Evidence for ERP biomarkers of eating disorder symptoms in women

Katie Groves*, Steffan Kennett, and Helge Gillmeister

Department of Psychology, University of Essex, Colchester, UK

*Corresponding author:

E-Mail: kegrov@essex.ac.uk

Abstract

Growing evidence suggests that the brain processes bodies distinctively from other stimuli, but little research has addressed whether visual body perception is modulated by the observer's thoughts and feelings about their own body. The present study thus investigated the relationship between body image and electrophysiological signatures of body perception, with the aim of identifying potential biomarkers of body image disturbances. Occipito-parietal (P1 and N1) and fronto-central (VPP) processing of body and non-body stimuli were assessed in 29 weight-restored eating disordered (ED) women and compared to 27 healthy controls. Rapid early visual processing was seen in the ED group, as the entire P1-N1 complex unfolded significantly earlier compared to controls. ED women also showed a gender-sensitive response to other women's bodies over N1 and VPP components. Such gender-sensitivity was not evident in controls. Moreover, ERP effects correlated with scores on the Eating Disorder Inventory-II (EDI-2), indicating a close link between the observers' ED symptomatology, including body image, and the visual analysis of human bodies during very early stages of cortical processing. The temporal dynamics of visual body perception may therefore serve as potential neural markers for the identification of ED symptomatology in 'at risk' populations.

Key words: Eating disorder, anorexia nervosa, bulimia nervosa, ERPs, body image, body representation

Highlights

- Abnormalities in visual processing may be at the heart of eating disorder symptomatology
- Women at risk of eating disorders show more rapid visual encoding of both body and control stimuli
- They also show selectively enhanced processing of bodies of their own gender
- Systematic variation of these effects with symptom severity identify them as potential biomarkers of eating disorder risk

1. Introduction

Over the past 15 years there have been significant advances in identifying the neural correlates of visual body perception (see Downing & Peelen, 2016 for a recent review). In a pioneering study, Downing, Jiang, Shuman, and Kanwisher (2001) suggested a module for body processing in the extrastriate body area (EBA), a bilateral region of the lateral occipital cortex that responds selectively to images of the human body. Research has since revealed that this area is largely concerned with processing body parts and perhaps the shape of the body (see also Downing & Peelen, 2016). There is also evidence to suggest that EBA contains separate networks that distinguish between own body and other body recognition (Chan, Peelen, & Downing, 2004; Myers & Sowden, 2008; Saxe, Jamal, & Powell, 2006).

The EBA is complemented by a second body-selective region, the fusiform body area (FBA) (Schwarzlose, Baker, & Kanwisher, 2005), which may contribute functionally distinct representations of the human body to perception (Taylor, Wiggett, & Downing, 2007). While there is some debate about the relative contributions of EBA and FBA, and about how they integrate information (e.g. Chan & Baker, 2011; Hodzic, Muckli, Singer, & Stirn, 2009; Urgesi, Calvo-Merino, Haggard, & Aglioti, 2007), there seems to be very little doubt that these areas are selective for the visual perception of human bodies (Sadeh et al., 2011).

Source localisation techniques (Meeren, de Gelder, Ahlfors, Hämäläinen, & Hadjikhani, 2013; Thierry et al., 2006) as well as direct intracranial recordings (Pourtois, Peelen, Spinelli, Seeck, & Vuilleumier, 2007) have linked EBA activity with

the enhancement of electrophysiological activity over occipito-temporal sites for bodies compared to non-body stimuli around 150-190ms after stimulus onset (Pourtois et al., 2007; Thierry et al., 2006). The present study investigates the event-related visual component associated with this enhancement, which has been variably referred to as N170, N190 or simply N1 (see also Peelen & Downing, 2007 for review). We will refer to this component as the body-sensitive N1 throughout the present paper. Inverting body stimuli has been found to modulate the body-sensitive N1 response (e.g. Bosbach, Knoblich, Reed, Cole, & Prinz, 2006; Minnebusch, Keune, Suchan, & Daum, 2010; Minnebusch, Suchan, & Daum, 2009). As a result, body-sensitivity in the N1 time range has been linked to late structural (de Gelder et al., 2010; Eimer, 2000c; Soldan, Mangels, & Cooper, 2006) and early configural encoding of bodies. This means that bodies, like faces, seem to be processed holistically according to the spatial relations between features, rather than the features themselves (Maurer, Le Grand, & Mondloch, 2002; Minnebusch & Daum, 2009).

Other early event-related potentials have also been linked to body selection. Thus, further to the N1, the present study will also investigate P1 responses and the vertex positive potential (VPP). P1 is the first positive deflection in the visual ERP waveform and is typically observed over occipito-parietal electrodes at around 80-120 ms after stimulus onset (see Luck, 2014, p72.). A handful of studies have found evidence for body-sensitivity in this time range, especially when stimuli contain emotional cues, or bodies are the only stimuli presented (Meeren, van Heijnsbergen, & de Gelder, 2005; Righart & de Gelder, 2007; Thierry et al., 2006; van Heijnsbergen, Meeren, Grèzes, & de Gelder, 2007). VPP is found in time ranges similar to N1, but is a positive

deflection occurring over fronto-central electrode sites, and has been implicated in the distinct visual processing of human bodies (Sadeh et al., 2011; Stekelenburg & de Gelder, 2004; van Heijnsbergen et al., 2007). In particular, evidence suggests that body-sensitive VPP responses are modulated by emotion (Stekelenburg & de Gelder, 2004; van Heijnsbergen et al., 2007). Despite some debate (e.g. Eimer, 2000b; Taylor, McCarthy, Saliba, & Degiovanni, 1999), the face processing literature indicates that VPP responses arise from the same cortical region as the N1, thus manifesting the same processes (Joyce & Rossion, 2005; Sadeh et al., 2011).

Visual body processing also includes the sight of our own bodies, which gives rise to two distinct constructs: 'body schema' and 'body image' (see Berlucchi & Aglioti, 2010 for short review). Body-schema has been described as the unconscious, physical representation of the body in space, sub-served and updated by bodily movements and the environment. Body-image on the other hand, should be understood as a conscious, mental representation of the body associated with perception and action (Berlucchi & Aglioti, 2010; Paillard, 1999). The relationship between body-related cortical processing and how observers experience their own body (body image) is of particular interest in the present study, as these introspective perceptions of one's own body do not always reflect reality. Instead, they can manifest as body image distortions that are consistently identified as contributing factors to the complex dynamics that sustain some eating disorders (EDs) (American Psychiatric Association, 2013). Such disorders are characterised by a range of abnormal food- and body-related attitudes and behaviours, including an undue tendency to emphasise the importance of body weight and shape, which can lead to

unhealthy eating habits such as binging, purging or fasting (see Skrzypek, Wehmeier, & Remschmidt, 2001).

Body image disturbances associated with EDs are multifaceted and are thought to arise from interrelated contributions from perception, cognition, affect and behaviour (see Cash, 2004). As such, their causes are still unclear (e.g. Stormer & Thompson, 1996). We were particularly interested in the perceptual facet of body image disturbance, as research is beginning to highlight how atypical functioning of the visual system might contribute to perceptual aspects of these distortions (see Suchan, Vocks, & Waldorf, 2015 for review). For example, it has been suggested that maladapted (Suchan et al., 2010) and underactive (Uher et al., 2005) EBA function, or at least disrupted communication between EBA and FBA, may underpin body image disturbance (Suchan et al., 2013). Despite evidence to suggest that early body-selective responses arise from EBA activity (e.g. Sadeh et al., 2011) little is known about the early stages of visual body-processing in EDs. Instead, studies to-date have focused on the relationship between stimulus salience and later, more conscious (see Sergent & Dehaene, 2004) stages of processing (e.g. Dodin & Nandrino, 2003; Gao et al., 2011; Horndasch, Heinrich, Kratz, & Moll, 2012; Mai et al., 2015). Therefore, the present study was designed to measure the latency and amplitude of body-sensitive P1, N1 and VPP components to shed light on the early cortical processing of male and female body stimuli in women with and without a history of EDs.

Previous ERP studies have shown that cortical alterations and pathologically related neurological differences (such as in response to food and body stimuli) are common

in those with EDs, even after weight gain (e.g. Blechert, Ansorge, Beckmann, & Tuschen-Caffier, 2011; Hatch et al., 2010; Li et al., 2015; Mai et al., 2015; Otagaki, Tohoda, Osada, Horiguchi, & Yamawaki, 1998; Pollatos, Herbert, Schandry, & Gramann, 2008; Sfärlea et al., 2016). Specifically, Mai et al. (2015) found evidence for an attentional processing bias for overweight body stimuli in participants with Bulimia Nervosa, illustrated by larger P2 amplitudes and higher arousal ratings. Li et al. (2015) found evidence for abnormal face processing mechanisms in participants with anorexia nervosa, such that anorexics showed reduced P1 amplitudes and reduced and delayed N170 amplitudes relative to control participants. This was interpreted as reflecting reduced configural processing for face stimuli in these individuals. In addition, Sfärlea et al. (2016) suggest that reduced early posterior negativity (EPN) amplitudes in anorexic girls is potentially indicative of other peoples faces being perceived as less intrinsically relevant.

In sum, despite research clearly showing that it is possible to establish links between ED symptomatology and ERP responses (e.g. Li et al., 2015; Mai et al., 2015; Sfärlea et al., 2016), no ERP study to-date has investigated the early temporal dynamics of body processing in both anorexic and bulimic populations. This is of interest as the shared core pathology of anorexia and bulimia is the tendency to over-evaluate weight and shape (see Fairburn & Harrison, 2003 for review). Moreover, with reports stating that anorexia nervosa still has the highest death rate of all psychiatric conditions (e.g. Arcelus, Mitchell, Wales, & Nielsen, 2011; Papadopoulos, Ekbom, Brandt, & Ekselius, 2009), which prompted recent calls for more evidence-based treatment and early interventions (World Eating Disorders Action Day, 2016), the identification of objective, biological markers of ED symptoms

would be timely. It is therefore important to investigate visual body processing not only in bulimia (Mai et al., 2015) but also in anorexia.

In addition, electrophysiological research on body representation suggests that the body-sensitive N1 is modulated by the gender of the body observed. This is because men (Hietanen & Nummenmaa, 2011) and women (Alho, Salminen, Sams, Hietanen, & Nummenmaa, 2015) have been found to elicit a larger body-sensitive N1 to female bodies in comparison to male bodies. Both Hietanen and Nummenmaa (2011) and Alho et al. (2015) proposed that the structural encoding of bodies may therefore trigger later attraction-related responses relevant for mating. For this argument to be convincing, however, one would expect these selective enhancements to hold across sexual orientations and gender (e.g. heterosexual women should show enhanced amplitudes to men, not women). Despite this, Alho et al. (2015) reasoned that the same-sex gender selectivity seen over the N1 for their female participants may be because women display similar physiological and evaluative sexual responses toward both genders (see Rupp & Wallen, 2008 for review). However, if this is the case and N1 gender-sensitivity truly reflects an early sexual response, then women should show an absence of N1 gender-sensitivity, rather than enhanced responses to the sight of female bodies. It appears then, that an alternative explanation may be more fitting and consequently, the temporal dynamics of gender-sensitive body perception warrants further investigation.

The aim of the present study therefore, was to investigate the early stages of visual body- and gender-sensitive processing in observers at risk of anorexia or bulimia, in order to identify potential biomarkers of ED symptoms. Body-sensitive P1 and N1

responses were sought over occipito-parietal electrodes, and body-sensitive VPP responses were sought over fronto-central regions, by comparing the brain's response to bodies and non-body stimuli (houses) in an oddball detection task (response to animals). This design was selected (similar to van Heijnsbergen et al., 2007) so that bodies were not the focus of the task, as evidence suggests attentional differences between ED participants and controls when viewing bodies (Blechert, Nickert, Caffier, & Tuschen-Caffier, 2009; Horndasch, Kratz, et al., 2012; Jansen, Nederkoorn, & Mulkens, 2005; Mahamedi & Heatherton, 1993; Shafran, Lee, Cooper, Palmer, & Fairburn, 2007; Vocks et al., 2010; Warschburger, Calvano, Richter, & Engbert, 2015), which could have influenced ERPs (Hillyard & Anllo-Vento, 1998). Both male and female bodies were shown in order to assess for any gender-sensitive effects over P1, N1 or VPP. Body stimuli were rated for valence and arousal, and the Eating Disorder Inventory-II (EDI-2) (Garner, 1991) was used as a measure of body image disturbances and characteristic traits of EDs in all participants.

We predicted that the early visual analysis of human bodies would differ between the groups, as reflected in P1, N1 and VPP responses. Although we did not specifically test for configural processing abnormalities, as Li et al. (2015) found altered early visual ERPs indicative of atypical configural face processing in anorexic participants, given that face and body processing mechanisms are reportedly similar (see de Gelder et al., 2010; Minnebusch & Daum, 2009 for review), there was a possibility of finding differences between the groups that might indicate atypical configural body processing in ED populations. We further expected that ED participants might feel differently about the body stimuli than controls, as Mai et al., (2015) found higher

arousal ratings for overweight bodies in bulimic participants and Uher et al. (2005) found higher aversion ratings for body stimuli in anorexic participants. We also expected the ED group to display higher scores on all subscales of the EDI-2. Finally, valence and arousal ratings, as well as EDI-2 scores, were predicted to linearly relate to potential ERP effects, indicating that body-sensitive processing is modulated by the way the observer thinks and feels about their own body and those of others.

2. Materials and methods

2.1 Participants

2.1.1 Eating disordered participants

Thirty weight-restored female ED participants (15 anorexic, 15 bulimic) from North East Essex, UK, and the surrounding area, were recruited via email advertisements to University of Essex mailing lists, as well as posters placed on notice boards at the University of Essex and 'The Gym' Colchester. At the time of testing, five of these participants were medicated with fluoxetine or sertraline for symptoms of anxiety and/or depression, three reported undergoing counselling and two reported receiving both medication (as above) and counselling for their eating disorder. Four participants reported having had children, with the most recent pregnancy occurring five years before testing. Information regarding age, height, weight, Body Mass Index (BMI) and hours of weekly exercise is reported in Table 1.

We chose to recruit weight-restored anorexic participants so that any differences in ERPs would not be attributable to the effects of malnourishment (although despite weight gain, two of these participants did not consider themselves even partially recovered). Similarly, BN participants who considered themselves at least partially recovered were sought. All participants self-reported a previous medical diagnosis for their ED. We chose to recruit women who had not been diagnosed with more than one ED in their lifetime so that potential differences between disorders could be assessed. Consequently, women who had been diagnosed with either anorexia only or bulimia only, and those who had no history of EDs, were recruited. No differences were evident between anorexic and bulimic participants with regards to demographic information, eating disorder symptomatology (with the exception of the bulimia subscale, see Table S1), valence and arousal ratings, or the amplitudes and latencies of early visual components (with the exception of a trend towards larger P1 amplitudes in anorexic participant's, see Table S1). Therefore, data from these women were combined into one ED group (see also Horndasch, Kratz, et al., 2012).

2.1.2 Control participants

Twenty-nine females with no clinical history of EDs or body image disturbances were recruited from the University of Essex as control participants. Two participants reported having had children, with the most recent pregnancy occurring three years before testing. Information regarding age, height, weight, Body Mass Index (BMI) and hours of weekly exercise is reported in Table 1.

2.1.3 Exclusion criteria

Individuals who had been diagnosed with more than one ED in their lifetime were not recruited. Those who had experienced a major psychiatric disorder, such as schizophrenia or bipolar disorder, were also not permitted to take part. Data from one ED participant (bulimic) and two control participants were not included due to excessive noise in the EEG recordings that made peak detection problematic.

2.1.4 Ethical declaration

The study was conducted in line with the 2008 Declaration of Helsinki, approved by the local Ethics Committee for the Psychology Department at the University of Essex, and endorsed by the Eating Disorders charity B-eat, whose advice was sought during the design phase.

Table 1.

Average demographic information for each group.

	ED group (N=29)	Control group (N=27)	T-test results
Age (years)	24.07 (8.34)	23.07 (5.35)	$t(54) = 0.54$, $p = 0.595$
Height (m)	1.66 (.07)	1.68 (.04)	$t(54) = 1.23$ $p = 0.226$
Weight (kg)	58.94 (9.33)	65.31 (12.39)	$t(54) = 2.18$, $p = 0.036$
BMI (kg/m^2)	21.38 (2.43)	23.11 (4.34)	$t(54) = 1.86$, $p = 0.075$
Weekly exercise (hrs)	5.81 (3.76)	2.91 (2.94)	$t(54) = 3.20$, $p = 0.002$
Total EDI-2 score	103.48 (48.05)	37.70 (25.05)	$t(54) = 6.486$, $p < .001$

Note. There were no differences between anorexic and bulimic participants on any of these measures, so the groups were combined to form one ED group. Standard deviation in parentheses.

2.2 Apparatus and stimuli

2.2.1 Eating Disorder Inventory-II (EDI-2)

The 'Eating Disorder Inventory-II' (EDI-2) (Garner, 1991) was used to measure the prevalence of any behavioural, cognitive and/or affective symptoms commonly associated with eating disorders. This explicit measure of unhealthy attitudes and behaviours towards one's body is a widely used, reliable and valid research tool (e.g. Clausen, Rokkedal, & Rosenvinge, 2009; Eberenz & Gleaves, 1994; Nevonen & Broberg, 2001; Thiel & Paul, 2006). The measure assesses 11 dimensions of clinical relevance by means of 91 self-report statements, for example; 'I think my hips are too big,' to which participants respond; 'Always,' 'Usually,' Often,' 'Sometimes,' 'Rarely,' or 'Never.'

2.2.2 EEG stimuli

In order to obtain realistic body stimuli representative of the bodies that might be encountered in everyday life, 96 pictures of bodies (49 female, 47 male) and 99 pictures of houses were downloaded from the World Wide Web. To further simulate realistic viewing, both body and house stimuli were selected in order to depict an array of shapes and sizes. These were classified as obese, overweight, average, thin and very thin by a focus group of University of Essex students and then assessed by UK national eating disorder charity 'B-eat'. Images of comparable background colour were selected (e.g. grey, beige, light blue) and then cropped and edited such that a similar amount of background space was evident across body

shapes. All images were matched with regards to complexity (i.e. each showing only one body or one house, rather than scenes) as this has been shown to affect attentional processes (see Miller & Fillmore, 2010).

Whilst waiting for B-eat's assessment, control data was collected. Based on B-eat's advice, the ED group did not view stimuli that had been deemed potentially triggering (e.g. bodies with visible bones or those that B-eat considered morbidly obese). Therefore, only data from the stimuli that all participants viewed were analysed for this report. Body stimuli (half side facing, half front facing) were edited to exclude the head, and all showed the full trunk but varying amount of upper and lower limbs. Fifteen pictures of animals were also included as deviant target stimuli to which a response was required. All stimuli were 267x200 pixels and luminosity was adjusted to control for brightness across all images (see *Figure 1* for examples).

Figure 1 here

2.2.3 Valence and arousal ratings of body stimuli

A computer-based task assessed responses of valence and arousal towards body stimuli. Two 9-point scales were used to represent ‘valence’ and ‘arousal’

dimensions, with adjective clusters to describe the extremes of the dimensions at either end. Scales were pictorial, using Self-Assessment Manikins (SAM) to illustrate the different points of the scale (Bradley & Lang, 1994). The centre was neutral. Each participant rated a random selection of 20 - 30 body stimuli pictures.

2.3 Procedure

A standardized overview of procedures was read, and written consent was obtained. The EDI-2 was completed during EEG preparation; an intermission of at least 45 minutes was ensured between questionnaire completion and the start of the task.

A computer-based oddball (animal) detection task (similar to van Heijnsbergen et al., 2007) was completed as EEG was recorded. Participants were asked to fixate on the centre of a grey screen (monitor resolution 1152x864 pixels). A black fixation cross was presented centrally except when it was replaced, for 250 ms on each trial, by a picture. After each 250 ms picture presentation there was a 1000 ms response interval and a random intertrial interval of between 300 and 700 ms. The picture was either a house, a male or female body, or occasionally an animal. Participants were instructed to press the space bar with both hands as quickly as possible whenever they saw an animal picture. For control participants, 195 images of bodies and houses were shown twice with the second presentation left-right reversed, and for ED participants 120 images of bodies and houses were shown three times each, with half of the total 360 presentations left-right reversed. Animals were shown twice to both ED participants and controls, with the second presentation left-right reversed. Thus, controls completed 420 trials (98 female bodies; 94 male bodies; 198 houses;

30 animals) and ED participants completed 390 trials (90 female bodies; 90 male bodies; 180 houses; 30 animals). Stimuli were shown in random order with a cumulative summary of animal detection times and errors displayed during inter-block intervals, timing of which was at the participant's discretion. Participants remained at the computer to rate some of the previously seen body pictures for valence and arousal. Upon completion the EEG cap was removed.

Digital scales were used to weigh participants and a wall chart was used to measure height. Participants were not told their height or weight, and were then debriefed and paid.

2.4 EEG recording

2.4.1 EEG acquisition

Continuous EEG was sampled at a rate of 500 Hz from 64 Ag/AgCl electrodes placed according to the international 10-10 system (EASYCAP GmbH, Herrsching, Germany). Online, the signal was referenced to the left earlobe with impedances kept below 10 kΩ. Bipolar channels recorded vertical (VEOG) and horizontal (HEOG) electro-oculogram from above and below the midpoint of the right eye and beside the outer canthi of both eyes. Recording and offline analysis of EEG and EOG data was conducted with Neuroscan Synamps2 system and SCAN 4.5 software (Compumedics, Melbourne, Australia). Offline, EEG and EOG signal were digitally filtered using a 0.15Hz - 30Hz bandpass filter and re-referenced to the average of the two earlobes.

2.4.2 Segmentation

The data were divided into 600-ms epochs beginning 100 ms prior to stimulus onset and baseline corrected against the mean voltage during the 100-ms pre-stimulus period.

2.4.3 Artifact detection

Trials with horizontal eye movements (HEOG exceeding ± 40 μV relative to baseline), eye blinks or other artefacts (a voltage exceeding ± 80μV at any electrode relative to baseline) were rejected from further analysis. ERPs to target stimuli (animals) were also not included.

2.5 Statistical analyses

2.5.1 Demographics

Bonferroni-adjusted independent samples t-tests were conducted on demographic data in order to compare, age, height, weight, BMI and amount of weekly exercise between the groups. T-tests are reported unsigned.

2.5.2. EDI-2

Scores pertaining to the eleven subscales of the EDI-2 were calculated according to the manual (Garner, 1991) and then averaged for each group. Bonferroni-adjusted

independent samples t-tests were conducted separately for each subscale in order to assess differences in ED symptomatology between the groups. T-tests are reported unsigned.

2.5.3 Valence and arousal ratings

Valence and arousal ratings given to body stimuli were subject to separate 2 x 2 mixed factorial analysis of variance (ANOVA) with gender (male body vs. female body) as the within subjects factor and group (ED vs. control) as the between-subjects factor. Greenhouse-Geisser adjustments to the degrees of freedom were applied when necessary and partial eta squared is reported as the measure of effect size. Follow-up pairwise comparisons of the estimated marginal means were Bonferroni corrected.

2.5.4 Electrophysiology

2.5.4.1 Electrode selection and ERP data extraction

In order to identify the electrodes on which ERP components should be measured, maximal P1 and N1 responses were assessed in each individual, at lateral posterior electrodes TP7/8, CP5/6, PO3/4, PO5/6, PO7/8, P3/4, P5/6, P7/8, O1/2, which are frequently implicated in body processing (e.g. Minnebusch et al., 2010; Minnebusch et al., 2009; Stekelenburg & de Gelder, 2004; Thierry et al., 2006; van Heijnsbergen et al., 2007). Discernible peaks for both the P1 and the N1 were seen in all participants only over electrodes P5/6, P7/8, PO5/6, PO7/8. P1 scalp topographies

associated with the aggregated grand averaged waveforms (see Figure 2) also indicated that this electrode selection captured the strongest P1 response in all groups of participants. We deemed N1 scalp topographies insufficiently informative, as the N1 remained in the positive range with a strong frontal negativity evident in the same time range. Instead, we computed P1 to N1 peak-to-peak amplitudes, and found that these, too, were most frequently maximal over P5/6, P7/8, PO5/6, PO7/8. To investigate body processing for both P1 and N1 time ranges, individual peak amplitudes and peak latencies were therefore extracted separately for male bodies, female bodies and houses at electrodes P5/6, P7/8, PO5/6, PO7/8. For the vast majority of P1 and N1 components, peak identification was straightforward within typical time windows based on the aggregated grand average waveform (P1: 70ms-140ms; N1: 120ms-190ms). However occasionally, for some participants, double peaks were observed for some components at some electrodes. The choice of which peak data to extract was informed by finding the same component peaks in surrounding electrodes in the same hemisphere or homologous electrodes on the opposite hemisphere. This approach was chosen over an automated approach because we noticed that latencies were very different from one person to the next and true component peaks would thus be missed by using a general time window.

To characterise the VPP, individual maximal positive peak amplitudes and latencies were assessed at fronto-central electrodes that have been implicated in previous VPP analyses (Ashley, Vuilleumier, & Swick, 2004; Eimer, 2000a; Luo, Feng, He, Wang, & Luo, 2010; Sadeh et al., 2011; Stekelenburg & de Gelder, 2004; van Heijnsbergen et al., 2007; Wheatley, Weinberg, Looser, Moran, & Hajcak, 2011). Strongest responses were seen at Fz, F1/2, and F3/4, with scalp topographies of the

grand averaged origins of the VPP waveform for each group supporting this (see Figure 2). Maximal peak amplitudes and peak latencies were therefore extracted separately for bodies and houses at electrodes Fz, F1/2, and F3/4. One ED participant (anorexic) did not show obvious VPP peaks to houses so their data was excluded from the body-sensitivity analysis. Again, to evaluate gender-sensitivity over the VPP, individual maximal peak amplitudes and latencies were extracted separately for male and female body trials at the same electrodes. The process to achieve this was identical to the process for the P1 and N1. Grand averaged VPP waveforms of all visual stimuli served as a guide for the timing of VPP deflections in each group (120ms -190ms for the ED group, 140ms – 190ms for controls). Manual identification and extraction of the VPP in each individual was then completed as previously described for P1 and N1.

2.2.4.2. ERP statistical analyses

To assess body-sensitivity, both amplitude and latency data for each component were subjected to separate mixed factorial ANOVA with group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses –as above) as the within-subjects factors. Gender-sensitivity was assessed similarly, with group as the between-subjects factor (control vs. ED) and picture type (male body vs. female body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (as above) as the within-subjects factors.

For the sake of brevity, non-significant statistics are not reported, and hemisphere and electrode effects are only reported if they interacted meaningfully with picture type or group (see Tables S2 – S6 for full ANOVA results). Greenhouse-Geisser adjustments to the degrees of freedom were applied when necessary and partial eta squared is reported as the measure of effect size. Pairwise comparisons were Bonferroni corrected and t-tests are reported unsigned.

2.5.5 Correlational analyses

In order to investigate the links between lifestyle, cognition and electrophysiology, we planned to conduct a Pearson's *r* correlational analysis between the demographic factors, EDI-2 scores, valence and arousal ratings and ERP effects, which were found to differ between groups. Thus, relationships between sociodemographic factors and ERP effects were of interest as they would inform an understanding of group differences. As evidence suggests that eating disorder symptoms occur on a spectrum (Bienvenu et al., 2000; Shisslak, Crago, & Estes, 1995; Widiger & Samuel, 2005) the analysis was conducted across groups, synonymous with the methods of previous studies that have employed groups with different eating pathology (e.g. Eshkevari, Rieger, Longo, Haggard, & Treasure, 2012; Mai et al., 2015; Mitchison, Crino, & Hay, 2013). In line with this, data on figures have been colour coded such that ED and control data can be identified (see S1 – S20). The false discovery rate method of correction for multiple comparisons (Benjamini & Hochberg, 1995) was applied to correlation results, results that did not survive correction are not reported.

3 Results.

3.1 Demographics

Bonferroni-adjusted independent sample t-tests assessing sociodemographic factors between the groups revealed no differences in age or height. However, ED participants were significantly lighter and performed more exercise on average per week than the controls ($t(1, 54) \geq 2.182$, $p \leq .036$). There was also a trend towards a lower average BMI in the ED group ($t(1, 54) = 1.861$, $p = 0.075$) although these were still in the healthy range (>18.5 kg/m^2; see Table 1; see Gallagher et al. (2000)).

3.2 EDI-2

Scores pertaining to the eleven subscales of the EDI-2 were calculated according to the manual (Garner, 1991) and then averaged for each group. Bonferroni-adjusted independent samples t-tests revealed that scores differed significantly between the groups on all subscales ($t(1, 54) \geq 2.153$, $p \leq .037$) with ED participants scoring higher than controls (see Table 2).

Table 2.

Mean scores and standard deviations for each group on the EDI-2 subscales.

EDI-2 Subscale	ED Group Mean Score (SD)	Control Group Mean Score (SD)	*T-test results*
Drive for Thinness	11.93 (5.46)	3.26 (4.03)	t(54) = 6.72 p < .001
Bulimia	6.31 (5.99)	1.56 (2.61)	t(54) = 3.89, p < .001
Body Dissatisfaction	14.66 (7.05)	8.44 (8.85)	t(54) = 2.92, p = 0.005
Ineffectiveness	9.93 (7.89)	3.11 (4.15)	t(54) = 4.09, p < .001
Perfectionism	9.10 (4.43)	5.81 (4.44)	t(54) = 2.77, p = 0.008
Interpersonal Distrust	5.79 (4.50)	1.04 (1.68)	t(54) = 5.31, p < .001
Interoceptive Awareness	11.21 (7.56)	2.59 (4.19)	t(54) = 5.32, p < .001
Maturity Fears	8.10 (7.18)	4.78 (4.05)	t(54) = 2.15, p = 0.037
Ascetism	9.07 (4.09)	1.96 (1.93)	t(54) = 8.41, p < .001
Impulse Regulation	9.00 (6.89)	2.18 (3.29)	t(54) = 4.78, p < .001
Social Insecurity	8.38 (5.41)	2.96 (2.78)	t(54) = 4.66, p < .001

Note. SD=Standard Deviation

3.3 Valence and arousal ratings

A 2 x 2 mixed factorial ANOVA on valence ratings of body stimuli, with gender (male body vs. female body) as the within subjects factor and group (ED vs. control) as the between-subjects factor, revealed a significant main effect of gender ($F(1, 54) = 7.294$, $p = 0.009$, $\eta_p^2 = 0.119$). Follow-up comparisons showed that female bodies were rated more positively than male bodies ($F(1, 54) = 7.573$, $p = 0.008$, $\eta_p^2 = 0.123$, see table 3). It should be noted nonetheless, that ratings for both male and female bodies were still rated around the neutral mark of '4.' This effect did not interact with group ($F(1, 54) = 2.184$, $p = 0.145$, $\eta_p^2 = 0.039$) and the between-

subjects main effect of group was also non-significant ($F(1, 54) = 0.232$, $p = 0.632$, $\eta_p^2 = 0.004$).

A 2 x 2 mixed factorial ANOVA on arousal ratings of body stimuli, with gender (male body vs. female body) as the within subjects factor and group (ED vs. control) as the between-subjects factor, did not yield any significant main effects or interactions.

Table 3.

Mean ratings and standard deviations of valence and arousal towards male and female stimuli in both groups.

Scale	ED Group Mean Rating (SD)	Control Group Mean Rating (SD)
Valence to male bodies	4.66 (.84)	4.42 (1.04)
Valence to female bodies	4.78 (.73)	4.80 (1.00)
Arousal to male bodies	5.13 (1.13)	4.95 (1.20)
Arousal to female bodies	5.03 (1.07)	5.23 (1.28)

Note. SD=Standard Deviation

3.4 Electrophysiology

3.4.1 Assessing for body-sensitivity

ERPs to body and house stimuli were compared to assess for selective responses to bodies over parietal-occipital (P1 and N1 components) and fronto-central (VPP component) electrodes. Body selectivity in the amplitudes and latencies of these components was compared between ED and control groups. Latency and amplitude

of all components were therefore subject to separate mixed factorial ANOVA with group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses – see method section) as the within-subjects factors.

3.4.1.1 P1 Amplitude

As suggested in *Figure 2* below, there were no significant main effects of picture type or group over the P1 amplitude. Still, there was a significant interaction between picture type, hemisphere and electrode ($F(3, 162) = 4.002$, $p = 0.014$, $\eta_p^2 = 0.069$). Follow-up comparisons of the estimated marginal means revealed that, despite a trend at electrode P5 showing marginally larger amplitudes to bodies ((7.369 μV vs. 4.914 μV) $F(1, 54) = 3.288$, $p = 0.075$, $\eta_p^2 = 0.057$), there were no amplitude differences between picture types at any of the electrodes. P1 amplitudes were, however, significantly larger to both bodies and houses in the right hemisphere at all electrodes ($F(1, 54) \geq 8.529$, $p \leq .005$, $\eta_p^2 \geq .136$) except PO5/6 where the pattern was marginal ($F(1, 54) = 3.529$, $p = 0.066$, $\eta_p^2 = 0.061$). The between-subjects effect of group was non-significant and there were no significant interactions with group.

This suggests that P1 amplitudes are generally larger in the right hemisphere despite some electrode differences, but are not specifically sensitive to the human form and are unrelated to ED symptomatology.

Figure 2 here

3.4.1.2 P1 Latency

Figure 2 also suggests that P1 latencies differed between stimuli and again between the groups. This was confirmed by ANOVA showing a main effect of picture type such that bodies evoked quicker P1 responses than houses (101.916 ms vs. 109.973 ms) ($F(1, 54) = 24.217$, $p < .001$, $\eta_p^2 = 0.310$) across groups. This suggests that body-sensitive responses may already be seen in P1 time ranges. In addition, P1 latencies to all stimuli were shorter in ED participants compared to controls (100.069 ms vs. 111.819 ms) as illustrated by a significant between-subjects effect ($F(1, 54) = 7.549$, $p = 0.008$, $\eta_p^2 = 0.123$). This suggests that shortened P1 latencies during visual processing, regardless of stimulus type, may be related to ED symptomatology (P1 latency did not differ between anorexic and bulimic participants – see Table S1).

3.4.1.3 N1 Amplitude

Observation of *Figure 2* also suggests clear amplitude differences between viewing house and body stimuli in the N1 time range. ANOVA confirmed this, showing that bodies evoked larger negative amplitudes than houses (-1.471 μV vs. 2.030 μV) ($F(1, 54) = 88.288$, $p < .001$, $\eta_p^2 = 0.620$). This pattern did not differ between the groups, and neither did the overall component as the between-subjects factor group was not significant. These findings support the existing claim that the N1 is body-sensitive, and further suggests that body-sensitivity in the N1 time range does not differ in those with EDs.

3.4.1.4 N1 Latency

With regards to the time course of the N1 in response to bodies and houses, ANOVA revealed a main effect of picture type ($F(1, 54) = 17.625$, $p < .001$, $\eta_p^2 = 0.246$) as houses evoked shorter N1 latencies than bodies (151.126 ms vs. 159.074 ms). This did not differ between groups but a significant between-subjects effect was found ($F(1, 54) = 5.115$, $p = 0.028$, $\eta_p^2 = 0.087$), as N1 responses in the ED group were significantly quicker overall in comparison to controls (149.987 ms vs. 160.216 ms). This suggests that the temporal dynamics of visual processing in both P1 and N1 time ranges may be related to ED symptomatology (N1 latency did not differ between anorexic and bulimic participants – see Table S1).

3.4.1.5 VPP Amplitude

Figure 3 below suggests that, similar to the N1, there was also a body-sensitive effect over VPP in both groups. This was confirmed as a main effect of picture type in the ANOVA ($F(1, 52) = 7.441$, $p = 0.009$, $\eta_p^2 = 0.125$), such that bodies evoked larger VPP amplitudes than houses (1.293 μV vs. .434 μV). This pattern did not differ between the groups and neither did the overall component, as the between-subjects factor group was not significant. These findings support the idea that VPP body selectivity might be a reflection of N1 body-sensitivity.

Figure 3 here

3.4.1.6 VPP Latency

Figure 3 suggests that VPP latencies to house stimuli may be shorter in both groups. ANOVA revealed a main effect of picture type ($F(1, 52) = 52.966$, $p < .001$, $\eta_p^2 = 0.505$) with follow-up comparisons showing faster responses to house stimuli (151.165ms) than to body stimuli (167.516). Unlike N1 latencies, average VPP latencies were not modulated by ED symptomatology, as there was no interaction with group.

3.4.2 Assessing for gender-sensitivity

ERPs to male and female body stimuli were compared to assess for selective responses to gender over parietal-occipital (P1 and N1 components) and fronto-central (VPP component) electrodes. As houses do not have a gender, these stimuli were not included in the analyses. Gender-sensitivity in the amplitudes and latencies of these components was compared between ED and control groups. Latency and amplitude of all components were therefore subject to separate mixed factorial ANOVA with group as the between-subjects factor (control vs. ED) and picture type (male body vs. female body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (as above) as the within-subjects factors.

3.4.2.1 P1 Amplitude

ANOVA found that P1 amplitudes were larger in the right hemisphere (7.842 μV vs. 5.598 μV) ($F(1, 54) = 28.528$, $p < .001$, $\eta_p^2 = 0.346$; see *Figure 4* below). No other significant main effects or interactions were found. The between-subjects effect of group was non-significant. This suggests that P1 amplitudes to bodies in general, are larger in the right than in the left hemisphere but are not sensitive to gender or related to ED symptomatology.

Figure 4 here

3.4.2.2 P1 Latency

Figure 4 does suggest however, that P1 latencies differed according to whether participants viewed a male or female body. This was confirmed by ANOVA, finding P1 latencies to be shorter in response to female bodies as compared to male bodies (100.133 ms vs. 104.855 ms) (F(1, 54) = 16.732, p < .001, η_p^2 = 0.237) across groups. This suggests that gender-sensitive responses may be seen in P1 time ranges. As to be expected, a significant between-subjects effect showed that P1 latencies to all bodies were shorter in ED participants (96.349 ms vs. 108.639 ms) (F(1, 54) = 10.023, p = 0.003, η_p^2 = 0.157), again supporting the idea that shortened P1 latencies may be related to ED symptomatology.

3.4.2.3 N1 Amplitude

Apparent differences in N1 amplitudes implicated in *Figure 4* were confirmed by ANOVA. There was a main effect of picture type (F(1, 54) = 25.631, p < .001, η_p^2 = 0.322), describing larger N1 amplitudes to female bodies in comparison to male bodies (-1.051 μV vs. -2.092 μV). This interacted with group (F(1, 54) = 7.081, p =

0.010, η_p^2 = 0.116), and pairwise comparisons of the estimated marginal means showed larger amplitudes to female than to male bodies in the ED group (-2.470 μV vs. -.870 μV) (F(1, 54) = 30.151, p < .001, η_p^2 = 0.358), but no such differences in the control group (-1.232 μV vs. -1.715 μV) (F(1, 54) = 2.561, p = 0.151, η_p^2 = 0.045). Nevertheless, the average amplitude of the component appears to be the same as there was no significant main effect of the between-subjects factor group. Overall, these patterns suggest that enhanced gender sensitivity in body-sensitive N1 amplitudes is related to ED symptomatology in women (both anorexic and bulimic participants showed this effects, see Table S7).

3.4.2.4 *N1 Latency*

As implicated in *Figure 4,* there were no differences in N1 latency when viewing male or female bodies, in either group. There was a significant between-subjects effect however, showing that overall, the N1 to bodies was faster in ED participants than in controls (153.470 ms vs. 164.954 ms) (F(1, 54) = 8.330, p = 0.006, η_p^2 = 0.134). This echoes previous suggestions that faster processing in both P1 and N1 time ranges may be related to ED symptomatology.

3.4.2.5 VPP Amplitude

Figure 5 below suggests a similar gender-sensitive effect for the ED group as that which was observed over the body-sensitive N1; this was not apparent in controls. ANOVA found a main effect of picture type (F(1, 53) = 6.549, p = 0.013, η_p^2 = 0.110),

showing that amplitudes to female body stimuli (1.657 μV) were significantly larger than amplitudes to male body stimuli (1.029 μV). A significant interaction with group was also found ($F(1, 53) = 4.596$, $p = 0.037$, $\eta_p^2 = 0.080$), with follow-up pairwise comparisons revealing the presence of this gender-sensitive effect in ED participants ($F(1, 53) = 11.075$, $p = 0.002$, $\eta_p^2 = 0.173$) but not in controls ($F(1, 53) = 0.069$, $p = 0.793$, $\eta_p^2 = 0.001$). This suggests that gender-sensitivity over the VPP is related to ED symptomatology (both anorexic and bulimic participants showed this effects, see Table S7).

Figure 5 here

3.4.2.6 VPP Latency

As suggested by *Figure 5,* ANOVA revealed no differences in VPP latency when viewing male or female bodies, in either of the groups. This suggests that the time course of body-sensitivity associated with this component is not modulated by gender or ED symptoms.

3.5 Correlations

3.5.1 Variables entered into Pearson's correlation

As there were significant differences between the groups on all eleven of the EDI-2 subscales, these scores were entered into the correlation as eleven variables (see also Eshkevari et al., 2012). Relationships between the subscales will not be reported because internal validity of the scale has been verified (e.g. Clausen et al., 2009; Eberenz & Gleaves, 1994; Nevonen & Broberg, 2001; Thiel & Paul, 2006). ANOVA results showed that early onset of visual P1 and N1 components might be characteristic of those who have experienced an ED. To quantify these effects, individual P1 and N1 peak latencies were averaged separately for body and house stimuli across electrodes P5/6, P7/8, PO5/6, PO7/8 and the resulting four variables entered into the correlation analysis. Gender-sensitive N1 and VPP peak amplitude differences were also implicated as characteristic of individuals who have experienced an ED. To reflect this, individual N1 and VPP peak amplitudes were averaged separately for male and female bodies across electrodes P5/6, P7/8, PO5/6, PO7/8 for the N1, and across electrodes Fz, F1/2, and F3/4 for the VPP. In both cases, amplitudes to male bodies were then subtracted from amplitudes to female bodies. For the N1, a more negative difference value is therefore indicative of gender-sensitivity towards higher N1 amplitudes in response to female bodies, whereas a more positive difference value is indicative of gender-sensitivity towards higher N1 amplitudes in response to male bodies. The opposite is true for the VPP. These difference values, representing gender-sensitivity for each component, were entered as two variables into the analysis. Weight and hours of weekly exercise also

differed between the groups, thus, a total of 19 variables were included in the Pearson's correlation.

3.5.2 EDI-2 subscale and ERP correlations

3.5.2.1 P1 latencies

A moderate, negative relationship between P1 latencies to houses and impulse regulation was found (r(54) = -.327, p = 0.014, see *Figure S1)*. Moderate, negative correlations were also found between P1 latencies to bodies and scores on the drive for thinness, interoceptive awareness and impulse regulation subscales (r(54) ≥ -.312, p ≤ .019, see *Figures S2 – S4).* This indicates that as P1 latencies to bodies got shorter, participants showed higher levels of preoccupation with their weight, lower ability and trust in recognising internal affective and bodily states, and poorer abilities to regulate impulse behaviour. Shorter P1 latencies to houses were also affiliated with poorer abilities to regulate impulse behaviour.

3.5.2.2 N1 latencies

Similar to the relationships found between EDI-2 scores and responses in the P1 time range, a moderate, negative relationship was found between N1 latencies to bodies and impulse regulation (*r*(54) = -.319, p = 0.016, see *Figure S5).* This shows that as N1 latencies got shorter, self-reported abilities to regulate impulse behaviour got poorer.

3.5.2.3 N1 gender-sensitive effect

There were no significant correlations between N1 gender-sensitivity and EDI-2 measures.

3.5.2.4 VPP gender-sensitive effect

Moderate, positive relationships were found between the gender-sensitive effect over VPP amplitudes and nine of the eleven EDI subscales, including drive for thinness, body dissatisfaction, ineffectiveness, interpersonal distrust, interoceptive awareness, maturity fears, asceticism, impulse regulation and social insecurity ($r(53) \geq .329$, $p \leq .022$, *see Figures S6 – S14).* This suggests that, as many of the cardinal symptoms of an ED increased, so did the difference between VPP amplitudes to males and females such that gender-sensitive responses were evident towards other women's bodies.

3.5.3 Correlations between sociodemographic variables and ERP effects

No significant relationships were found between weight and ERP effects or amount of weekly exercise and ERP effects. This suggests that sociodemographic group differences are not accountable for the ERP effects.

3.5.4 Correlations between ERPs

Latencies to all stimuli in the P1 time range were associated with the same changes in latency seen in the N1 time range. P1 latencies to house stimuli were strongly and positively associated with P1 latencies to body stimuli as well as N1 latencies to both body and house stimuli *(r*(54) ≥ .690, p <.001). It was also the case that P1 latencies to body stimuli were strongly and positively associated with N1 latencies to both house and body stimuli *(r*(54) ≥ .696, p <.001). There was also a strong, positive relationship between N1 latencies to body stimuli and N1 latencies to house stimuli (r(54) = 0.743, p<.001).

Amplitude effects did not correlate with the latencies of either component or each other.

4 Discussion

To the best of our knowledge, this is a novel study investigating the temporal dynamics of body- *and* gender- selective visual processing in observers at risk of body image disturbances, with the aim of identifying potential biomarkers of ED symptoms related to both anorexia and bulimia nervosa. P1, N1 and VPP responses to body and house stimuli over occipito-parietal and fronto-central sites were compared between women with ED history and healthy controls. This revealed that the entire P1-N1 complex was earlier in the ED group than in controls. Further comparisons were made between responses to male and female body stimuli in order to investigate gender selectivity during body perception. A gender-sensitive

effect was seen over N1 and VPP amplitudes in ED participants such that significantly larger component amplitudes were evident to female bodies in comparison to male bodies for the ED group but not controls. Findings were then correlated with scores on each of the EDI-2 subscales to assess the relationship with ED symptomatology. An earlier P1-N1 complex was associated with higher scores on several EDI-2 subscales, whilst gender selectivity in VPP amplitudes was related to all but two of the EDI-2 subscales. Ultimately, atypical ERP effects increased alongside the severity of ED symptoms and may therefore serve as potential neural markers of ED symptomatology.

Clear differences were also found between ED participants and controls with regards to how they felt about their own body. The ED group scored significantly higher on all EDI-2 subscales, indicating more unhealthy attitudes and behaviours towards their own body. There was no evidence that those with an ED and controls felt differently about other bodies however, as valence and arousal ratings in response to body stimuli did not differ between the groups. This contrasts with other findings that report higher arousal ratings for overweight body stimuli in bulimic individuals and higher aversion ratings for bodies in anorexic participants (Mai et al., 2015; Uher et al., 2005). However, Spring and Bulik (2014) found no differences in affective responses to body stimuli between recovered anorexic participants and controls. It is likely then, that as the majority of ED participants in our study reported partial recovery, this accounts for why body stimuli were not rated differently between the ED group and controls. This finding is of interest as it suggests that bodies are only abnormally salient during the acute stages of an ED. Further investigations would thus benefit from identifying when bodies begin to lose their emotional salience

during recovery from an ED. With that in mind, it may also be of interest to identify at what point bodies begin to acquire emotional salience during the development of an ED.

The following sections will now proceed to discuss each of the ERP effects in turn, and to assess, where applicable, their potential as biomarkers for ED symptomatology.

4.1 Evidence for ERP body-sensitivity in ED participants and controls

In line with previous literature (see Peelen & Downing, 2007 for review), a body-sensitive N1 amplitude enhancement was found over occipito-parietal electrodes bilaterally. A body-sensitive VPP enhancement was also observed over fronto-central electrodes, supporting evidence that the N1 and the VPP may be generated from the same neural sources (cf. Eimer, 2000b; Joyce & Rossion, 2005; Sadeh et al., 2011; Taylor et al., 1999). We also found shorter P1 latencies in response to bodies compared to houses. This suggests that there may be an early distinction between bodies and other stimuli in the P1 time range. Early effects of face-sensitivity have also been seen over the P1 (Itier & Taylor, 2004, 2004b; Rossion et al., 1999; Rossion et al., 2000) so this finding in response to bodies is perhaps unsurprising. Our results indicate then, that the P1 effects may be a global response to bodies, reflecting the perception of a stimulus as a body, in a similar way to the process that has been proposed for faces (Itier & Taylor, 2004b).

N1 and VPP latencies on the other hand, were both longer to body stimuli in comparison to houses. Differences in N1 and VPP latency between bodies and other stimuli seem to be relatively undiscussed, although Stekelenburg and de Gelder (2004) describe the N1 to bodies as peaking earlier than the N1 to objects. The difference between findings might be attributed to the difference in stimuli as studies have consistently found longer N1 latencies to bodies without heads compared to bodies with heads (faces masked) (Alho et al., 2015; Minnebusch et al., 2010; Minnebusch et al., 2009). Further studies including both types of body stimuli and objects are therefore needed to verify the exact time course of body processing in the N1 time range.

4.2 Visual processing differences between ED women and controls

One of the most important findings to emerge from the present study was the difference in the temporal dynamics of the P1 and N1 between groups. To the best of our knowledge, this is the first study to find that the temporal dynamics of early visual processing are related to the severity of ED symptoms.

4.2.1 Early P1-N1 complex found in ED participants

In the ED group, P1 and N1 responses to all visual stimuli were significantly earlier than those elicited by the control group. While no previous ERP study has reported on the P1, our N1 latency shifts clearly differ from Mai et al. (2015), who report no N1 differences between bulimic and control participants whilst viewing overweight body

stimuli. Our findings also differ from Li et al. (2015), who found longer N1 latencies to both faces and houses in anorexic participants compared to controls.

Unlike Li et al. (2015), the present study shows a clear relationship between P1 and N1 latency and several measures of ED symptoms (drive for thinness, interoceptive awareness and impulse regulation). Responses to both bodies and houses in the P1 time range were linearly associated with impulse regulation scores such that early responses were indicative of poorer abilities to regulate impulsive behaviour. This relationship remained only for body stimuli in the N1 time range. As P1 responses are thought to primarily reflect processing of the low-level visual properties of a stimulus (Latinus & Taylor, 2006; Rossion & Caharel, 2011), whereas the N1 is thought to primarily reflect structural encoding processes (Eimer, 2000c; Soldan et al., 2006), poor impulse regulation may therefore be associated with atypical low-level visual analysis of a stimulus but only with atypical structural encoding of bodies. Early P1 responses to bodies were also associated with a greater drive for thinness and less ability to recognise internal bodily states. Thus, the relationship between aberrant early visual processing of bodies and ED symptoms is more extensive than the relationship between aberrant early visual processing of houses and ED symptoms. This suggests that there may be general visual processing differences that are amplified for disorder-relevant stimuli in individuals who have experienced anorexia or bulimia nervosa.

As early P1 responses have been associated with the detection of fear in body stimuli (see Minnebusch & Daum, 2009 for review), it may be possible that the latency shifts we observed occurred because ED participants found the stimuli

emotionally rousing. Two of our findings challenge this explanation however. First, valence and arousal ratings for body stimuli did not differ between the groups. Second, P1 and N1 responses to all stimuli were faster in those with EDs, not just those to bodies. In line with this, it may be posited that, due to the random nature of our stimulus presentation, ED participants were in a heightened state of arousal or attention throughout the EEG task, as they could not predict the occurrence of the more emotionally salient body pictures. This, and not the pictures themselves, may have evoked early visual responses, (see also Gazzaley, Cooney, McEvoy, Knight, & D'Esposito, 2005) explaining why the entire P1-N1 complex was early and not just responses to body stimuli. However, van Heijnsbergen et al. (2007) reported early P1 and early VPP responses to fear, whilst Stekelenburg and de Gelder (2004) also found early emotional modulations of VPP amplitude. So if a general state of arousal accounts for our results then we would also expect to see latency differences over the VPP for the ED group, which was not the case. Further investigations, perhaps employing a blocked design, are thus clearly necessary to determine the underlying mechanisms of these latency effects. Moreover, it is possible that explicit self-report ratings were not sensitive enough to detect differences in arousal and affect between the populations. Bodies are clearly salient stimuli for women with eating disorders and consequently, the threshold for arousal and valence are likely to be different in those who are partially recovered compared to controls. For example, a woman who has experienced an ED reporting feeling ‘slightly’ aroused to body stimuli might be the equivalent of women without an ED reporting to be ‘extremely’ aroused. This is because in comparison to how salient bodies are to individuals in the grips of the illness, they are likely to be less prominent after some recovery. As far as we know, this has not been investigated. Therefore, it may be beneficial to assess autonomic

nervous system activity as an additional, more objective indication of emotional arousal to disorder-relevant stimuli when making comparisons between ED participants and controls, especially if those with EDs are not in the acute stages of illness. Nevertheless, the temporal dynamics of the P1-N1 complex appears to be a meaningful neural marker of ED symptoms.

4.2.2 No differences in body-sensitive amplitudes between ED participants and controls

As expected, body-sensitivity was observed in N1 and VPP amplitudes but not in P1 amplitudes. The extent of these effects did not differ between the groups and no general amplitude differences were found between the groups for any of the components. This suggests that the magnitudes of P1, N1 and VPP responses, as well as that of body-sensitive effects, are not modulated by the experience of ED symptomatology.

Although not directly tested for, given the findings of Li et al. (2015), there was a possibility of observing amplitude differences between the groups that would perhaps indicate configural processing abnormalities in ED populations. Specifically, Li et al. (2015) argued that larger visual P1 amplitudes are indicative of more configural processing (Goffaux, Gauthier, & Rossion, 2003; Nakashima et al., 2008), and as anorexic participants in their study displayed reduced visual P1 and N170 amplitudes, this indicates a configural-processing deficit.

As no group differences in amplitude measures were found, does this imply that weight-restored ED participants do not have problems with configural processing? We believe such an interpretation should be drawn with caution. First and foremost, there is still debate as to whether bodies, especially those without head, recruit configural-processing mechanisms in a similar way to faces, or whether they are processed on a feature-by-feature basis similarly to objects (e.g. Itier & Taylor, 2004, 2004b; Rossion et al., 1999; Rossion et al., 2000). The difference between stimulus sets must therefore be considered as a possibility for the difference between findings. For example, if configural processing mechanisms are not elicited in response to (headless) bodies, or indeed if the processes are different, as has been suggested (e.g. Minnebusch & Daum, 2009; Minnebusch et al., 2009) then participants in this study would have been engaging in feature-based processing throughout. Thus, without a stimulus category such as faces to prompt configural processing, any configural processing deficit in ED participants would not have been measured in our study.

However, at least one study has found evidence for the configural processing of headless body stimuli over P1 (Minnebusch et al., 2010) as well as N1 amplitudes (Minnebusch et al., 2010; Soria Bauser & Suchan, 2013). In addition, if the bodies in our study were being processed like objects, we would expect to see no body-sensitive enhancements of the N1. As this was not the case, we may assume that headless bodies in our study were processed configurally. Thus, it is at least plausible that similar ERP effects of configural processing should be seen for bodies as are seen for faces, especially as body processing mechanisms are thought to arise from distinct but adjacent neural sources (Sadeh et al., 2011). We therefore

propose that future studies should explicitly test for the neural correlates of configural processing deficits in EDs, such as by inverting or scrambling stimuli, before any firm conclusions can be drawn about potential configural processing deficits in these populations.

4.3 Evidence for ERP gender-sensitivity in ED participants but not controls

As far as we are aware, this is the only study to-date that investigated gender-sensitive visual body processing in EDs, and one of few studies to investigate gender-sensitive visual body processing in healthy women. As such, gender-sensitive effects were observed over N1 and VPP amplitudes in the ED group but not in the control group. This was reflected as a significant amplitude enhancement in response to viewing other women's bodies compared to men's bodies.

Observing no N1 gender-sensitivity in the control group supports what is reported by Hietanen and Nummenmaa (2011) but challenges results from Alho et al. (2015). Both papers argue that amplified N1 responses to nude female bodies are early affective responses that may be related to sexual drives and mating behaviours in men and women alike. Alho et al. (2015) elaborate by suggesting that the presence of any nude stimulus, irrespective of gender, might be enough to trigger sexual responses in women. Even if this were true, this does not explain why they found enhanced amplitudes to clothed female bodies in comparison to clothed male bodies. Irrespective, their explanation would predict similar N1, and by extension,

VPP, responses to male and female body stimuli, which is exactly what our study has found for the healthy female control group.

Previous studies have not considered that female bodies might be salient stimuli for women in ways that are not driven by the primal urge to procreate. Findings from the present study thus suggest an alternative interpretation to that of Hietanen and Nummenmaa (2011) and Alho et al. (2015). In particular, the clear differences between N1 gender-sensitivity in the ED group and controls indicate that the effect is a potential biomarker of ED symptomatology in women. The mechanisms underpinning the effect are unclear, however, as N1 gender-sensitivity did not correlate with EDI-2 measures. Moreover, previous studies have consistently found the effect in men so any interpretation must take this into account.

We propose objectification of the female form as a possible explanation, because enhanced body-sensitive N1 amplitudes are associated with a switch from configural to feature-based processing mechanisms in ERP inversion studies (see Minnebusch & Daum, 2009 for review). Individuals showing enhanced amplitudes to female bodies relative to male bodies may therefore initially recruit configural processing mechanisms upon recognising the stimulus as a body, but then switch to feature-based processing when recognising the body as female. In other words, these individuals perceive women's bodies like objects.

This is supported by western societal norms that encourage the objectification of female bodies (Jones, 2001), which is evident in men more so than women (Strelan & Hargreaves, 2005). Additionally, women without body image disturbance are not

found to objectify women's bodies more so than men's bodies (Strelan & Hargreaves, 2005), perhaps explaining why controls do not show gender-sensitivity in the N1 time range. Furthermore, when women do objectify other women's bodies, this is related to self-objectification and body dissatisfaction (Strelan & Hargreaves, 2005), both of which are ED traits (Calogero, Davis, & Thompson, 2005). By this reasoning, it is understandable that gender-sensitive N1 effects did not correlate with EDI-2 measures, as this questionnaire does not assess objectification. Future studies of gender-sensitive body processing should therefore include measures of objectification in order to test this potential explanation.

Alternatively, it is possible that top-down attentional processes may explain the effect, as women's bodies may be particularly salient to those with EDs (e.g. Horndasch et al., 2015; Vocks et al., 2010). Although studies in the face processing literature often do not find effects of attention within the N1 time range (e.g. Carmel & Bentin, 2002; Lueschow et al., 2004), it is not altogether unheard of (Crist, Wu, Karp, & Woldorff, 2008; Sreenivasan, Goldstein, Lustig, Rivas, & Jha, 2009). Thus, despite our efforts to reduce attention effects with an oddball detection task, hypervigilance to relevant body information in EDs (Vitousek & Hollon, 1990), in this case the female form, could have resulted in a greater allocation of attentional processes to other women's bodies than men's bodies, leading to the observed N1 enhancement. This possibility should be addressed in future investigations.

We also observed a novel gender-sensitive effect in VPP amplitudes for the ED group, but not for the control group. This did not relate to gender-sensitive N1 amplitudes. The recruitment of extra neural resources over fronto-central sites whilst

ED participants viewed same-sex stimuli may therefore represent processing mechanisms that are at least partly separable from those occurring more posteriorly. Importantly, the effect was positively associated with all but two of the EDI-2 subscales. This is a strong indication that VPP gender-sensitivity is a biomarker of ED symptomatology.

As it is argued that body-sensitive VPP amplitudes are modulated by fear (Stekelenburg & de Gelder, 2004; van Heijnsbergen et al., 2007), gender-sensitivity found over the VPP in the ED group might indicate that other women's bodies are a source of anxiety for this population. Moreover, Stekelenburg and de Gelder (2004) found that fearful body expressions modulated VPP amplitudes but not N170 amplitudes. This indicates that the body-sensitive N1 is reflective of structural encoding processes whilst the body-sensitive VPP is (additionally) indicative of early emotion processing. As such, whilst N1 gender-sensitivity might be informative of the differences in structural encoding of gender body stimuli between controls and those with EDs, VPP gender-sensitivity could be an insight into the affective processes concerned with this. With that in mind, our results suggest that ED women may not only encode the structure of other women's bodies differently to men's bodies, but at a neural level, other women's bodies are being recognised as emotionally salient.

It is possible that the foundations of such emotional responses could be rooted in social comparison behaviour. Evidence from Vocks et al. (2010) strongly supports this idea as enhanced limbic activity was found in anorexic participants during the viewing of other women's bodies. The authors suggest that this represents a stronger emotional response and perhaps more vigilance to other women's bodies

that is likely due to social comparison processes. Corning, Krumm, and Smitham (2006) further support this, as women with ED symptoms evaluated their bodies more negatively during same-sex social comparisons than women without ED symptoms. Similarly, eye-tracking has shown that those with bulimia nervosa engage in upward comparisons whilst fixating for longer on bodies with a lower BMI, and reporting more body dissatisfaction after the comparison process (Blechert et al., 2009). Social-self concerns have also been linked to body dissatisfaction in bulimic individuals (Striegel-Moore, Silberstein, & Rodin, 1993) with such comparative processes reportedly inducing body-focused anxiety even in asymptomatic populations (Halliwell & Dittmar, 2004). However, as our design did not allow for, or indeed encourage, extensive rumination over body stimuli, it is unlikely that direct social comparison processes drive this effect. Instead, evaluative conditioning theory would dictate that female bodies might become affective stimuli if these anxiety-inducing comparisons are made frequently enough (Hofmann, De Houwer, Perugini, Baeyens, & Crombez, 2010). It is possible then, that the learned salience of other women's bodies, rather than direct social comparison, accounts for the gender-sensitive VPP effect observed in those with EDs. This may also explain why VPP and N1 responses both show gender-sensitivity without being related; essentially they are different mechanisms contributing to the same process.

5 Limitations

The interpretation of our findings must take into account some limitations. Firstly, participants were not clinically assessed for anorexia, bulimia, or other mental health issues. It is therefore possible that other mental health conditions were not disclosed

during the recruitment procedure or that ED participants had not experienced the illness they claimed to. However, we were careful to advertise in such a way that potential participants did not know exact exclusion criteria and were thus encouraged to disclose everything. Furthermore, we did not advertise the amount of money participants would be reimbursed with, in order to discourage those who might apply solely for the monetary gain. The ED group also scored significantly higher than controls on all EDI-2 subscales, which suggests that those participants were drawn from an ED population. Nonetheless, future replications should aim to clinically assess participants for EDs and other mental health conditions.

Secondly, we chose to combine data from anorexic and bulimic participants into one overarching ED group, which it could be argued, might reduce disease-specific findings. However, whilst anorexia and bulimia should be understood as separate illnesses (American Psychiatric Association, 2013) there is also evidence for shared pathologies (see O'Brien & Vincent, 2003 for review). In our study, the absence of differences between anorexic and bulimic participants on sociodemographic factors (Table S1) and ERP effects (Table S7) justified combining their data (as in Eshkevari et al., 2012; Eshkevari, Rieger, Longo, Haggard, & Treasure, 2014; Horndasch, Kratz, et al., 2012 for example) .

A third limitation relates to the difference in protocol, as control participants completed 30 more trials than the ED group. It could therefore be argued that fatigue was responsible for the results rather than genuine group differences. However, as 30 trials would have taken less than a minute to complete, we feel that fatigue is an unlikely explanation for the difference between groups. Similarly, ED participants

were presented stimuli three times whereas controls were only presented stimuli twice. Although not presented in succession, such repetition of stimuli could have led to a decrease in component amplitudes, known as repetition suppression (see e.g. Grill-Spector, Henson, & Martin, 2006 for review) and perhaps altered latencies (see the neural 'facilitation' model reviewed in Gotts, Chow, & Martin, 2012) for the ED group compared to the control group. There are several reasons why we do not think the extra repetition of stimuli for the ED group could explain our results. First and foremost, Henson (2012) argues that attenuated neural responses may be due to shorter duration of neural activity, and thus where latency differences have been observed due to repetition this is always accompanied by altered amplitudes (e.g. Itier & Taylor, 2004). Whilst latency differences were observed between the ED group and controls in this study, reduced components were not found. Moreover, these latency shifts were related to EDI-2 subscales, which would unlikely occur if they were an artefact of the task. Additionally, repetition only affects ERPs from 200 ms onwards if there is at least one item in between the repeated stimuli (see Grill-Spector et al., 2006 for review) and all effects reported here fall within the first 200ms post stimulus onset.

It should also be noted that control participants viewed stimuli that B-eat considered potentially triggering to those with an ED. Consequently, it could be argued that these stimuli are generally more arousing, which may have led to altered ERP effects between the groups. Arousal is usually found to modulate ERP amplitude, not latency (e.g. Junghöfer, Bradley, Elbert, & Lang, 2001; Kissler, Herbert, Winkler, & Junghofer, 2009; Olofsson & Polich, 2007; Rozenkrants, Olofsson, & Polich, 2008), with effects often evident on later, rather than earlier components (e.g. Kissler et al.,

2009). As a result, it seems unlikely that the affective nature of the additional stimuli viewed by controls could be responsible for the latency shifts observed between groups. Moreover, if the gender-sensitive effects reported were a manifestation of such arousal then we might expect controls, not those with an ED, to elicit enhanced amplitudes to bodies (e.g. to female in comparison to male bodies). Further to this, there were no differences in body ratings indicative of a general increased state of arousal in controls. Therefore, whilst we suggest that future studies adhere to comparable protocol between groups, we are confident that the differences in protocol in this study could not account for the ERP differences observed between groups.

It is also important to take into account that we did not investigate whether sexual orientation was related to the gender-sensitive effects we observed in ED women. Hietanen and Nummenmaa (2011) suggest that the sexual preference of the observer effects gender-sensitive N1 responses, as they found that homosexual men did not elicit enhanced amplitudes to female bodies, whereas homosexual women did. However, they did not include heterosexual men or women in their analysis and as such, the effect of sexual orientation is not directly compared, it is only inferred. Moreover, sample sizes were very small; data from only four men and six women were analysed. It is therefore likely that statistical power was not sufficient to detect an effect in the male sample. In their later study (Alho et al., 2015), heterosexual men and women both elicited enhanced body-sensitive N1 responses to female bodies in comparison to male bodies. Here they argued that sexual orientation does not matter in the case of the women, as any sexual stimulus is likely salient to them. This not only directly contradicts claims from their first study but also does not hold

as a theory because in fact, it suggests that no gender differences should be found in female observers' body-sensitive neural responses. As it is unlikely that we recruited 27 heterosexual controls and 29 homosexual ED participants (Feldman & Meyer, 2007), which would account for the observed differences in gender-sensitive processing, we are confident that sexual orientation cannot explain all of our gender-sensitive findings. Moreover, as evidence is mixed with regards to the relationship between sexual orientation and gender-sensitive body processing (Alho et al., 2015; Hietanen & Nummenmaa, 2011) a purely sex-related explanation of this effect seems unsatisfactory. Nonetheless, future studies should seek to investigate the relationship between ED symptomatology, sexual orientation and gender-sensitive body processing.

As a final limitation, we used an oddball detection task (similar to van Heijnsbergen et al., 2007) to reduce the attention paid to bodies, as studies have shown that those with EDs may visually analyse bodies differently to controls (e.g. Blechert et al., 2009; Horndasch, Kratz, et al., 2012; Jansen et al., 2005; Vocks et al., 2010) . It must be discussed then, that findings might differ when bodies are actively, rather than passively viewed.

Studies have shown that headless bodies evoke selective activity in lateral (EBA) and ventral (FBA) occipitotemporal cortex regardless of whether they are passively viewed (Downing et al., 2001; Morris, Pelphrey, & McCarthy, 2006; Saxe et al., 2006) or viewed in order to classify, discriminate or memorise them (see de Gelder et al., 2010 for an overview of tasks; Downing et al., 2001; Hodzic, Kaas, Muckli, Stirn, & Singer, 2009; Peelen & Downing, 2007; Schwarzlose et al., 2005; Taylor et

al., 2007). ERP findings also suggest that regardless of the task, structural encoding of bodies typically occurs in the N1 time range (Minnebusch & Daum, 2009; Minnebusch et al., 2010; Stekelenburg & de Gelder, 2004). The same body-sensitive N1 component is affected by body distortion during passive viewing of headless bodies (Gliga & Dehaene-Lambertz, 2005) and during a discrimination task with similar, headless bodies (Soria Bauser & Suchan, 2013). Irrespective, fMRI or ERP studies have not addressed the possibility that bodies may be processed *more* selectively when they are task-relevant than when they are ignored or passively viewed. While future studies should directly compare the early cortical effects of attending and not attending to bodies on body-sensitive processes and on ED-related group differences, it seems unlikely that the task irrelevance of bodies in the present study would suffice to explain all of our findings.

6 Conclusions

This is the first study to demonstrate that the time course of visual processing in both anorexia and bulimia occurs earlier than in controls. Moreover, we found that amplified responses to female relative to male bodies are evident posteriorly in N1 time ranges, and reflected frontocentrally over VPP. Neuroimaging studies have already shown that the EBA is underactive and maladapted in those with EDs (Suchan et al., 2010; Suchan et al., 2015; Uher et al., 2005). It has also been shown that bulimic women display an attentional bias for processing overweight body stimuli (Mai et al., 2015) whereas those with anorexia and body dysmorphic disorder might engage in atypical visual processing of faces (Li et al., 2015). The present results therefore add to this body of literature, providing support for the hypothesis that

visual body processing is modulated by body image. The evidence for this in the present study is particularly compelling as general posterior latency effects and anterior gender-sensitive amplitude effects systematically varied with ED symptomatology.

We propose that these differences in electrophysiological body processing may serve as potential biomarkers of EDs, offering an insight into disorder-relevant cognitive processes. These processes likely include social comparison and body surveillance behaviours that ultimately result in feature-based and anxious affective processing of bodies, and perhaps in giving other women's bodies an unusually salient status during structural analysis. Future studies should seek to replicate these findings with measures of social comparison tendencies and implicit anxiety (i.e. physiological arousal) in response to viewing body stimuli. Modulation of visual body processing in EDs should also be investigated in clinical and fully recovered populations, so as to profile whether these differences are characteristic of ED symptomatology or represent on-going maladaptation. Should it be the former, then such biomarkers hold the potential to identify 'at risk' individuals, whilst offering an insight into the efficacy of treatment for individuals in the acute stages of illness.

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Author contributions

K.G. and H.G. conceived and designed the study, K.G. conducted the study, K.G., H.G. and S.K analysed the results and wrote the paper.

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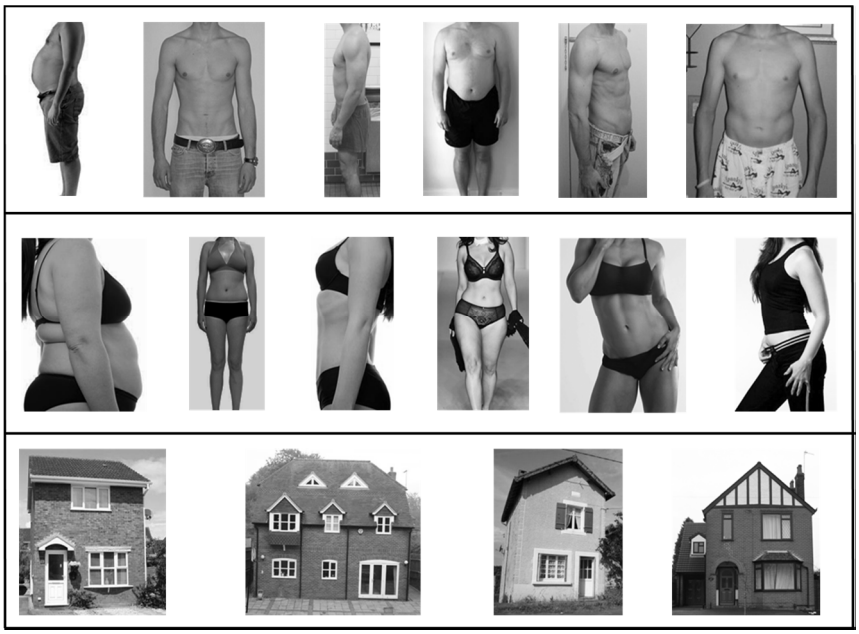

Figure 1. Example stimuli controlled for overall image brightness. (Top to bottom: male bodies, female bodies, houses).

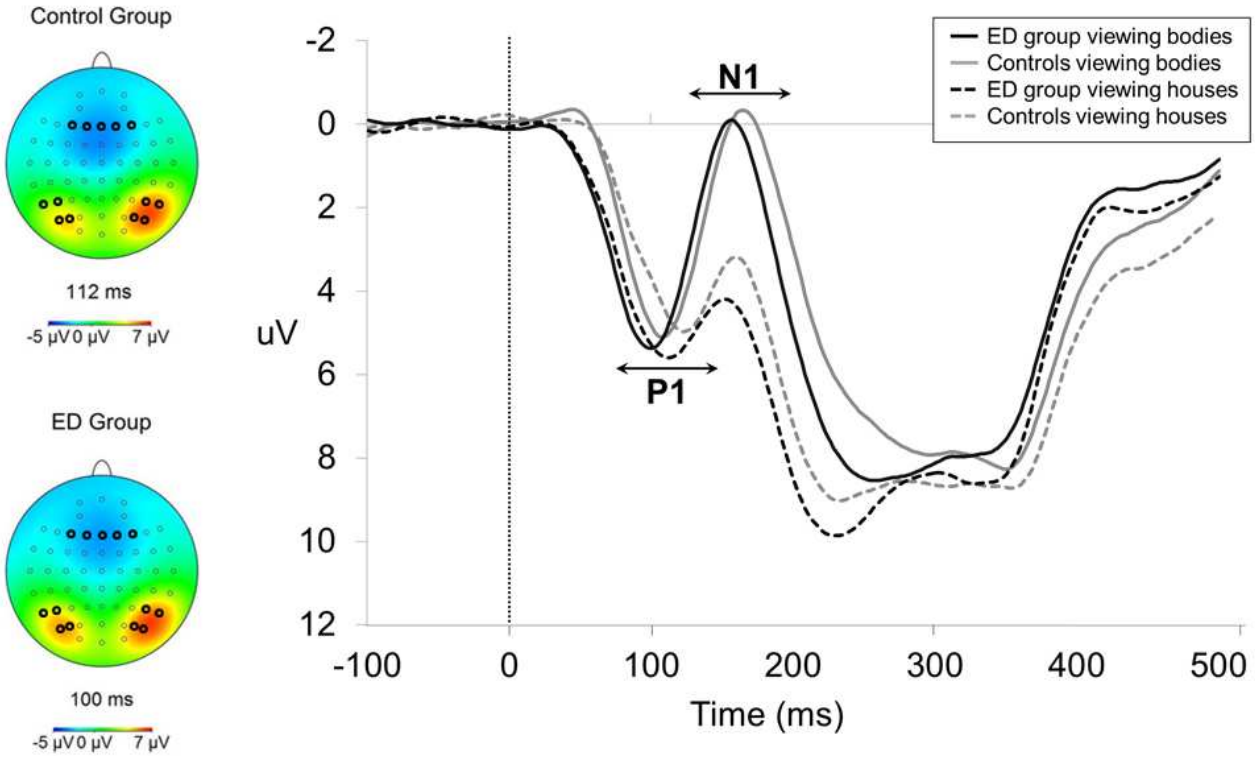

Figure 2. Left panel shows voltage maps for the time window of the visual P1 component (Controls 112ms, ED participants 100ms), collapsed over viewing conditions. Visual N1/VPP, which P1-N1 peak-to-peak amplitudes indicated was maximal over similar regions, has not been illustrated because traces remained in the positive range throughout (120ms – 190ms; see right panel) with an additional strong frontal negativity that decreased the visibility of N1 topographies. Anterior electrodes analysed for VPP and posterior electrodes analysed for P1/N1 have been highlighted. The right panel shows grand averaged ERP responses during house and body viewing (ED participants in black, controls in grey) collapsed over electrodes P5/6, P7/8, PO5/6, PO7/8. A body-sensitive N1 response is evident in both groups and shorter P1 and N1 latencies to all stimuli can be seen in the ED group.

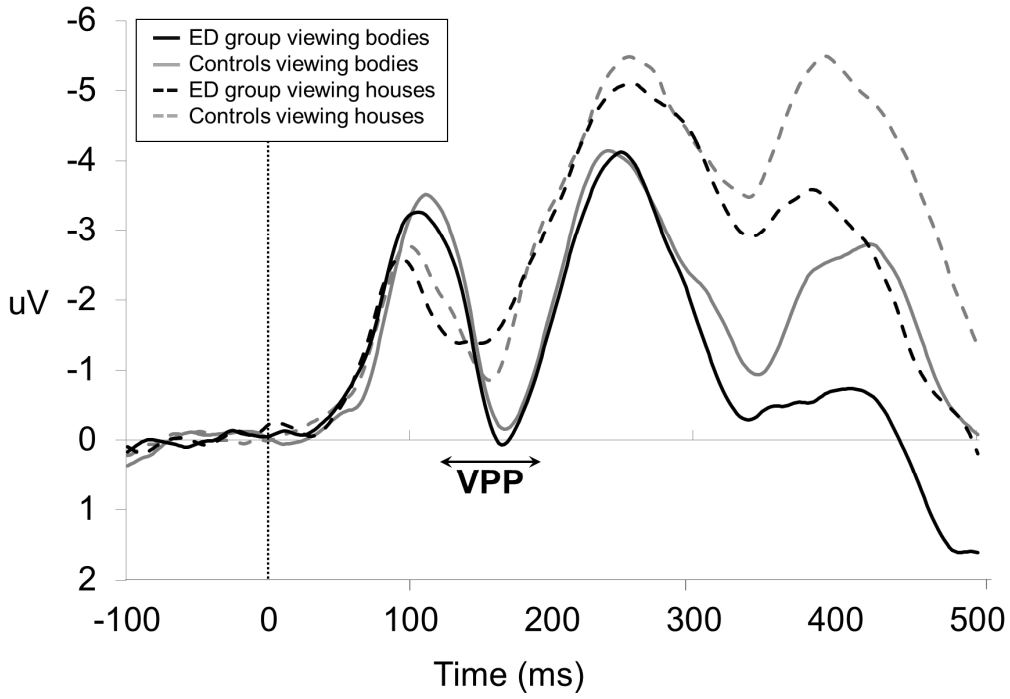

Figure 3. Grand averaged ERP responses depicting house and body viewing (ED group in black, controls in grey) collapsed over electrodes Fz, F1/2, F3/4, showing VPP latency differences between stimuli and higher VPP amplitudes to bodies in both groups.

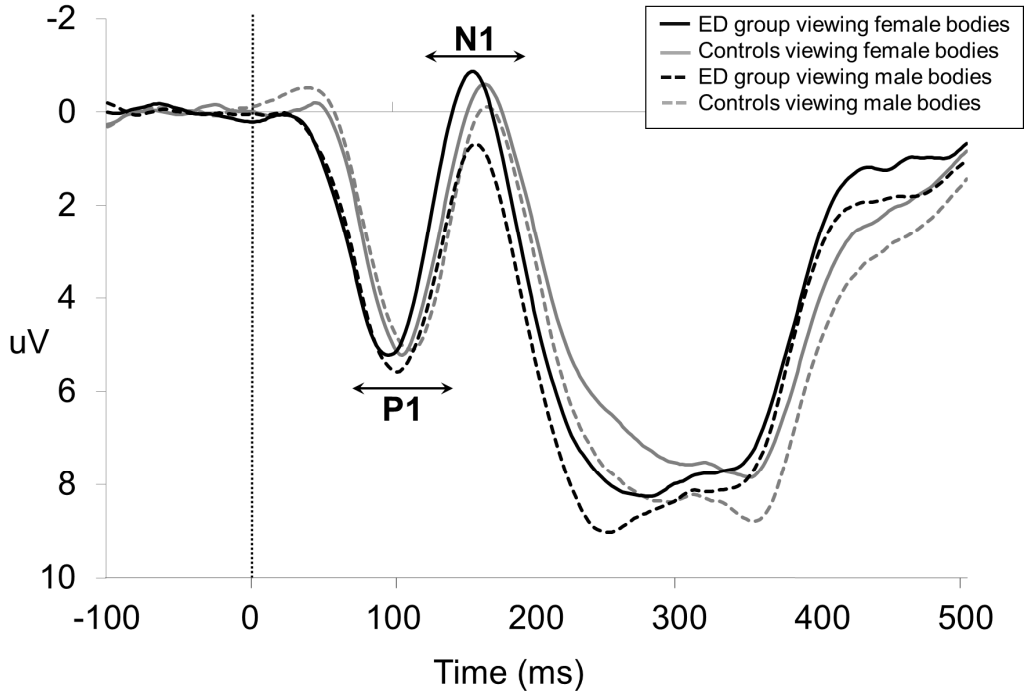

Figure 4. Grand averaged ERP responses depicting male and female body viewing separately (ED group in black, controls in grey) collapsed over electrodes P5/6, P7/8, PO5/6, PO7/8. An enhanced gender-sensitive effect in ED participants is evident in the ERP amplitudes in the N1 time range.

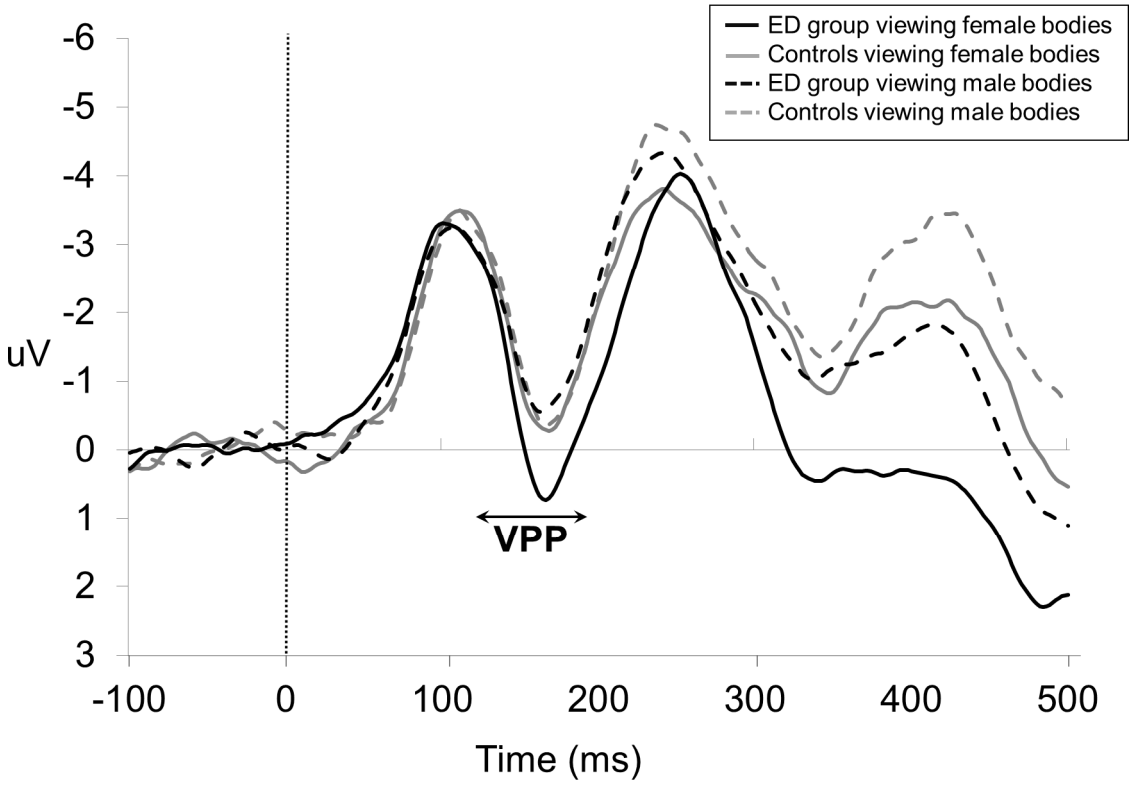

Figure 5. Grand averaged ERP responses depicting male and female body viewing separately (ED group in black, controls in grey) collapsed over electrodes Fz, F1/2, and F3/4. Increased gender selectivity in VPP amplitudes in the ED group is clear.

Supplementary Materials for

Evidence for ERP biomarkers of eating disorder symptoms in women

Katie Groves*, Steffan Kennett, and Helge Gillmeister

Department of Psychology, University of Essex, Colchester, UK

Correspondence to: kegrov@essex.ac.uk

This PDF file includes:

Tables S1 –S7
Figs. S1 - S14 with ED data in black and control data in grey. With regards to VPP gender-sensitivity, a more positive difference value is indicative of higher VPP amplitudes in response to female bodies as compared to male bodies, whereas a more negative difference value is indicative of gender-sensitivity towards higher VPP amplitudes in response to male bodies as compared to female bodies.

Table S1.

Average demographic information, EDI-2 scores, valence and arousal ratings and ERP latencies for anorexic and bulimic participants.

	Anorexic participants (N=15)	Bulimic participants (N=14)	T-test results
Age (years)	23.27 (8.71)	24.92 (8.16)	*t*(27) = .529, p = .601
Height (km)	1.65 (.05)	1.67 (.078)	*t*(27) = .523, p = .605
Weight (kg)	56.39 (8.95)	61.68 (9.25)	*t*(27) = 1.565, p = .129
BMI	20.60 (2.38)	22.21 (2.25)	*t*(27) = 1.868, p = .073
Weekly exercise (hrs)	6.00 (4.00)	5.61 (3.62)	*t*(27) = .277, p = .784
Drive for Thinness	10.87 (5.17)	13.07 (5.73)	*t*(27) = 1.090, p = .286
Bulimia	4.07 (4.13)	8.71 (6.85)	*t*(27) = 2.229, p = .034**
Body Dissatisfaction	14.33 (7.25)	15.00 (7.08)	*t*(27) = .250, p = .804
Ineffectiveness	9.60 (8.27)	10.29 (7.76)	*t*(27) = .230, p = .820
Perfectionism	9.80 (3.75)	8.36 (5.11)	*t*(27) = .872, p = .391
Interpersonal Distrust	6.13 (3.72)	5.43 (5.33)	*t*(27) = .415, p = .681
Interoceptive Awareness	12.27 (7.99)	10.07 (7.18)	*t*(27) = .776, p = .444
Maturity Fears	9.07 (7.27)	7.07 (7.21)	*t*(27) = .741, p = .465
Ascetism	9.87 (4.53)	8.21 (3.51)	*t*(27) = 1.091, p = .285
Impulse Regulation	10.67 (7.56)	7.21 (5.83)	*t*(27) = 1.370, p = .182
Social Insecurity	8.87 (4.91)	7.86 (6.05)	*t*(27) = .495, p = .625
Total EDI-2 score	105.53 (49.91)	101.29 (47.76)	*t*(27) = .234, p = .817
Valence to male bodies	4.62 (1.04)	4.72 (.59)	*t*(27) = .311, p = .758
Valence to female bodies	4.74 (.78)	4.82 (.75)	*t*(27) = .293, p = .771
Arousal to male bodies	5.00 (1.00)	5.28 (1.28)	*t*(27) = .649, p = .522
Arousal to female bodies	4.97 (1.18)	5.09 (.99)	*t*(27) = .299, p = .767
Visual P1 latency (ms)	100.21 (18.59)	99.92 (17.92)	*t*(27) = .042, p = .967
Visual N1 latency (ms)	147.99 (18.42)	152.13 (18.42)	*t*(27) = .604, p = .551
Visual VPP latency (ms)	150.37 (25.90)	161.71 (27.58)	*t*(26) = 1.122, p = .272
Visual P1 amplitude (μV)	8.32 (3.33)	6.06 (2.55)	*t*(27) = 2.039, p = .051
Visual N1 amplitude (μV)	1.06 (2.53)	-.25 (2.55)	*t*(27) = 1.378, p = .179
Visual VPP amplitude (μV)	.87 (3.30)	1.25 (3.68)	*t*(26) = .288, p = .755

Note. Standard deviation in parentheses. **significant at .05 level.

Table S2. *Mixed factorial ANOVA results for valence and arousal scores to body stimuli. Group as the between-subjects factor (control vs. ED) and gender (male body vs. female body) as the within-subjects factor.*

	Valence			Arousal		
	F-value	*p*-value	η_p^2	*F* -value	*p*-value	η_p^2
Gender	7.924	.009**	.119	.587	.447	.011
Gender*Group	2.184	.145	.039	3.472	.068	.060
Group	.232	.632	.004	.002	.967	.060

Note. **significant at .05 level.

Table S3.

Mixed factorial ANOVA results assessing for body-sensitivity in P1, N1 and VPP amplitudes (μV). Group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses – see method section)

	P1			N1			VPP		
	F-value	*p*-value	η_p^2	*F* -value	*p*-value	η_p^2	*F*-value	*p*-value	η_p^2
Picture type	.468	.497	.009	88.288	<.001**	.620	7.441	.009**	.125

Picture*Group	.094	.760	.002	2.327	.133	.041	.001	.974	<.001
Hemisphere	28.370	<.001**	.344	.827	.367	.015	-	-	-
Hem*Group	2.349	.131	.042	.382	.539	.007	-	-	-
Electrode	30.659	<.001**	.362	37.573	<.001**	.410	1.705	.187	.032
Electrode*Group	.270	.761	.005	.222	.831	.004	.899	.409	.017
Picture*Hem	.703	.405	.013	.453	.504	.008	-	-	-
Picture*Hem *Group	.957	.332	.017	.526	.471	.010	-	-	-
Picture*Electrode	6.071	.004**	.101	7.616	<.001**	.124	1.672	.199	.031
Picture*Electrode* Group	.203	.800	.004	.854	.445	.016	.311	.681	.006
Hem*Electrode	15.439	<.001**	.222	2.693	.065	.011	-	-	-
Hem*Electrode *Group	.587	.546	.011	.588	.578	.011	-	-	-
Picture*Hem *Electrode	4.002	.014**	.069	18.976	<.001**	.260	-	-	-
Picture*Hem *Electrode*Group	.256	.821	.005	.268	.757	.005	-	-	-
Group	1.763	.186	.032	.201	.656	.004	.197	.659	.004

Note. **significant at .05 level.

Table S4.

Mixed factorial ANOVA results assessing for body-sensitivity in P1, N1 and VPP latency (ms). Group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses – see method section)

	P1			N1			VPP		
	F-value	*p*-value	η_p^2	*F* -value	*p*-value	η_p^2	*F*-value	*p*-value	η_p^2
Picture type	24.217	<.001**	.310	17.625	<.001**	.246	52.966	<.001**	.505
Picture*Group	<.001	.986	<.001	.381	.540	.007	.318	.575	.006
Hemisphere	.532	.473	.010	.387	.536	.007	-	-	-
Hem*Group	.013	.910	<.001	.068	.765	.001	-	-	-
Electrode	6.371	.001**	.106	28.362	<.001**	.344	2.473	.068	.045
Electrode*Group	.212	.860	.004	1.150	.328	.021	.519	.659	.010
Picture*Hem	4.938	.030**	.084	.136	.714	.003	-	-	-
Picture*Hem *Group	.292	.591	.005	.177	.676	.003	-	-	-
Picture*Electrode	5.374	.004**	.091	2.796	.067	.049	1.985	.120	.037
Picture*Electrode* Group	1.233	.298	.022	.776	.460	.014	.171	.913	.003
Hem*Electrode	2.007	.132	.036	1.799	.161	.032	-	-	-
Hem*Electrode *Group	.760	.487	.014	1.106	.342	.020	-	-	-
Picture*Hem *Electrode	.255	.844	.005	.385	.707	.007	-	-	-
Picture*Hem *Electrode*Group	.743	.519	.014	.248	.808	.005	-	-	-
Group	7.549	.008**	.123	5.115	.028**	.087	.968	.330	.018

Note. **significant at .05 level.

Table S5.

Mixed factorial ANOVA results assessing for gender-sensitivity in P1, N1 and VPP amplitudes (μV). Group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses – see method section)

	P1			N1			VPP		
	F-value	*p*-value	η_p^2	*F* -value	*p*-value	η_p^2	*F*-value	*p*-value	η_p^2
Picture type	2.632	.111	.046	25.631	<.001**	.322	6.549	.013**	.110
Picture*Group	.346	.559	.006	7.081	.010**	.116	4.596	.037**	.080
Hemisphere	28.528	<.001**	.346	1.139	.291	.021	-	-	-
Hem*Group	1.104	.298	.020	.025	.876	<.001	-	-	-
Electrode	40.824	<.001**	.431	42.704	<.001**	.442	1.852	.165	.034
Electrode*Group	.263	.771	.005	.280	.792	.005	.821	.434	.015
Picture*Hem	.024	.876	<.001	4.679	.035**	.080	-	-	-
Picture*Hem *Group	.227	.636	.004	.012	.914	<.001	-	-	-
Picture*Electrode	.386	.714	.007	5.208	.006**	.088	.592	.581	.011
Picture*Electrode* Group	.668	.538	.012	.154	.857	.003	.717	.511	.013
Hem*Electrode	10.904	<.001**	.168	5.174	.007**	.087	-	-	-
Hem*Electrode *Group	.658	.510	.012	.154	.857	.003	-	-	-
Picture*Hem *Electrode	1.652	.187	.030	.225	.839	.004	-	-	-
Picture*Hem *Electrode*Group	.627	.576	.011	1.950	.137	.035	-	-	-
Group	1.847	.180	.033	.050	.824	.001	.262	.611	.005

Note. **significant at .05 level.

Table S6.

Mixed factorial ANOVA results assessing for gender-sensitivity in P1, N1 and VPP latency (ms). Group as the between-subjects factor (control vs. ED) and picture type (house vs. body), hemisphere (left vs. right, for P1/N1 analyses only) and electrode (4 electrodes for P1/N1 analyses or 5 electrodes for VPP analyses – see method section)

	P1			N1			VPP		
	F-value	*p*-value	η_p^2	*F* -value	*p*-value	η_p^2	*F*-value	*p*-value	η_p^2
Picture type	16.732	<.001**	.237	2.222	.142	.040	1.124	.294	.021
Picture*Group	.585	.448	.011	1.088	.302	.020	.686	.411	.013
Hemisphere	.005	.943	<.001	.199	.657	.004	-	-	-
Hem*Group	.680	.413	.012	.554	.460	.010	-	-	-
Electrode	5.620	.003**	.094	26.032	<.001**	.325	1.110	.344	.021
Electrode*Group	.767	.483	.014	1.514	.220	.027	2.017	.120	.037
Picture*Hem	.670	.417	.012	.156	.695	.003	-	-	-
Picture*Hem *Group	.058	.811	.001	.065	.800	.001	-	-	-
Picture*Electrode	.490	.643	.009	1.006	.377	.018	.039	.983	.001
Picture*Electrode* Group	1.671	.187	.030	1.136	.329	.021	1.907	.140	.035
Hem*Electrode	4.487	.007**	.077	3.877	.020**	.067	-	-	-
Hem*Electrode *Group	.289	.802	.005	.873	.429	.016	-	-	-
Picture*Hem *Electrode	3.548	.018**	.062	.242	.803	.004	-	-	-
Picture*Hem *Electrode*Group	.700	.546	.013	1.407	.249	.025	-	-	-
Group	10.023	.003**	.157	8.330	.006**	.134	1.106	.318	.019

Note. **significant at .05 level.

Table S7.

Evidence for gender-selective ERP effects over N1 and VPP amplitudes (μV) in both anorexic and

bulimic participants.

	Male Bodies		Female Bodies		Follow-up pairwise comparison results	
	N1	VPP	N1	VPP	N1	VPP
Anorexic participants (N=15)	.163 (1.01)	.562 (1.05)	-1.642 (.91)	1.886 (1.02)	$t(14) = 4.090$, $p < .001$**	$t(14) = 2.622$, $p = .014$**
Bulimic participants (N=14)	-1.977 (1.04)	1.381 (.97)	-3.357 (.96)	2.403 (.95)	$t(13) = 3.026$, $p = .005$**	$t(13) = 2.174$, $p = .039$**

Note. Standard deviation in parentheses and amplitudes given in μV. **significant at .05 level.

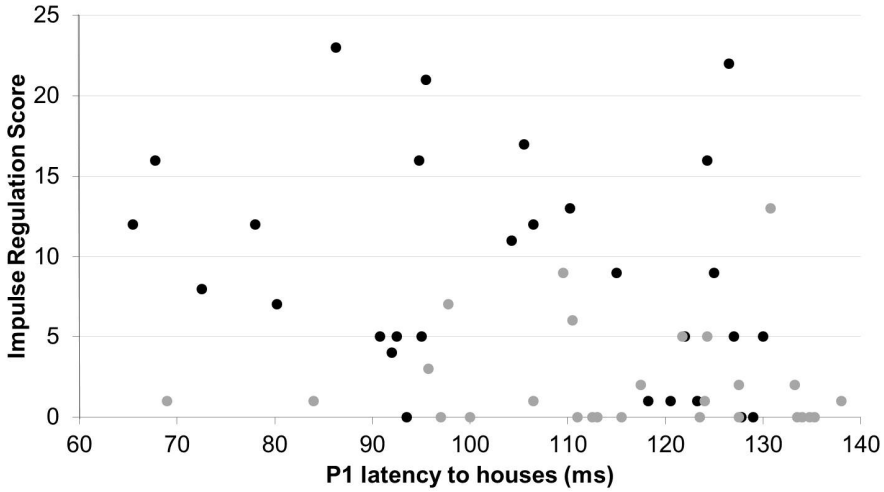

Figure S1. Moderate, negative relationship between P1 latency to house stimuli and impulse regulation score, $r(54) = -.327$, $p = .014$.

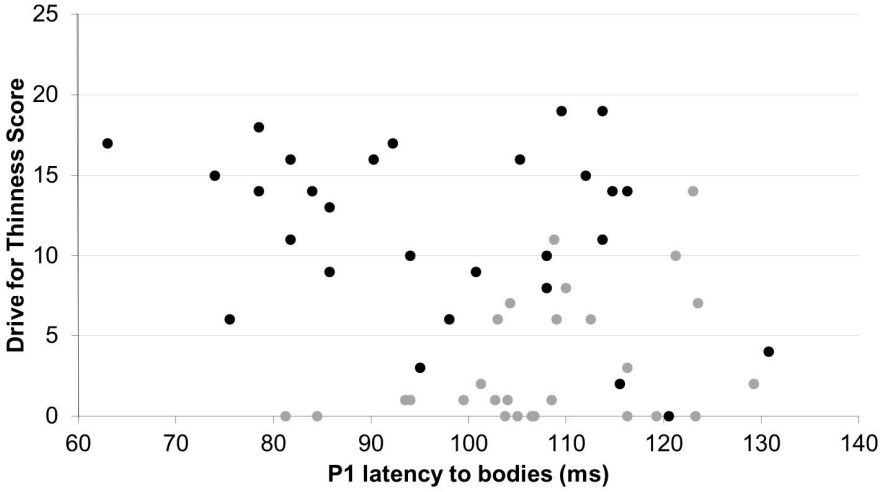

Figure S2. Moderate, negative relationship between P1 latency to body stimuli and drive for thinness score, $r(54) = -.356$, $p = .007$.

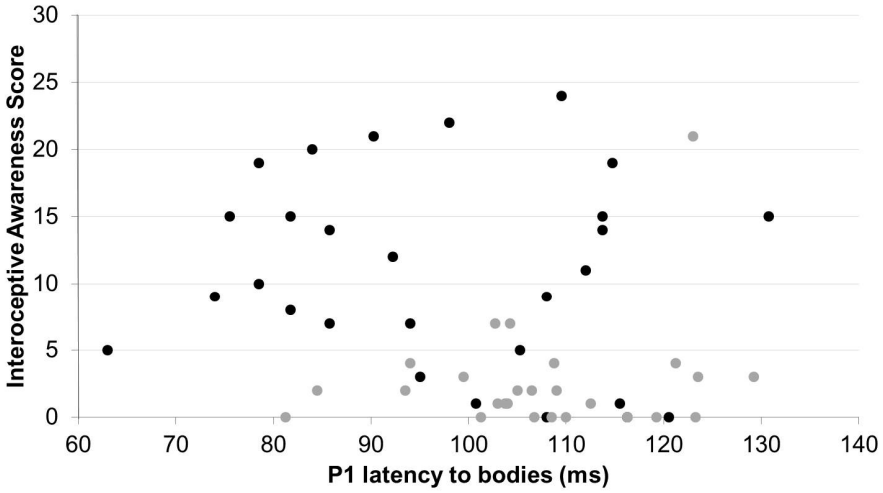

Figure S3. Moderate, negative relationship between P1 latency to body stimuli and interoceptive awareness score, $r(54) = -.312$, $p = .019$.

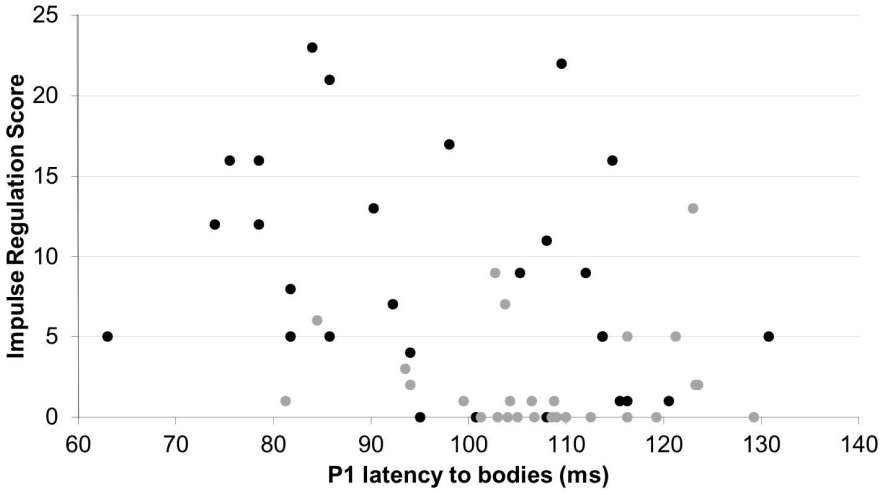

Figure S4. Moderate, negative relationship between P1 latency to body stimuli and impulse regulation score, $r(54) = -.384$, $p = .003$.

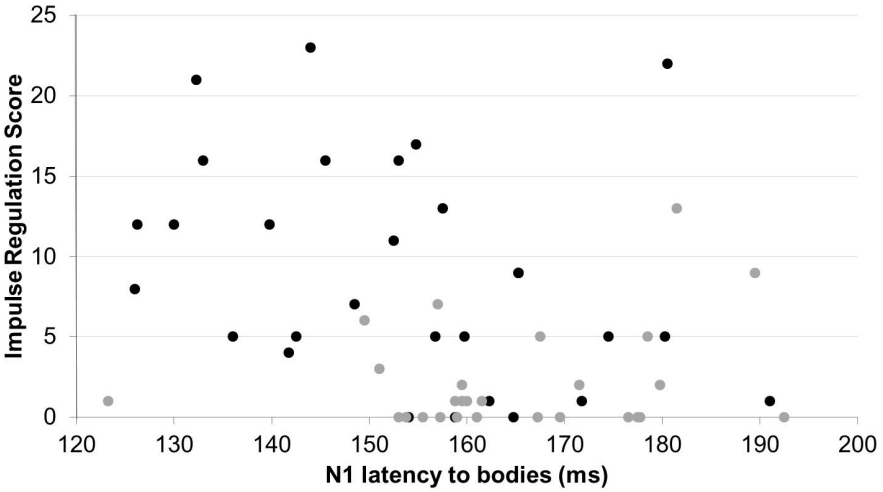

Figure S5. Moderate, negative relationship between N1 latency to body stimuli and impulse regulation score, $r(54) = -.319$, $p = .016$.

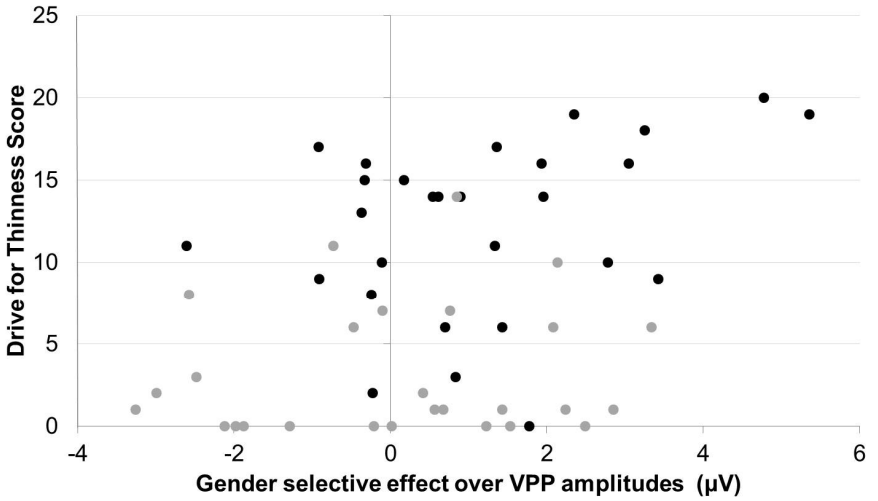

Figure S6. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and drive for thinness score, $r(53) = .371$, $p = .005$.

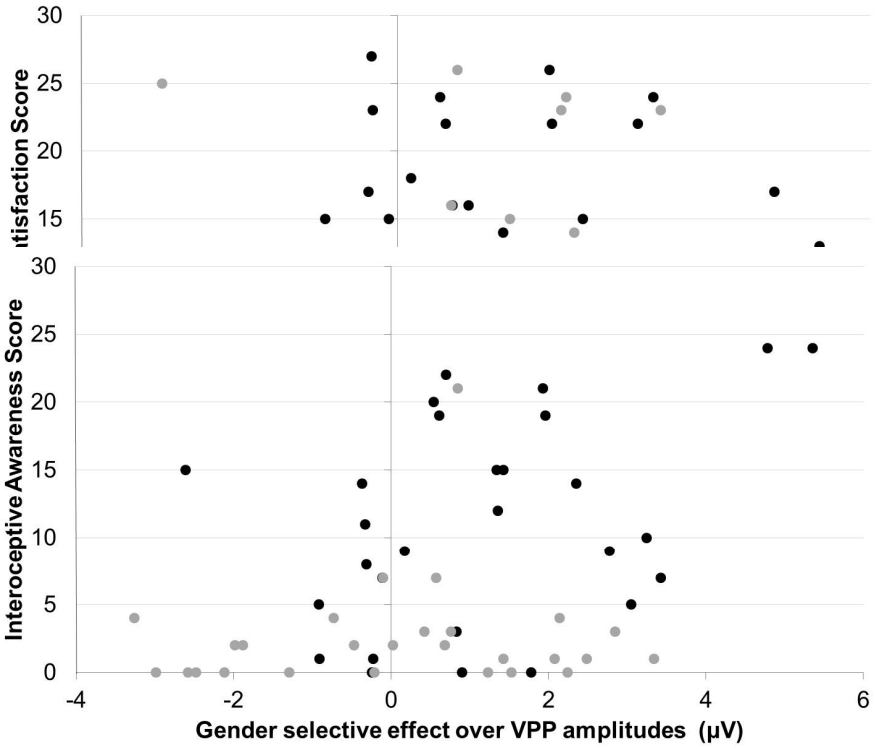

Figure S10. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and interoceptive awareness score, $r(53) = .375$, $p = .005$.

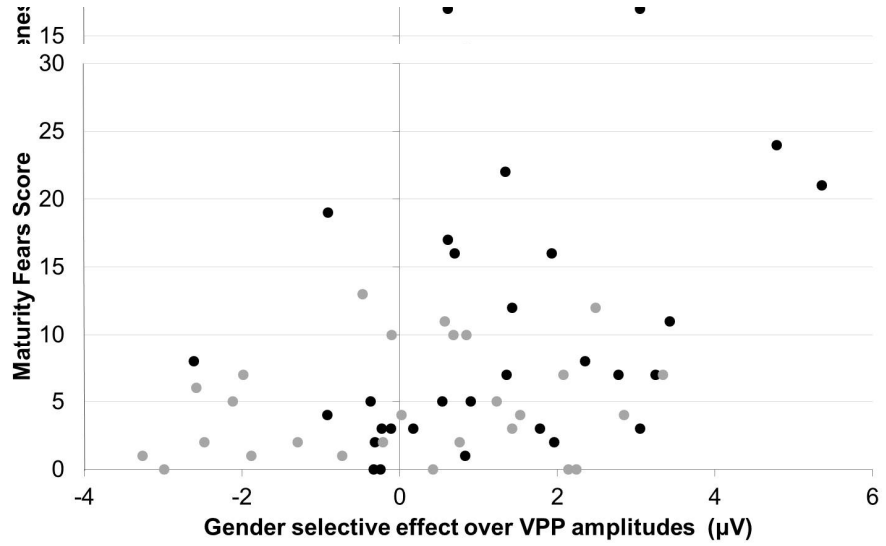

Figure S11. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and maturity fears score, $r(53) = .409$, $p = .002$.

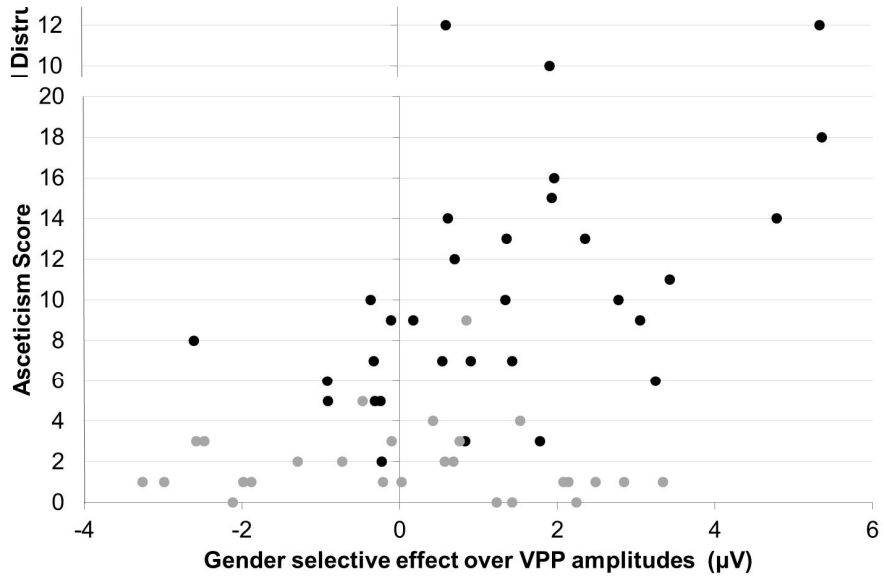

Figure S12. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and asceticism score, $r(53) = .435$, $p = .001$.

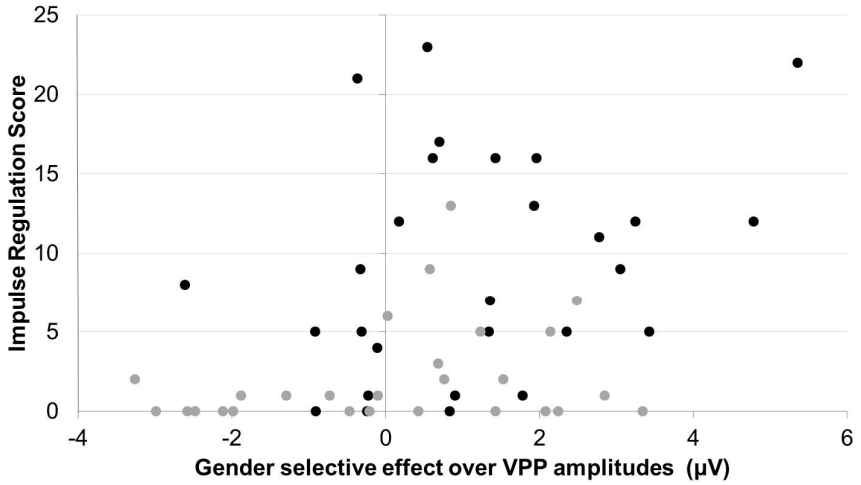

Figure S13. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and impulse regulation score, $r(53) = .373$, $p = .005$.

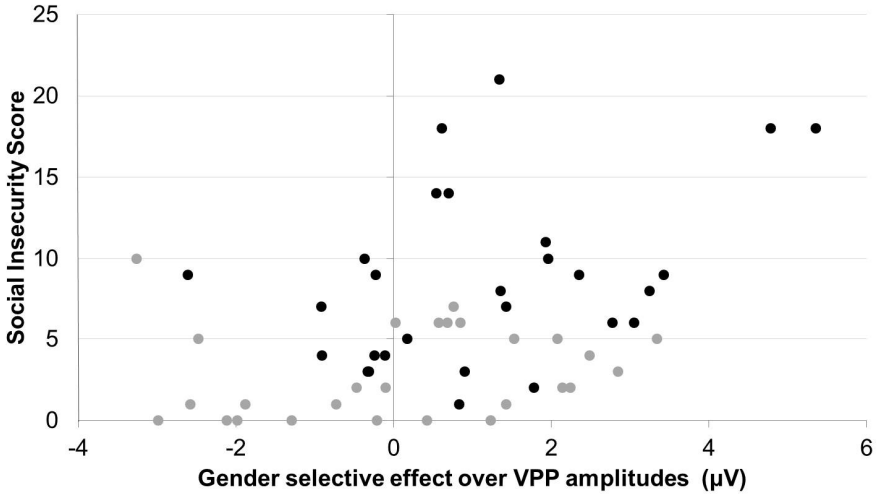

Figure S14. Moderate, positive relationship between the gender-selective effect over VPP amplitudes and social insecurity score, $r(53) = .392$, $p = .003$.