional conditions. Our results indicate that short-term food deprivation amplifies an electrophysiological correlate of the cortical representation of visceral-afferent signals originating from the cardiovascular system. This effect could not be attributed to increased cardiac activation, as estimated by heart rate and nLF HRV, after food deprivation.

Descriptors: Eating behavior, Fasting, Food deprivation, Interoception, Heartbeat detection, Heartbeat-evoked potentials (HEPs), Nutrition, Symptom perception, Visceral perception

Neurovisceral processes play an important role in eating behavior, hunger, and satiety. On the one hand, high interoceptive accuracy and healthy eating behavior are strongly associated, indicated by substantial correlations ($r = .45–.53$) with self-reported intuitive eating (Herbert, Blechert, Hautzinger, Matthias, & Herbert, 2013). On the other hand, individuals with pathological eating behavior, such as patients with anorexia nervosa, show lower interoceptive accuracy than noneating disordered individuals (Pollatos et al., 2008), and altered activity in the insular cortex (Oberndorfer et al., 2013; Strigo et al., 2013), an important cortical region for the processing of interoceptive signals (Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004). One possible conclusion is that high interoceptive accuracy represents a trait that predicts healthy eating behavior and may be a protective factor against the development of eating disorders. However, the correlative and quasiexperimental designs of previous studies do not allow for interpretations on the causality of this effect.

Experimental manipulations of nutrition status by 24 h of food deprivation have been shown to increase interoceptive accuracy as indicated by a heartbeat perception task compared to a condition of normal food consumption (Herbert, Herbert et al., 2012). Nevertheless, short-term food deprivation induces a variety of endocrinological and metabolic changes, involving hormones such as leptin (white adipose tissue), ghrelin (stomach), or insulin (pancreas), whose concentrations are transmitted to the hypothalamus and the nucleus tractus solitarius (Neary, Goldstone, & Bloom, 2004). Since these structures are also involved in the regulation of autonomic nervous system activity, short-term fasting may also be accompanied by an increase of sympathetic tone (Landsberg, 2006; Rayner & Trayhurn, 2001). It has been suggested, therefore, that an increase in sympathetic tone, reflected in higher heart rate and contractility, as well as lower normalized heart rate variability (HRV) in the high-frequency spectrum and a shorter preejection period, is responsible for the enhancement of interoceptive accuracy in food deprivation (Herbert, Herbert et al., 2012).

Heartbeat perception tasks represent a common and repeatedly validated method to index interoceptive accuracy. One could argue that the accuracy in these tasks may be limited to sensitivity for

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cardiac sensations, but it was previously shown that there is a moderate correlation ($r = .50–.51$) between the sensitivity for the perception of cardiovascular and gastrointestinal sensations (Herbert, Muth, Pollatos, & Herbert, 2012; Whitehead & Drescher, 1980). Nevertheless, the study by Herbert, Herbert, and colleagues (2012) leaves two questions open, which may be important to understand the mechanisms behind the relationship between food deprivation and interoception. First, it remains unclear whether the reported effects are due to peripheral processes or central nervous system (CNS) processes. Peripheral processes would involve increased stimulation of cardiovascular interoceptors (e.g., arterial baroreceptors) due to sympathetic activation. Alternatively, food deprivation may only change CNS representations of interoceptive signals. Secondly, heartbeat perception tasks may not differentiate if food deprivation induces a physiological process that amplifies genuine CNS representations, or feelings of hunger may select and allocate attention towards bodily processes. Following the model of interoception by Vaitl (1996), (a) the stimulation of interoceptors, (b) CNS representation of interoceptive signals, and (c) conscious interoceptive awareness represent three hierarchical levels of interoception that are all collectively reflected by heartbeat perception tasks. The aim of the current study was to address both questions.

Prolonged short-term food deprivation stimulates lipolysis, a process mainly initiated by the sympathetic nervous system (Dodt, Lonnroth, Wellhoner, Fehm, & Elam, 2003), which mobilizes energy resources. Food deprivation of up to 74 h, for example, has been demonstrated to be associated with increased sympathetic activity (Chan, Mietus, Raciti, Goldberger, & Mantzoros, 2007; Webber & Macdonald, 1994). In contrast, fasting of short duration (14–22 h) is not sufficient to provoke sympathetic activation in the adipose tissue (Horowitz et al., 1999). Nevertheless, a 6-h food deprivation period is enough to provoke feelings of craving, higher food consumption, and higher arousal ratings of food cues (Drobes et al., 2001). To investigate whether sympathetic activation is required for the increase of interoceptive accuracy, we therefore reduced the time interval of fasting compared to the study by Herbert, Herbert, and colleagues (2012) from 24 h to 18 h. We expected that this reduction would allow for the investigation of short-term fasting without engaging the sympathetic nervous system (Hypothesis 1).

To avoid artificial manipulation of participants' attention towards visceral signals, and to examine if food deprivation affects the genuine CNS representation of afferent signals from the cardiovascular system without the awareness to bodily sensations, we assessed heartbeat-evoked brain potentials (HEPs) during rest, without instructions. HEPs represent positive electrocortical potentials, which are related to the processing of cardiac signals, such as heartbeats. HEPs can be measured 250–600 ms after a cardiac R wave and have their largest amplitude over the right hemisphere (Leopold & Schandry, 2001; Pollatos & Schandry, 2004; Schandry, Sparrer, & Weitkunat, 1986). HEP amplitudes have been demonstrated to correlate with an individual's performance in heartbeat detection tasks (Katkin, Cestaro, & Weitkunat, 1991; Pollatos & Schandry, 2004; Schandry & Montoya, 1996), motivation to perform in those tasks (Weitkunat & Schandry, 1990), and attentional focus on heartbeats (Montoya, Schandry, & Muller, 1993; Schandry et al., 1986; Schandry & Weitkunat, 1990). Importantly, HEPs can also be found without the conscious perception of heartbeats. Since afferent signals from the cardiovascular system continuously reach cortical structures, HEP amplitudes assessed during rest can be interpreted as a psychophysiological indicator for CNS representation of cardiac interoceptive signals independent from active awareness to cardiac sensations (Schulz, Strelzyk et al., 2013; Shao, Shen, Wilder-Smith, & Li, 2011). We expected that food deprivation alters genuine CNS representation of afferent signals from the cardiovascular system (Hypothesis 2).

In a previous study, we observed generally higher HEP amplitudes during open than during closed eyes while resting, but this effect was superseded by an interaction effect of higher order (Schulz, Strelzyk et al., 2013). These differences in HEP amplitudes were probably due to the effects of cortisol infusion, which induced higher HEP amplitudes during open than during closed eyes. Nevertheless, in an attempt to empirically test the assumption that HEP amplitudes are not affected by open versus closed eyes per se (Hypothesis 3), we also included a comparison of HEP amplitudes both during open and closed eyes in the current study.

We investigated 16 healthy women in both a condition of standardized food intake and approximately 18 h of fasting. The participants underwent eight EEG and electrocardiography (ECG) measurement sessions during rest (with open and closed eyes) for the later calculation of HEPs. Furthermore, we assessed heart rate and normalized LF HRV as indices of sympathetic tone. We expected higher HEP amplitudes in the condition of food deprivation as compared to the normal food consumption (Hypothesis 2), but no effect of the food condition on indicators for sympathetic activation (Hypothesis 1).

Method

Participants

Twenty right-handed, healthy women were recruited via advertisement to take part in the study. To increase deceivability, participants from previous studies in our lab were not eligible. Participants were offered financial reimbursement for their effort. Medical status was carefully screened by a psychologist (DSFS). Exclusion criteria were acute or chronic physical or mental health complaints, smoking, or current medication. Body mass index was required to be within normal range for women (19–24 kg/m^2, according to the German Nutrition Society). Furthermore, scores on subscales of the Three-Factor Eating Questionnaire (Stunkard & Messick, 1985; German version: Pudel & Westenhöfer, 1989) were used as exclusion criteria for abnormal eating habits. All scores were in the low to medium range (dietary restraint: $M = 3.5$, $SD = 3.0$; disinhibition: $M = 5.5$, $SD = 3.2$; hunger: $M = 3.1$, $SD = 2.0$) of the German norms. All participants provided written informed consent and were made aware of their right to withdraw from participation in the study at any time without any negative consequences. The study procedure was approved by the ethics committee of the University of Trier. Due to technical problems, psychophysiological data of four participants were lost and were therefore excluded from further analysis. The remaining 16 women had a mean age of 22.6 years (range: 20–26), and none were taking oral contraceptives.

Experimental Procedure

Participants were tested in a state of normal food consumption (NC) and of food deprivation (FD) according to a randomized and counterbalanced order, to control for sequence effects. Both laboratory sessions were 1 month apart and at exactly the same time of day. The study was part of an extended protocol that has in part been reported elsewhere (Ferreira de Sá et al., 2014). Participants were randomly assigned to two groups (between variable): the

midfollicular phase of the menstrual cycle (FP, $n = 9$) or the midluteal phase (LP, $n = 7$). FP was considered from 6–10 days after the first day of menses, while LP was calculated from 6–10 days after ovulation (Li, Tsang, & Lui, 1999), which was measured with a luteinizing hormone urine test (Clearblue). Since sex hormones and menstrual cycle may affect eating behavior (Dye & Blundell, 1997; Johnson, Corrigan, Lemmon, Bergeron, & Crusco, 1994; Li et al., 1999), these behavioral changes may be associated with modulated perception of bodily states, such as hunger and satiety. The sessions took place between 11:30 and 14:00 to avoid increased variance from time-of-day effects on food intake (Stroebele & De Castro, 2004). For the FD condition, participants were instructed to have the last meal at 18:00 of the day prior to testing in order to achieve an average fasting period of approximately 18 h. In the NC condition, participants were asked to have a normal breakfast around 8 am; in addition, they received a standard snack (dark whole wheat bread with fresh cheese, garnished with chives) immediately after arriving in the laboratory. For standardization purposes, participants were instructed to arrive 60 min before starting the experiment on both experimental days, since in the NC condition extra time was needed for the standard snack and a pause period to diminish eating arousal.

A cover story was used to reinforce participant compliance: they were told that on the day of the experimental sessions a saliva sample would be collected to check their nutritional status, with loss of financial reimbursement if the required nutritional state was not achieved. On both experimental days, saliva samples were collected and, as an additional way to encourage compliance, participants were asked to recall their routine from waking until arriving at the experiment (diary methods; Stone, Kessler, & Haythornthwaite, 1991). Upon arrival in the laboratory, participants were asked to seat themselves in a comfortable chair, and, after 60 min of waiting time, electrodes for ECG, EEG, and electrooculogram (EOG) were attached. Participants were then asked to attend to the instructions given via a computer screen. On the first screen, participants rated subjective feelings of stress, arousal, current hunger, and motivation to eat using continuous visual analogue scales, ranging from 0 to 1,280 (full horizontal screen resolution), and anchored at the left margin with 0 (*not at all*) and at the right margin with 7 (*very much*). The participants were then instructed to focus on a cross in the middle of a black screen and to follow the instructions given by earphones.

The psychophysiological measurements (simultaneous EEG, EOG, and ECG) were separated into eight segments of 1-min length each, during which participants were instructed either to keep their eyes open (O) or closed (C) in an intra- and interindividually balanced order that alternated between and within the individual EEG measurement blocks (O-C-O-C-O-C-O-C or C-O-C-O-C-O-C-O). Participants were asked to relax and neither to speak nor to move during the EEG measurements.

EEG Measurement and Data Preprocessing

EEG was recorded continuously by 13 Ag/AgCl pin-type passive electrodes (Nihon Kohden EEG 4421G) with the Easy-Cap electrode system (Falk Minow Services). The electrodes F(7, 3, z, 4, 8), C(3, z, 4), P(3, z, 4), A(1, 2) were mounted according to the 10–20 system (Chatrian, Lettich, & Nelson, 1988) and referenced to linked mastoids. Bipolar horizontal and vertical EOG were recorded with Ag/AgCl electrodes. Electrode sites on the scalp and face were cleaned with alcohol and gently abraded before electrode placement. Impedances of the EEG electrodes were below 5 kOhm. The EEG signals were recorded with a hardware time constant of 5 s and a low-pass filter of 120 Hz. The signals were digitized at 512 Hz and stored for offline analysis. The EEG was digitally refiltered (band-pass: 0.1–35 Hz; 24 dB/octave) to minimize drifts and noise that were present in some data channels, and resampled to 256 Hz. Continuous EEG recordings were visually inspected. Epochs with nonstereotyped artifacts (e.g., electrode cable movements, swallowing, etc.) were excluded from further analysis. As described in earlier studies on heartbeat-evoked potentials (Pollatos, Kirsch, & Schandry, 2005; Pollatos & Schandry, 2004; Schulz, Strelzyk et al., 2013), eye blink correction was conducted using the Gratton-Coles algorithm (Gratton, Coles, & Donchin, 1983). All steps of EEG analysis were performed with Brain Vision Analyzer 2.0 (BrainProducts, Munich, Germany).

Calculation of Heartbeat-Evoked Potentials

R waves were automatically detected and manually confirmed in offline ECG signals. EEG data were segmented relative to the detected R waves in epochs ranging from 200 ms before the R wave to 1,000 ms after the R wave. Segments of R-wave-triggered EEG were averaged according to whether they were recorded during open or closed eyes, separately for each EEG measurement session. Since the duration of periods with open and closed eyes was kept constant (1 min per period, each four periods of open and closed eyes, total length: 8 min), the total number of heartbeats during the acquisition periods varies with the participants' actual heart rate ($M = 589$, $SD = 60$, heartbeats per session, range: 439–677). Up to 450 ms after the R wave, the HEP amplitude and the electrocardiac field partially overlap (Dirlich, Vogl, Plaschke, & Strian, 1997; Schandry et al., 1986). In the literature, there were different approaches described to overcome this possible confounding factor: in some studies, the electrocardiac field was eliminated using the Hjorth source derivation method (Pollatos et al., 2005; Pollatos & Schandry, 2004), others subtracted components possibly originating from the ECG via principal (Yuan, Yan, Xu, Han, & Yan, 2007) or independent component analysis (Terhaar, Viola, Bar, & Debener, 2012), and some newer studies limited the analysis on a time window of 455–595 ms after the R wave, during which the electrocardiac field is considered minimal (Gray et al., 2007; Schulz, Strelzyk et al., 2013). Here, we used the latter approach and analyzed HEP amplitudes during the previously established interval of R +455–595 ms. HEP amplitudes of F3 and F7, as well as F4 and F8, were collapsed to create an equal-sized 3 (frontal, central, parietal) × 3 (left, midline, right) electrode field on the scalp. To test whether a potential impact of food condition is specific to HEPs or may be a general effect on event-related brain potentials, we also derived mean activity in two control intervals of the same duration: one early interval (180–320 ms), during which the cortical processing of afferent cardiac signals and the electrocardiac field may overlap (Dirlich et al., 1997), and one late interval (860–1,000 ms), which is temporally located after the cortical processing of cardiac signals (Gray et al., 2007).

Cardiovascular Data

Electrodes for ECG measurement (ECG Tyco Healthcare H34SG Ag/AgCl electrodes of 45-mm diameter) were placed according to a standard lead II configuration. The ECG signal was high-pass filtered (0.05 Hz) and stored on an external disk (512 Hz). ECG data were analyzed with WinCPRS 1.160 software (Absolute Aliens Oy, Turku, Finland). Interbeat intervals were calculated

from the ECG and manually corrected, with a normal cycle RR-interval time series as output signal, of which mean heart rate data were derived. The spectral analysis of the RR-interval series was done using a fast Fourier transform (FFT) routine. The RR-interval time series was linearly interpolated and resampled with a sampling rate of 5 Hz, the resampled data were tapered using a Hanning window, and the windowed data zero padded to the next power of 2. The FFT spectrum was smoothed using a sliding triangular weighting function in order to increase the number of freedoms and thus improve the statistical relevance of the spectrum. The high frequency band (HF) was defined as 0.14 to 0.4 Hz, the low frequency band (LF) was defined as 0.06 to .013 Hz, whereas oscillations below 0.06 Hz were defined as very low frequency (VLF). LF was expressed in terms of normalized values (i.e., nLF), which represents the proportion of the LF power relative to the total power, except for the VLF component (nLF = LF/[Total Power – VLF]). Normalized values of LF power relative to the total power show a positive relationship with sympathetic activation (Montano et al., 1994; Pagani et al., 1986, 1997).

Statistical Analysis

Event-related potentials (HEPs, control intervals) were analyzed using a $2 \times 2 \times 3 \times 3 \times 2$ mixed design analysis of variance (ANOVA) comprising the between-subjects factor menstrual cycle phase (FP, LF), and the within-subjects factors food consumption (NC, FD), scalp location (frontal, central, parietal), laterality (left, midline, right), and eyes (open, closed). Post hoc analyses were performed using Bonferroni-corrected *t* tests for dependent samples. For any effect with repeated measures and more than two conditions, Greenhouse-Geisser corrected *p* values are reported. Cardiovascular data (heart rate, nLF HRV) were analyzed using a $2 \times 2 \times 2$ mixed design ANOVA with the between-subjects factor menstrual cycle phase and the within-subjects factors food consumption and eyes. Critical α level was set to .05. Subjective ratings of stress, arousal, hunger, and motivation to eat were analyzed by a one-factorial, two-level, repeated measures ANOVA with the factor food consumption (normal food consumption, food deprivation). All statistics were conducted with SPSS 19.0 (SPSS, Inc.).

Results

Heartbeat-Evoked Potential (HEP) Amplitudes

HEPs did not differ between the two phases of the menstrual cycle ($F[1,14] < 1$), and there was also no interaction between menstrual cycle and any other factor. HEP amplitudes were dependent on laterality ($F[2,28] = 14.50$; $p < .001$; $\eta^2 = .51$), indicating higher HEPs at electrodes over the midline ($M = .61$ [$SEM = .09$] μV), and the right hemisphere (.56 [.08] μV), than over the left hemisphere (.41 [.07] μV; *p*s < .01). Furthermore, we observed higher HEP amplitudes after FD (.63 [.10] μV), as compared to NC (.42 [.08] μV; $F[1,14] = 5.91$; $p = .029$; $\eta^2 = .30$). There was no effect of food consumption on mean activity in the early (180–320 ms) or the late (860–1,000 ms) control interval (*F*s[1,14] < 1). Neither were there main effects of open (47 [.08] μV), versus closed eyes on HEP amplitudes (.59 [.12] μV; $F[1,14] = 1.29$; $p = .29$), or of scalp location (frontal: .56 [.10] μV; central: .57 [.09] μV; parietal: .46 [.07] μV; $F[2,28] = 1.72$; $p = .21$) nor an interaction effect of food consumption with any other factor. Averaged waveforms of heartbeat-evoked potentials ranging from -200 to +1,000 ms relative to a respective R wave are illustrated in Figure 1.

Cardiovascular Data

There was no main effect of menstrual cycle on any indicator of cardiovascular activity, except for a trend of lower heart rate during the FP ($M = 71.1$ [$SEM = 1.9$] bpm), than during the LP (76.3 [2.1] bpm; $F[1,14] = 3.33$; $p = .089$; $\eta^2 = .19$). The different nutrition conditions had no effect on heart rate or normalized low frequency (nLF) heart rate variability (*F*s < 1). Heart rate was higher during open eyes (74.0 [1.4] bpm), than during closed eyes (72.8 [1.6] bpm; $F[1,14] = 7.18$; $p = .018$; $\eta^2 = .34$), whereas the factor eyes did not affect nLF heart rate variability ($F[1,14] = 2.14$; $p = .17$). We also observed a significant interaction of Menstrual Cycle × Eyes on nLF ($F[1,14] = 5.19$; $p = .039$; $\eta^2 = .27$), indicating higher nLF in the open (.68 [.05] n.u.), than in the closed eyes condition (.62 [.06] n.u.; $p < .05$), only in the LP, but not in the FP (open: .54 [.05] n.u.; closed: .55 [.05] n.u.). None of the remaining effects were significant (all *p*s > .10). Cardiovascular data arranged across factors menstrual cycle, food consumption, and eyes are presented in Table 1.

Subjective Ratings

Ratings from an overlapping sample have been reported elsewhere (Ferreira de Sá et al., 2014). Analysis of subjective ratings revealed no differences between food conditions for arousal (NC: 429 [29]; FD: 435 [39]; $F[1,15] < 1$; $p = .71$) or stress (NC: 495 [41]; FD: 481 [40]; $F[1,15] < 1$; $p = .84$). Hunger ($F[1,15] = 121.05$, $p < .001$; $\eta^2 = .89$), and motivation to eat ($F[1,15] = 117.40$, $p < .001$; $\eta^2 = .89$), were significantly higher during FD (hunger: 741 [40]; motivation to eat: 788 [42], compared to NC, hunger: 313 [15]; motivation to eat: 346 [18]).

Discussion

In the current study, HEPs were investigated during normal and restricted food consumption. Participants were tested both after a normal standardized food intake and after approximately 18 h of food deprivation. The first aim of the study was to clarify whether potential effects of food deprivation on interoception are due to peripheral effects, such as higher stimulation of interoceptors by increased sympathetic tone, or to altered cortical representation of afferent signals from the cardiovascular system. In accordance with Hypothesis 1, 18 h of food deprivation increased subjective feelings of hunger and motivation to eat, but did not induce an increase in heart rate or in normalized LF HRV. The second aim of the present study was to determine whether food deprivation might alter genuine CNS representations without the active awareness of bodily processes. As expected in Hypothesis 2, we observed higher HEP amplitudes after food deprivation compared to normal food consumption. The results of the present study show that short-term food deprivation alters an electrophysiological correlate of genuine cortical representation of visceral-afferent signals originating from the cardiovascular system. This effect could not be explained by increased peripheral cardiac activity after food deprivation.

HEPs represent a cortical phenomenon with dominant frontal-to-parietal distribution (Dirlich, Dietl, Vogl, & Strian, 1998; Montoya et al., 1993; Schandry & Montoya, 1996). Thus, the current investigation focused on these scalp regions. No differences were observed between frontal, central, and parietal electrodes.

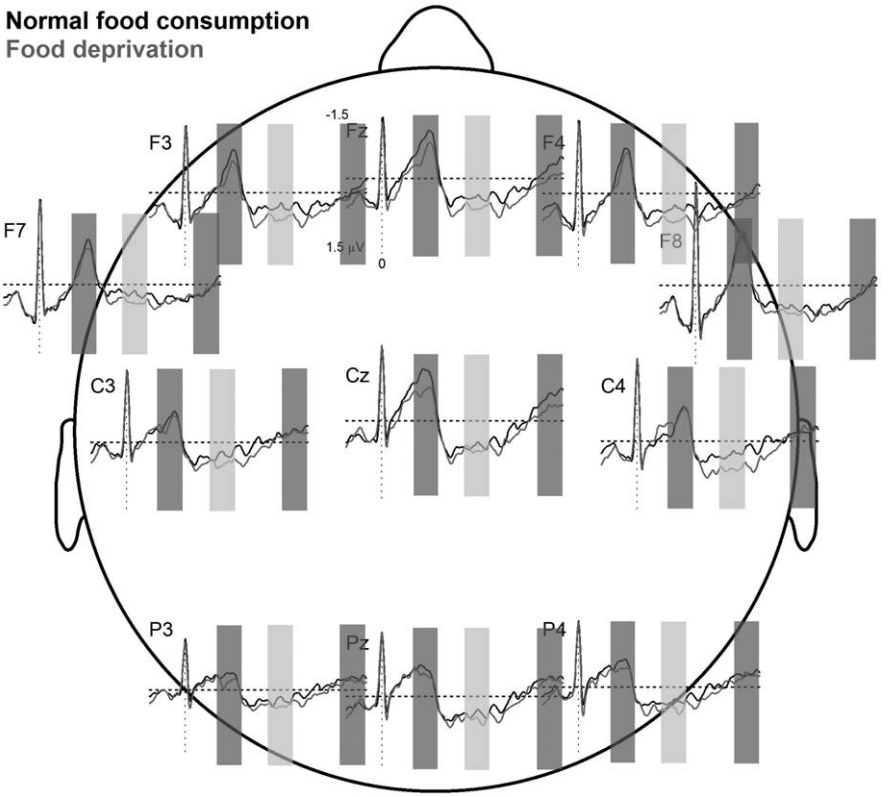

Figure 1. Waveforms of averaged heartbeat-evoked potentials (HEPs) in both nutritional conditions (NC, FD). All potentials are aligned to the R wave of the ECG, ranged from -200 to +1,000 ms. HEPs were derived from the time period of 455–595 ms after the R wave (light gray bar), which were compared to two control intervals (dark gray bar). Differences between nutritional conditions were only observed in HEPs, but not in control intervals.

However, in accordance with earlier findings (Leopold & Schandry, 2001; Montoya et al., 1993; Pollatos & Schandry, 2004; Schandry et al., 1986), we found higher HEP amplitudes at electrodes over the midline and over the right hemisphere than over the left hemisphere, which may reflect advantages of the right hemisphere to process cardiac interoceptive sensations. It remains unclear why HEP amplitudes in the current study show hemispheric lateralization, while the food deprivation effect on HEPs was not lateralized. As HEPs are generated by multiple sources, presumably located in the insula, the anterior cingulate, the medial frontal, and the inferior parietal gyrus (Pollatos et al., 2005), it seems plausible that only some of these sources are responsible for the largest amplitudes over Cz and the right hemisphere, while others are affected by food deprivation. It remains for future research to further elucidate the role of cortical dipoles responsible for HEP generation in processing visceral-afferent signals and nutritional states. However, since food deprivation only affected HEP amplitudes but not mean activity in the control intervals, it is plausible that the reported effect can be attributed to altered cortical representation of visceral-afferent signals, and does not represent a general effect on electrocortical activity.

The main finding of the current study is the increase of HEP amplitudes after food deprivation. This is in accordance with an earlier study showing that food deprivation enhances accuracy in a

Table 1. *Heart Rate and Normalized Low Frequency Heart Rate Variability*

		Normal food consumption				Food deprivation			
		Eyes open		Eyes closed		Eyes open		Eyes closed	
	Unit	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Midfollicular phase ($n = 9$)									
Heart rate[a]	bpm	70.2	2.1	69.1	2.3	73.3	2.4	71.8	2.6
nLF HRV[b]	n.u.	.52	.05	.53	.05	.56	.07	.57	.06
Midluteal phase ($n = 7$)									
Heart rate[a]	bpm	78.8	2.3	77.8	2.6	75.0	2.7	73.5	3.0
nLF HRV[b]	n.u.	.67	.05	.67	.06	.70	.07	.58	.07

Note. nLF HRV = normalized low frequency heart rate variability.
[a]Main effect eyes: $F(1,14) = 7.18$; $p = .018$; $\eta^2 = .34$.
[b]Interaction effect Menstrual Cycle Phase × Eyes: $F(1,14) = 5.19$; $p = .039$; $\eta^2 = .27$.

heartbeat counting task (Herbert, Herbert et al., 2012), since HEP amplitudes and interoceptive accuracy as indexed by heartbeat perception tasks are positively correlated (Katkin et al., 1991; Pollatos & Schandry, 2004; Yuan et al., 2007). Herbert, Herbert, and colleagues (2012) reported a substantial increase of sympathetic activation in response to 24 h of food deprivation, indicated by higher heart rate and cardiac contractility, as well as lower normalized HF HRV (which implicate higher normalized LF HRV) and preejection period. The authors argue that this sympathetic activation may be responsible for the increase in interoceptive accuracy after food deprivation. In line with earlier findings (Horowitz et al., 1999), in the current study we could not find an effect of 18 h of food deprivation on sympathetic activation, as indexed by normalized LF HRV. It could be argued, therefore, that the effect of food deprivation on the central representation of visceral-afferent signals is independent of sympathetic activation. Nevertheless, it remains for future research to clarify whether this may also account for the conscious perception of interoceptive signals.

The current results are limited to CNS processing of interoceptive signals originating from the cardiovascular system. Since it was repeatedly shown that interoceptive accuracy for gastrointestinal and cardiac sensations partially overlap (Herbert, Muth et al., 2012; Whitehead & Drescher, 1980), one could argue that food deprivation results in greater attentional focus on bodily sensations in general because of subjective feelings of hunger. Follow-up studies could benefit from the inclusion of indicators reflecting cardiac and gastrointestinal interoceptive accuracy.

Short-term food deprivation induces a complex interplay of endocrine and neural signaling. While we did not assess changes in hormonal concentrations in the current study, future research should address the relationship between certain hormones and interoceptive signal processing. Some of the hormonal changes seen during fasting include reduced secretion of leptin, insulin, peptide YY, and cholecystokinin, and increased secretion of ghrelin (Neary et al., 2004; Smith & Ferguson, 2008; Woods, Seeley, Porte, & Schwartz, 1998). These hormones convey metabolic information to the brain by afferent signaling into structures, such as the hypothalamus and the nucleus tractus solitarius (NTS), as well as cortical regions (e.g., anterior insula, orbitofrontal cortex), which are involved in the regulation of the autonomic nervous system and the processing of interoceptive signals. Ghrelin, for example, has been reported to inhibit catecholaminergic neurons in the NTS (Cui, Li, & Appleyard, 2011) and to modulate brain activity in the orbitofrontal cortex and the anterior insula (Malik, McGlone, Bedrossian, & Dagher, 2008). Intranasal insulin may also increase blood perfusion in the insula (Schilling et al., 2013), which plays an important role in the regulation of eating behavior (Small, 2010). The present results, however, do not show any effect of the menstrual cycle phases on HEPs. This suggests that menstrual cycle differences in eating behavior might not be related to altered processing of afferent bodily sensations. Notwithstanding, it is important for future research in female samples to also include the ovulation phase, since menstrual cycle differences in eating behavior are more evident during the periovulatory period (Marino et al., 2011).

In a previous study investigating effects of cortisol on HEP amplitudes, we observed lower heart rate during closed eyes compared to open eyes, implying a state of decreased alertness and increased relaxation (Schulz, Strelzyk et al., 2013). The current results confirm Hypothesis 3, assuming that there is no general effect of open versus closed eyes on HEP amplitudes, supporting the theory that the previously found main effect was provoked by cortisol infusion. Moreover, there was no interaction of food condition and open versus closed eyes on HEPs, suggesting that visual load or potential states of alertness versus relaxation does not play a role for the relationship of food deprivation and the processing of interoceptive signals.

Interestingly, our findings contrast with existing results on interoception in anorexia nervosa. Based on the current findings on increases in interoception after short-term food deprivation, one might speculate that long-term food deprivation, as in anorexia nervosa, leads to even more enhanced interoceptive signal processing. Opposed to this notion is the finding that interoceptive accuracy in anorexia nervosa is reduced as compared to healthy controls (Pollatos et al., 2008). One possible explanation for this discrepancy lies in the finding that physiological processes in response to long-term food deprivation (> 72 h) are not identical with those after short-term deprivation: increased ghrelin and decreased leptin are chronically dysregulated (Misra & Klibanski, 2010; Stoving et al., 2009), while peptide YY concentration reverses into a down-regulation (Misra & Klibanski, 2010), and the autonomic nervous system into parasympathetic dominance (Mazurak, Enck, Muth, Teufel, & Zipfel, 2010). Although Pollatos and colleagues (2008) hypothesized that reduced disposition of interoceptive accuracy may represent a risk factor for the development of anorexia nervosa, the current findings imply that the relationship between interoception and eating behavior could also be the reverse. Eating behavior could represent a (dysfunctional) coping strategy either to regulate the perception of bodily sensations per se or phenomena that are closely related to interoception, such as the experience of emotions (Wiens, 2005) or stress, in particular (Schulz, Lass-Hennemann, Sutterlin, Schächinger, & Vögele, 2013; Schulz, Strelzyk et al., 2013).

In existing literature, there is an ongoing debate on the time period relative to an R wave, during which HEPs are considered to reflect cortical processing of cardiac sensations. Applied time windows range from early latencies, such as 100–450 ms (Yuan et al., 2007), 250–350 ms (Pollatos & Schandry, 2004), 280–330 ms (Leopold & Schandry, 2001), or 250–430 ms (Fukushima, Terasawa, & Umeda, 2011), which require an elimination of the partially overlapping electrocardiac field, over mixed latencies of 350–550 ms (Montoya et al., 1993) or 200–600 ms (Shao et al., 2011), and late latencies of 455–595 ms (Gray et al., 2007; Schulz, Strelzyk et al., 2013), which are largely unrelated to electric fields originating from the heart. As argued by Gray and colleagues (2007), HEP activity during that time interval may rather be related to afferent neural signals originating from arterial baroreceptors, which are cortically processed about 400–800 ms after the R wave (Fagius & Wallin, 1980) and represent one important neural correlate of cardiac interoception (Dworkin, 2007).

Limitations

In the current study, a sample of young, healthy female students was investigated. Despite the fact that investigating eating behavior in women may be of particular interest, since the prevalence of eating disorders is much higher in women than men, it remains to be seen whether the current findings can be generalized to different age groups or to male individuals, as these factors may affect the relationship between nutritional state and interoception. In addition, the present study does not allow for conclusions about interoceptive processes in clinical manifestations of altered eating behavior (i.e., eating disorders).

Conclusions on nonsignificant effects regarding the factor menstrual cycle have to be interpreted with caution given the limited power of the between factor in the present design. To ensure absolute compliance with fasting, a biological measure would have been valuable, although a previous study suggests that invasive biological measures of compliance are not essential when studying healthy individuals (Mauler, Hamm, Weike, & Tuschen-Caffier, 2006). Still, we applied two different strategies to ensure compliance with fasting instruction (diary method, cover story with saliva sample). Furthermore, ratings of hunger and motivation to eat were higher during FD, supporting the successful manipulation of food condition. Although our findings suggest that food deprivation may selectively affect HEPs in a time frame that is associated with the cortical representation of visceral-afferent signals, further studies will have to show whether there may be an effect of fasting on other event-related potentials. In the current study, by deriving HEPs from the period of 455–595 ms, we have followed repeatedly published standards to minimize cardiac field artifacts (Gray et al., 2007; Schulz, Strelzyk et al., 2013). Nevertheless, we cannot rule out the possibility that ECG-related processes, which occurred later than 595 ms after the R wave, may have affected electrocortical potentials in the selected interval to some degree.

We assessed HEPs during rest without any instruction to focus attention toward cardiac sensations and concluded that awareness of interoceptive signals may not be required for the observed effects. The availability of a behavioral variable reflecting the attentional focus on bodily sensations would support this line of argumentation. Unfortunately, this was not possible since the assessment of any behavioral variable would also experimentally manipulate attention toward bodily sensations. Nevertheless, a parallel study incorporating behavioral measures is suggested for the future. In the current study, we used nLF HRV to estimate sympathetic activation. Since the relationship of nLF and sympathetic activation has not consistently been observed in the literature, nLF HRV may also be affected by other sources (Berntson et al., 1997; TaskForce, 1996). Although a previous study has shown that this index is sensitive to sympathetic effects of food deprivation (Herbert, Herbert et al., 2012), the use of a more direct measure (e.g., muscle sympathetic nerve activity) would have been desirable and is suggested for future studies.

Finally, in the current study we decided against the direct assessment of hormone levels, as this would have implied a level of invasiveness (i.e., continuous blood drawings) that could have challenged the intended resting character of the measurement. Data on hormone levels, however, would have been useful as these may have affected the current results. It remains for future research, therefore, to further elucidate these effects including endocrinological measures.

Conclusion

The current results demonstrate that short-term food deprivation alters an electrophysiological correlate of the central cortical processing of visceral-afferent signals originating from the cardiovascular system. This effect could not be attributed to increased cardiac activation, as estimated by heart rate and nLF HRV, after food deprivation.

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