

## Research Article

# Healthy Restrained Eaters Diminish Consummatory Food Reward and Inhibit Prepotent Feeding Responses: An EEG Study

David John Hume

Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Newlands, South Africa

Fleur Margaret Howells

Department of Psychiatry and Mental Health, Faculty of Health Sciences, University of Cape Town, Observatory, South Africa

HG Laurie Rauch

Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Newlands, South Africa

Jacolene Kroff

Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Newlands, South Africa

Estelle Victoria Lambert

Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Newlands, South Africa

## ABSTRACT

**Background:** Some individuals with modest elevations in dietary restraint exhibit the ability to diminish consummatory and anticipatory food reward. In this paper we aim to identify the underlying mechanisms of food-specific cortical inhibition responses in restrained vs unrestrained eaters.

**Methods:** Restrained ( $n = 43$ ) and unrestrained eaters ( $n = 38$ ) completed self-report surveys and a food- vs non-food Stroop task protocol with record of electroencephalography (EEG). Cortical rhythms and EEG ERPs were assessed.

**Results:** Compared to their unrestrained peers, restrained eaters showed several differences in food task-related event-related potential (ERP) waveform presentation: ERP P300 component latency was significantly longer during food image viewing over the left parietal cortex ( $P_3$ ), and the amplitudes of the ERP P300 and LPP components were significantly greater over the right central electrode ( $C_4$ ) whilst responding to Stroop

color-word cues. Food task-related conflict cue elicited ERP P300 amplitude correlated positively with eating restraint, and negatively with bioelectrical impedance assessed % body fat.

**Conclusions:** Restrained eaters free of eating pathology attenuate conscious visual food cue processing and show enhanced executive brain functioning during late attentional processing despite the presence of distractor food cues. Our data propose robust executive governance as the primary underlying neurophysiological mechanism by which healthy restrained eaters diminish consummatory food reward and inhibit prepotent feeding responses.

**MeSH Headings/Keywords:** Eating restraint; Food cue-reactivity; Attentional processing; Modified Stroop Protocol Executive function; Electroencephalography (EEG); Event-related potentials (ERPs)

## Introduction

Commercialized societies are characterized by environments and marketing styles which promote food consumption in ever-greater quantities [1]. Opposing these environmental threats, modest elevations in eating restraint – or the deliberate cognitive exercise of limiting food consumption – is commonly observed in successful dieters [2-4]. The underlying neurophysiological mechanisms supporting this internal self-regulation of eating style, however, remain unclear.

Research consistently shows us that weight loss maintenance associates with greater dietary vigilance [5,6], whereas individuals

who experience weight recidivism typically show marked reductions in eating restraint over time [7]. Adding to this idea, prior reports illustrate that successful dieters exhibit elevated Three Factor Eating Questionnaire (TFEQ) dietary restraint scores comparable to patients exiting a weight loss intervention programme (mean = 7.1) [8], although not nearly as pronounced as records seen in patients with clinical eating disorders (mean = 13.3) [9]. Neuroimaging studies furthermore show that regions of the brain involved in visual attention and behavioral inhibition - such as the visual and dorsal prefrontal cortices - are particularly activated in successful dieters compared to normal weight non-dieters following meal consumption [10]. These

findings are supported by empirical accounts of blunted skin conductance orienting responses to food odours [11], decreased heart rate, gastric activity and salivation upon orosensory food cue exposure [12], and diminished electroencephalography (EEG) measured brain activity upon food cue exposure in restrained versus unrestrained dieters [13,14]. Together, these reports propose that restrained eaters, much like those who maintain substantial weight losses [15], willfully diminish their sensitivity to food cues as to forestall externally-modulated food consumption.

EEG event-related potential (ERP) waveform components reflect neurotransmitter activity modulating various sensory, affective and motivational processes and, thus, are particularly useful for evaluating time-locked changes in cortical processing related to poignant stimuli. For instance, the amplitude of the P200 component associates with preconscious attentional processing [16], whereas the amplitude of the P300 component relates to maintained conscious attention and cortical updating [17]. While visual food cues have been shown to generate pronounced ERP P200 and P300 component amplitudes in individuals presenting with obesity [18], how these components are implicated in eating styles which may protect against weight gain remains unclear. Moreover, the late positive potential (LPP) ERP waveform – representative of explicit memory recall and motivated behavior – remains unstudied in the context of dietary vigilance [19]. There is, in addition, a notable absence of work exploring whether EEG ERP component latency presents differently among persons with varying degrees of intrinsic dietary control.

Finally, while there is a paucity of work on restrained eating and cognition, a substantial body of evidence suggests that eating styles associated with overweight and obesity relate to poor cognitive governance [20]. Executive function – the most frequently observed cognitive deficit in individuals who present for weight loss – includes various high-level cortical processes which facilitate decision making, inhibition and complex motivation-related behaviors [20,21]. Insofar as restrained eaters demonstrate the ability to reduce their external drive to eat without any negative effects on mental or physical well-being [7], the current literature supports further inquiry into whether neurobehavioral control, specific to food cues, associates with healthy dietary vigilance.

In the present study we test the thesis that healthy individuals with modest elevations in eating restraint are able to down-regulate external food cue processing, as indicated by smaller and/or more delayed EEG ERP component peak amplitudes sourced during passive food image viewing. We furthermore hypothesize that – despite the presence of distractor food stimuli – restrained eaters will be more attentive to cues which prompt behavioral responses contingent on executive brain processes.

## Methods

### Study sample

The sample comprised eighty-one healthy female adults (mean BMI = 26.3 kg/m<sup>2</sup>; mean age = 31.7 years), recruited from the community for a study on electrophysiological responses during food cue processing in persons of normal weight, overweight and obese BMI indices [22]. Participants were free

of clinically diagnosed conditions (both mental and physical) at the time of data collection and for the three-month period prior to study commencement. Additional exclusion criteria included pregnancy, a history of uncontrollable or disinhibited overeating, or current anorexia nervosa, bulimia nervosa, or binge eating disorder (per DSM-IV diagnostic criteria). As is most widely implemented in the literature [23], participants were classified as either “restrained eaters” or “unrestrained eaters” via a median split on TFEQ assessed eating restraint.

### Experimental procedure

Participants were asked to desist from food/beverage consumption for a minimum of two hours prior to EEG record. Pre-trial instructions discouraged participants from arriving at their appointments having refrained from food intake for longer than four hours as elevations in hunger might have jeopardized the validity of our outcomes. Participants were, however, permitted to consume unflavored/uncarbonated water freely at all times. As a final precautionary measure of controlling for differential levels of hunger at baseline, participants were asked to subjectively rate their momentary appetitive drive using a standard visual analogue scale (VAS). The VAS consists of five subscales (VAS 1: hunger; VAS 2: fullness; VAS 3: desire to eat; VAS 4: prospective consumption; VAS 5: satiety) which have been shown to be both valid and reliable for the assessment of subjective accounts of appetite and food craving [24].

Trials began with the assessment of basic anthropometric characteristics which included height, weight, waist and hip circumferences, and % body fat. Participants were then instructed to sit calmly while completing three standard computerized attention tasks with EEG record. A familiarization task was completed initially, followed by completion of a food-related task and an office-related task at random. All trials were conducted in a light and sound-attenuated room, and took place between 12h00-16h00.

### Anthropometry

Weight (BW-150, NAGATA, Tainan, Taiwan) and height (3PHTROD-WM, Detecto, Missouri, USA) were recorded for all participants, both measures obtained while unshod and in lightweight garments. Waist circumference (the narrowest girth amid the xiphoid and the umbilicus) and hip circumference (the widest girth measured at the mid-gluteal region with the medial malleoli of the ankles placed together) were determined with a standard tape measure. Body fat (%) was assessed via Bioelectrical Impedance Analysis (BIA) (Quantum II, RJL Systems, Michigan, USA) using the Sun et al. algorithm [25].

### Self-report questionnaires

**Screening and sample attribute questionnaires:** A battery of self-developed surveys enquired about participants’ socio-demographic profile, and was used to confirm that study groups were homogenous in this regard. Medical health and reproductive history questionnaires were administered to exclude women with any chronic physical or mental health concerns, those on chronic prescription medications, and those indicating that they are pregnant, lactating, or have reached natural menopause.

**Three-Factor Eating Questionnaire (TFEQ):** The TFEQ consists of fifty-one items, and is designed to gauge three

independent domains of human eating behavior: 1) eating restraint, 2) disinhibition and uncontrollable eating, and 3) trait-related hunger [26]. Each domain is scored independently, with each item yielding between 0 and 3 points. Higher scores for each TFEQ sub-component are indicative of a greater extent of each respective eating behavior.

### Modified Stroop tasks

Modified versions of the Stroop task (one task containing food images, the other containing neutral/office images) were run with record of EEG, details of which have been described at length elsewhere [22]. Briefly, tasks comprised twenty images embedded at random amid standard Stroop color-word prompts [27]. Stroop prompts yielded data on the accuracy and reaction times of participants' responses to cognitive cues, and EEG frequency and ERP data supplied electrophysiological data specific to cognitive (color-word) processing as well as to passive food-related and food-unrelated image viewing. Last, the paradigm included the assessment of working memory as participants were instructed to count the number of images (i.e. blank squares in the practice task, food images in the food task and office images in the neutral task) which were presented during each five-minute task as this would be recalled upon completion of each trial. For Stroop cues, mean reaction time was calculated for correct responses only, and outliers (i.e. mean accuracy and reaction times exceeding the population mean by  $\pm 2$  standard deviations) were discarded from analyses.

### Electroencephalography (EEG)

A bioamplifier system (MP150 system, Biopac Systems Inc.) with 10 EEG100C amplifiers and 1 electrooculogram (EOG) module was used for record of EEG data via a standard PC running Acqknowledge 4.1 software. Linked ear referencing was used to obtain data from the following scalp sites:  $F_{p1}$ ,  $F_{p2}$ ,  $F_3$ ,  $F_4$ ,  $F_7$ ,  $F_8$ ,  $C_3$ ,  $C_4$ ,  $P_3$  and  $P_4$  (positioning via universal 10/20 montage system, sampling rate = 500 Hz). Participants were grounded peripherally and eye movements were detected by 2 EOG electrodes so that raw EEG data could be corrected for EOG artefact using automated Independent Component Analyses (ICA).

Cortical rhythms and ERPs were extracted using an in-house designed graphical user interface (GUI) (Matlab, Mathworks, MA, USA). Data were band pass filtered (FIR) with a Hamming window of 0.1 - 30 Hz. The filtered EEG data of the food and office tasks were Fourier transformed extracting delta ( $\delta$ , 0.1-4 Hz), theta ( $\theta$ , 4-7 Hz), alpha ( $\alpha$ , 7-14 Hz), and beta ( $\beta$ , 15-30 Hz) frequency bands. Absolute power was converted to relative power (%). Epochs for ERPs were set at 200ms prior to and 600ms post cue presentation (i.e. yielding an 800ms window).

Averaged waveforms were reproduced to classify ERP components. For each ERP component, amplitude ( $\mu V$ ) and latency (ms) values were obtained. ERPs were baseline corrected by the 200ms window prior to cue presentation, and were rejected when exceeding  $\pm 100\mu V$ . Components extracted around image exposure included an early positive P150 (100-200ms window, central electrodes ( $C_3$ ,  $C_4$ )) and an early negative N150 component (100-200 ms window, parietal electrodes ( $P_3$ ,  $P_4$ )). During image exposure, P200-like (200-300ms window, central and parietal electrodes ( $C_3$ ,  $C_4$ ,  $P_3$ ,  $P_4$ )),

P300-like (300-400ms window in central electrodes ( $C_3$ ,  $C_4$ ), 200-550ms window in parietal electrodes ( $P_3$ ,  $P_4$ )), and LPP (450-550ms, central and parietal electrodes ( $C_3$ ,  $C_4$ ,  $P_3$ ,  $P_4$ )) wave components were extracted. Importantly, each participant must have provided  $\geq 75\%$  valid ERPs (i.e. no less than 15/20 within rejection thresholds) for each task to be included in the analyses.

The STATA 12 software package (Stata, StataCorp, TX, USA) was used for the statistical analyses. Shapiro-Wilk  $W$  tests were conducted to examine data normality. Student's  $t$ -tests were performed to assess for differences in parametric data and Mann-Whitney  $U$  tests (indicated by  $z$ -scores) were performed to examine non-parametric data. The assumptions of statistical tests regarding the homogeneity of variances were tested with Bartlett's test for parametric data and Levene's test for non-parametric data. Data in text, tables and figures are described as means  $\pm$  standard deviation or medians and quartiles, as appropriate. Since EEG data were parametric, Pearson correlation coefficients were used to determine relationships between measures of eating behavior and body composition against significantly different relative band power and ERP measures recorded during the modified Stroop tasks by group. Analysis of covariance (ANCOVA) trends tests were used to test for the effect of interrelated and potentially confounding variables. Specifically, we controlled for body size (BMI), as well as for reported state- and trait-related hunger (as measured with the VAS questionnaire and TFEQ respectively). In the event of significant EEG findings, Bonferroni-corrected post hoc  $t$ -tests were conducted to control for possible inflations of the Type I error rate due to multiple comparison. The alpha level was set at  $< 0.05$ . Results are presented as a secondary analysis of data which appear elsewhere in the literature.<sup>22</sup>

## Results

### Physical characteristics

Eighty-one healthy women (mean BMI = 26.3kg/m<sup>2</sup>; mean age = 31.7 years) with no known illness or disorders and of similar socioeconomic and demographic status participated in the study. Forty-three participants were classified as restrained eaters (mean BMI = 26.3kg/m<sup>2</sup>; mean age = 31.7years) and thirty-eight as unrestrained eaters (mean BMI = 26.3kg/m<sup>2</sup>; mean age = 31.7years). Participants were comparable for all anthropometric measures (Table 1).

### Three Factor Eating Questionnaire (TFEQ)

Per study design, TFEQ measured eating restraint was significantly greater in restrained as compared to unrestrained eaters ( $t_{(79)} = -13.28$ ,  $p < 0.001$ ) (Table 1). While this survey revealed that groups were alike for measures of disinhibited eating, restrained eaters reported lower levels of habitual hunger than their unrestrained peers ( $z = 2.66$ ,  $p = 0.01$ ).

### Pre-experimental satiety

Subjective ratings of momentary satiety (as measured with the VAS) were different between groups prior to the performance of the modified Stroop tasks with record of EEG (VAS 1:  $z = 2.41$ ,  $p = 0.02$ ; VAS 2:  $t_{(79)} = -2.52$ ,  $p = 0.01$ ; VAS 3:  $z = 2.25$ ,  $p = 0.02$ ; VAS 4:  $z = 3.01$ ,  $p < 0.01$ ; VAS 5:  $t_{(79)} = -2.55$ ,  $p = 0.01$ ). Even so, the time interlude between testing and most recent

**Table 1:** Measures of clinical anthropometry and eating behavior for restrained and unrestrained eaters.

		Restrained eaters ( <i>n</i> = 43)		Unrestrained eaters ( <i>n</i> = 38)	
<b>Anthropometry</b>					
Age (yrs)		31.7	± 6.7	31.7	± 7.4
Height (cm)		166.9	± 6.6	165.1	± 5.4
Weight (kg)		73.3	± 17.2	72.0	± 22.2
BMI (kg/m <sup>2</sup> )		26.3	± 5.7	26.3	± 7.7
Lean body mass (kg)		48.5	± 5.7	46.8	± 7.3
Lean body mass (%)		67.8	± 8.3	67.6	± 10.8
Body fat mass (kg)		24.9	± 12.6	25.3	± 16.0
Body fat mass (%)		32.2	± 8.3	32.4	± 10.8
Waist circumference (cm)		75.5	± 13.2	75.6	± 14.3
Hip circumference (cm)		106.1	± 13.0	105.4	± 14.7
Waist/hip ratio		0.71	± 0.07	0.71	± 0.06
<b>Three Factor Eating Questionnaire</b>					
TFEQ-I (restraint factor)	***	12	± 3	5	± 2
TFEQ-II (disinhibition factor)		8	± 4	8	± 5
TFEQ-III (trait-related hunger factor)	**	5	± 3	7	± 4

Abbreviations: BMI, Body Mass Index; TFEQ, Three Factor Eating Questionnaire.  
Notes: \*\**p* < 0.03, \*\*\**p* < 0.01.

caloric or caffeinated food/beverage intake was alike (Table 2).

### Stroop task performance scores

Behavioral measures were comparable between groups. Error rates were diminutive (average mistakes < 3 per group during food and office tasks) thereby precluding erroneous EEG measures due to error-related negativity (ERN) (Table 2).

### EEG measures

**Relative EEG band power:** Indices of cortical arousal were similar between groups during both tasks (Figure 1).

**ERP amplitudes and latencies:** Left parietal (P<sub>3</sub>) P300 latency, as measured during food image viewing, presented differently between groups. More explicitly, the amplitude of this component peaked later upon cue exposure in restrained (441.8 ± 82.1 ms) as compared to unrestrained eaters (404.9 ± 83.3 ms) ( $t_{(76)} = -1.968, p = 0.042$ , Figure 2A). Restrained eaters furthermore showed significantly greater Stroop conflict (or color-word) cue induced ERP P300 ( $t_{(77)} = -2.363, p = 0.012$ ) and ERP LPP ( $t_{(77)} = -2.209, p = 0.013$ ) component amplitudes over the right central cortex (C<sub>4</sub>) during execution of the food task (ERP P300: restrained eaters = 4.51 ± 2.97 μV, unrestrained eaters = 3.15 ± 2.01 μV; ERP LPP: restrained eaters = 3.07 ± 3.65 μV, unrestrained eaters = 1.48 ± 2.75 μV; Figure 2B). Image- and conflict-related ERP component amplitude or latency scores recorded during neutral/office task performance were, however, comparable between groups.

**Analysis of covariance (ANCOVA) trends tests:** Inclusion criteria for adjustment covariates were based on a prior review of EEG ERP studies showing evidence for strong association between BMI and elevated food cue-reactivity [28], and upon the detection of significantly different indices of eating behavior in the preliminary between-group results of the present report (Tables 1 & 2). As shown in Table 3, all differential ERP responses identified between restrained and unrestrained eaters

remained statistically significant after controlling for BMI (kg/m<sup>2</sup>), TFEQ measured trait-related hunger, and VAS assessed momentary appetitive drive. Due to clear co-linearity between all VAS subscales ( $r > 0.7, p < 0.0001$ ) adjustment for merely one of these measures (VAS 5, or momentary satiety) was sufficient to statistically detect and account for potential spurious effect.

Adjusted means – as obtained via ANCOVA post-estimation analyses – indicated that restrained eating retained its association with abated visual food cue saliency (left parietal ERP P300 amplitude: restrained eaters = 444.42 ± 13.8 ms, unrestrained eaters = 402.02 ± 14.6 ms,  $p = 0.047$ ) and with greater cognitive processing of food task-related Stroop conflict cues (right central ERP P300 amplitude: restrained eaters = 4.52 ± 0.42 μV, unrestrained eaters = 3.13 ± 0.44 μV,  $p = 0.033$ ; ERP LPP amplitude: restrained eaters = 3.31 ± 0.53 μV, unrestrained eaters = 1.25 ± 0.56 μV,  $p = 0.012$ ).

**EEG correlational analyses:** Food task conflict-related ERP P300 amplitude correlated positively with eating restraint for restrained and unrestrained eaters (groups combined:  $r = 0.294, p = 0.011$ ; Figure 3A) and negatively with percent fat mass for unrestrained eaters only ( $r = -0.363, p = 0.029$ ; Figure 3B).

### Discussion

In the present study, we aimed to identify the underlying mechanisms of food-specific cortical inhibition responses in restrained vs unrestrained eaters. Our data show that food cue-reactivity is attenuated in restrained eaters during maintained, conscious processing (delayed left parietal ERP P300 latency), and that these individuals demonstrate greater conscious conflict-related attentional processing and memory recognition (increased right central ERP P300 and LPP amplitudes, respectively) than their unrestrained controls.

Illustrating our first finding, the restrained eaters who partook in our study showed a delay in food image-elicited ERP P300

**Table 2:** Pre-experimental satiety and Stroop task results for restrained and unrestrained eaters.

		Restrained eaters ( <i>n</i> = 43)		Unrestrained eaters ( <i>n</i> = 38)	
<b>Pre-experimental satiety</b>					
Last intake (h)		3.1 ± 1.9		3.6 ± 2.2	
VAS: hunger (%)	**	31.2 ± 24.6		45.1 ± 23.5	
VAS: fullness (%)	**	52.3 ± 26.8		37.9 ± 24.3	
VAS: desire to eat (%)	**	31.7 ± 25.5		45.0 ± 25.6	
VAS: prospective consumption (%)	***	23.7 ± 19.1		37.7 ± 21.4	
VAS: satiety (%)	**	62.7 ± 25.6		48.2 ± 25.6	
<b>Stroop task performance</b>					
Food task reaction time (ms)		722.4 ± 208.5		680.2 ± 179.4	
Office task reaction time (ms)		724.4 ± 219.6		676.3 ± 159.5	
Food task mistakes		1.4 ± 1.3		2.0 ± 1.9	
Office task mistakes		1.3 ± 1.2		1.5 ± 1.7	
Food task picture count		19.9 ± 1.5		19.3 ± 1.8	
Office task picture count		20.0 ± 1.8		20.2 ± 2.8	

Abbreviations: VAS, Visual Analogue Scale.

Notes: \*\**p* < 0.03, \*\*\**p* < 0.01.**Table 3:** Effects for food cue processing and executive function controlling for sources of confound.

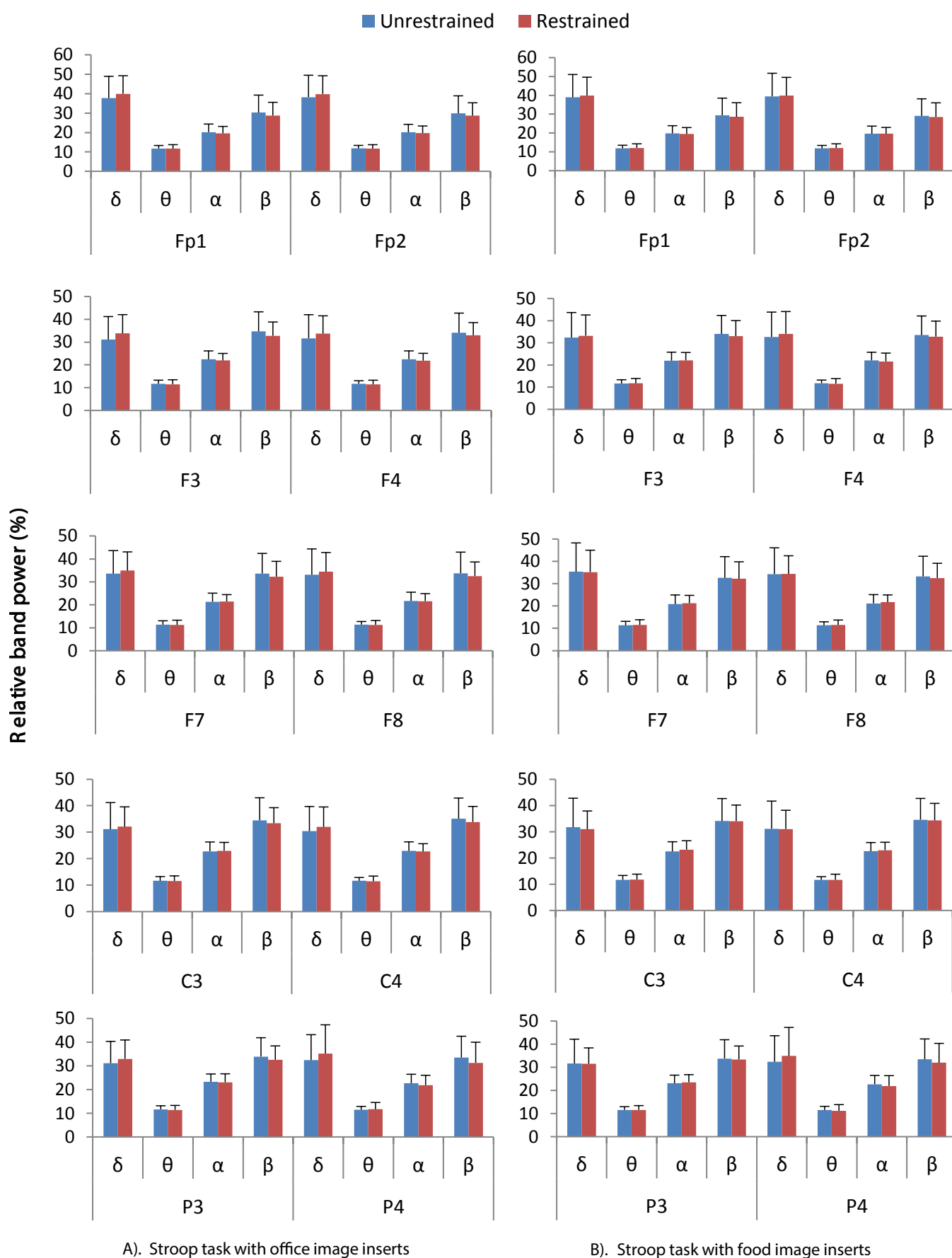
Outcome variable	Parameter	SS	df	Mean Square	F	Sig.
ERP P300 Latency (P <sub>3</sub> ) (Food images)	Model <sup>a</sup>	30143.9	4	7536.0	1.07	0.379
	BMI (kg/m <sup>2</sup> )	1277.0	1	1277.0	0.18	0.672
	TFEQ-III	3262.1	1	3262.1	0.46	0.499
	VAS 5	133.5	1	133.5	0.02	0.891
	Group	28703.4	1	28703.4	4.06	0.047*
ERP P300 Amplitude (C <sub>4</sub> ) (Food task Stroop cues)	Model <sup>b</sup>	43.9	4	11.0	1.64	0.173
	BMI (kg/m <sup>2</sup> )	0.0	1	0.0	0.00	0.952
	TFEQ-III	2.3	1	2.3	0.34	0.564
	VAS 5	5.6	1	5.6	0.84	0.363
	Group	31.3	1	31.3	4.68	0.033*
ERP LPP Amplitude (C <sub>4</sub> ) (Food task Stroop cues)	Model <sup>c</sup>	80.2	4	20.1	1.89	0.121
	BMI (kg/m <sup>2</sup> )	8.2	1	8.2	0.77	0.382
	TFEQ-III	2.8	1	2.8	0.27	0.607
	VAS 5	20.4	1	20.4	1.93	0.169
	Group	69.0	1	69.0	6.51	0.012**

Abbreviations: SS, Sum of Squares; BMI, Body Mass Index; TFEQ-III, Three Factor Eating Questionnaire trait-related hunger; VAS 5, Visual Analogue Subscale 5 (momentary satiety).

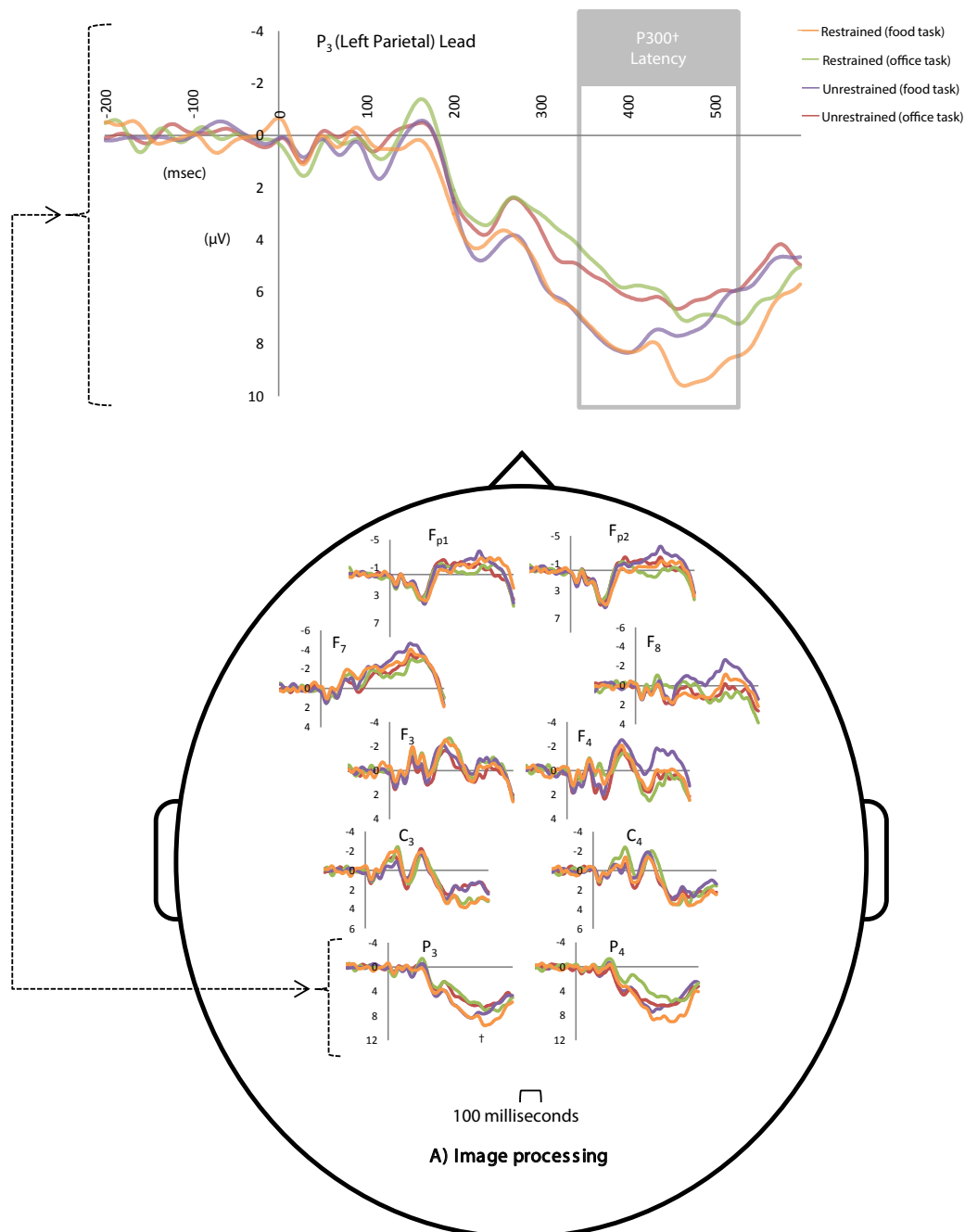
Notes: *N* = 81; <sup>a</sup>*R*<sup>2</sup> = 0.06 (adjusted *R*<sup>2</sup> = 0.004); <sup>b</sup>*R*<sup>2</sup> = 0.08 (adjusted *R*<sup>2</sup> = 0.032); <sup>c</sup>*R*<sup>2</sup> = 0.09 (adjusted *R*<sup>2</sup> = 0.044) \**p* < 0.05, \*\**p* < 0.03.

latency, indicative of deferred visual food cue-reactivity during maintained attentional processing in the posterior network. This finding corroborates the ideas of Nijs and colleagues (2009) who reported lower ERP P300 amplitudes over several parieto-occipital electrodes during food image viewing in females with lower external eating scores as measured via the Dutch Eating Behavior Questionnaire (DEBQ) [29]. DEBQ-identified low-external eating has furthermore been shown to correlate with blunted indices of attentional bias and lower degrees of approach behavior measured during a food image-adapted visual probe task [30].

Greater ERP P300 and ERP LPP amplitudes were evident in our restrained eaters over the right central cortex (C<sub>4</sub>) during color-word conflict resolution. Behavioral research has consistently shown us that modulations in the amplitude of the LPP waveform are directly attributable to the incentive value of affective stimuli [31,32]. As such, researchers propose the amplitudes of the ERP P300 and ERP LPP components as robust endophenotypic markers of the activation of neural circuitry specific to motivation, memory recognition and cortical updating [16,33]. In accord with our findings, these reports imply that restrained eaters demote visual food cue processing and utilize



**Figure 1:** Relative EEG band power for frontal ( $F_{p1}$ ,  $F_{p2}$ ,  $F_3$ ,  $F_4$ ,  $F_7$ ,  $F_8$ ), cingulate ( $C_3$ ,  $C_4$ ) and posterior electrodes ( $P_3$ ,  $P_4$ ) during office- and food-related Stroop task performance for restrained and unrestrained eaters. No group differences were found during (A) office task or (B) food task execution.



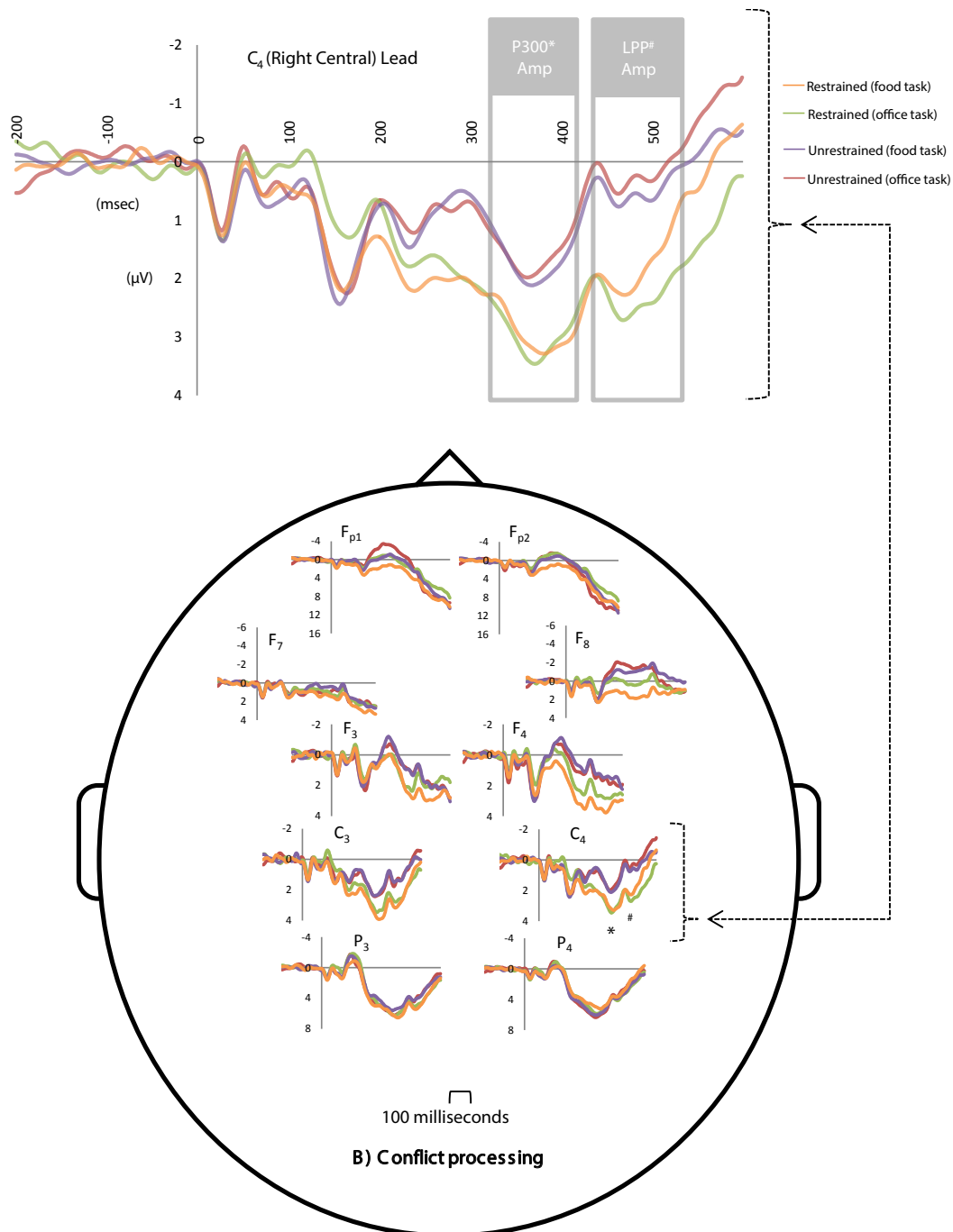
**Figure 2a:** Grand mean event-related potentials (ERPs) for frontal ( $F_{p1}$ ,  $F_{p2}$ ,  $F_3$ ,  $F_4$ ,  $F_7$ ,  $F_8$ ), central ( $C_3$ ,  $C_4$ ) and posterior electrodes ( $P_3$ ,  $P_4$ ) for image processing for restrained and unrestrained eaters. Group differences were found for food image processing for left parietal ( $P_3$ ) ERP P300 latency ( $\dagger$ unres. < res.,  $p = 0.042$ ). (B) Group differences were found for food task conflict processing for right central cortex ( $C_4$ ) ERP P300 amplitude ( $\ast$ unres. < res.,  $p = 0.012$ ) and ERP LPP amplitude ( $\#$ unres. < res.,  $p = 0.013$ ).

cognitive self-regulation more effectively to down-regulate the perceived attractiveness of highly salient external cues.

Numerous cross-sectional neuroimaging studies have unearthed sound evidence that overeating and prospective weight gain are related to executive dysfunction [34-36]. The results from one longitudinal study, in particular, suggest that cognitive deficits may be a cause rather than an effect of weight gain, concluding that lower gray matter volume in cognitive control structures (specifically, along the middle and superior frontal gyri) predicts weight gain at 1-year follow-up [37]. Other researchers have extended this notion by stressing that unusually high levels of activation in brain reward regions in

response to trigger cues (such as food) go hand in hand with an ever-declining capacity of the brain to exert self-control over prepotent or maladaptive behavioral responses [38]. However, additional studies are needed to determine causality between executive dysfunction and obesogenic eating behavior.

Lastly, electrocortical indices of cognitive performance during the food task correlated positively with self-reported eating restraint and negatively with total body adiposity. These findings support the notion that higher degrees of executive function coexist with heightened dietary control and lower overall body fatness. The finding of strong co-linearity between executive control and eating restraint provides further support



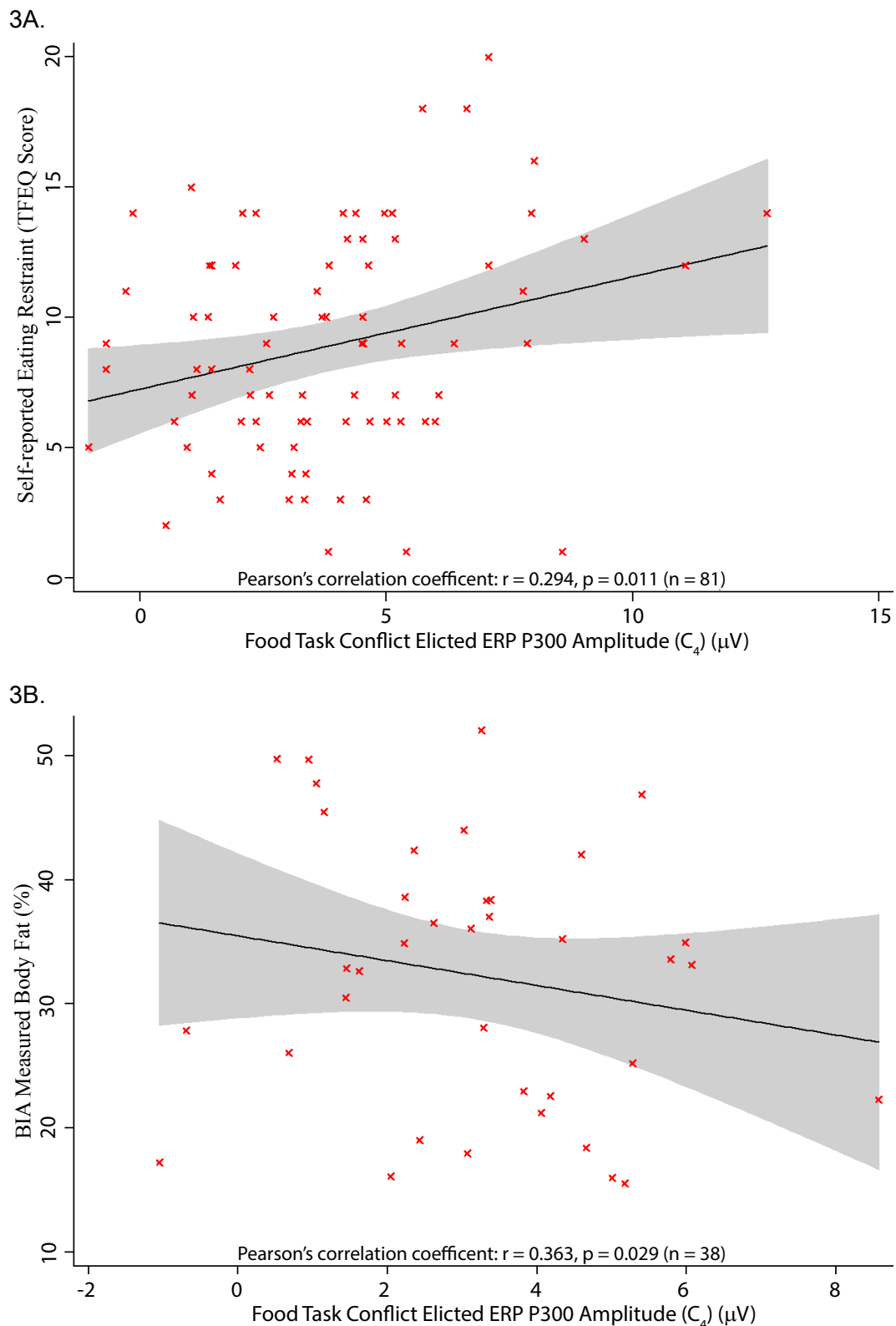
**Figure 2b:** Grand mean event-related potentials (ERPs) for frontal ( $F_{p1}$ ,  $F_{p2}$ ,  $F_3$ ,  $F_4$ ,  $F_7$ ,  $F_8$ ), central ( $C_3$ ,  $C_4$ ) and posterior electrodes ( $P_3$ ,  $P_4$ ) for Stroop color-word conflict cue processing for restrained and unrestrained eaters. Group differences were found for food task conflict cue processing over the right central cortex ( $C_4$ ) as shown by ERP P300 amplitude (\*unres. < res.,  $p = 0.012$ ) and ERP LPP amplitude (#unres. < res.,  $p = 0.013$ ) data. No differences were found for conflict cue processing measured during the office task.

for our proposed mechanism of top-down regulatory control over food valuation in restrained eaters, and is in agreement with work showing that a lack of cortical inhibition in external eaters associates with the severity of disinhibited, reward-driven food intake [39].

EEG offers several clinical advantages compared to alternative neuroimaging techniques. Perhaps the most noteworthy of these strengths is that EEG sourced ERP waveforms presently offer the highest temporal resolution for record of cortical activity [40]. Unlike fMRI and PET scanners which render individuals spatially confined, EEG

modalities allow participants to remain relatively mobile and free to execute cognitive tasks or activities while data are being collected [41]. Moreover, patterns in baseline cortical rhythms (as determined via EEG power spectral analyses) have been shown to predict future effort expenditure related specifically to reward attainment [42]. Hence, EEG could be used as a screening tool to identify those at risk for developing aberrant eating behaviors and, in so doing, allows for early intervention and preventative treatments to ensue. In consideration of these points, there is abundant room for the implementation of EEG in clinical settings, and for integration of this tool in routine medical examinations for risk stratification purposes.





**Figure 3:** Pearson correlations for maintained, conscious attentional processing of Stroop-specific color-word conflict cues with measures of eating behaviour and body composition. Significant associations were found for food task conflict cue elicited right central ( $C_4$ ) ERP P300 amplitude and: (3A.) Self-reported (TFEQ measured) eating restraint in restrained and unrestrained women (whole sample analysis); (3B.) Bioelectrical Impedance Analysis (BIA) assessed whole-body fatness (%) in unrestrained women.

The present study has at least three limitations. First, no standardized food/neutral picture resources were in existence when our study was designed. Implementation of the new “Food-pics” experimental research image catalogue would have allowed for greater methodological validity [43]. Second, our EEG system was

limited to 10 EEG electrodes, and a greater number of sensors would have allowed for detailed topographical mapping and analyses. Last, although our experimental design was piloted carefully to prevent an unfavorable signal-to-noise ratio, inclusion of additional image trials may have ensured further quality control.

In conclusion, this paper sought to assess electrocortical indices of visual food cue-reactivity and executive function in restrained versus unrestrained eaters free of clinical eating pathology. Our findings are in accord with the literature showing that restrained eaters attenuate food orienting responses, and add the novel contribution that these individuals display high levels of executive function during maintained attentional processing of visual food cues. Thus, our results suggest enhanced executive control as the underlying neurophysiological mechanism by which restrained eaters self-regulate eating behavior and mute the incentive value of external food cues.

### Role of funding sources

The financial assistance of the Medical Research Council (MRC) of South Africa and the South African National Research Foundation (NRF) towards this research is hereby acknowledged.

### Contributors

DJH conducted the research, analyzed the data, and is the primary author of this work. DJH, EVL and JK conceptualized the study design (2011). DJH and LR developed the Stroop and EEG testing protocols (2012). FMH, EVL, LR and JK supervised the study and provided input to the analyses and the working manuscript. All authors read and approved the final manuscript. This research paper formed part of DJH's PhD dissertation (completed Aug 2015).

### Conflict of Interest

We declare no conflicts of interest.

### Acknowledgements

The authors wish to thank David Karpul for the development of in-house EEG relative band power and ERP extraction software, and Louise Clamp for assisting with research participant recruitment.

### REFERENCES

1. Stanton RA. Food Retailers and Obesity. *Curr Obes Rep.* 2015; 4: 54-59.
2. Phelan S, Wing RR, Raynor HA, Dibello J. Holiday Weight Management by Successful Weight Losers and Normal Weight Individuals. *J Consult Clin Psychol.* 2008; 76: 442-448.
3. Herman CP, Mack D. Restrained and Unrestrained Eating. *J Pers.* 1975; 43: 647-660.
4. Herman CP, Polivy J. Anxiety, Restraint, and Eating Behavior. *J Abnorm Psychol.* 1975; 84: 666.
5. Vieira PN, Silva MN, Mata J, Coutinho SR, Santos TC, et al. Correlates of Health-related Quality of Life, Psychological Well-being, and Eating Self-regulation After Successful Weight Loss Maintenance. *J Behav Med.* 2013; 36: 601-610.
6. Hume DJ, Kroff J, Clamp LD, Lambert EV. Compensations for Weight Loss in Successful and Unsuccessful Dieters. *Am J Health Behav.* 2015; 39: 589-600.
7. Thomas JG, Bond DS, Phelan S, Hill JO, Wing, et al. Weight-loss Maintenance for 10 years in the National Weight Control Registry. *Am J Prev Med.* 2014; 46: 17-23.
8. Wing R, Hill J. Successful Weight Loss Maintenance. *Annu Rev Nutr.* 2001; 21: 323-341.
9. Huber J, Salatsch C, Ingenerf K, Schmid C, Maatouk I, et al. Characteristics of Disorder-Related Autobiographical Memory in Acute Anorexia Nervosa Patients. *Eur Eat Disord Rev.* 2015; 23: 379-389.
10. DelParigi A, Chen K, Salbe AD, Hill JO, Wing RR, et al. Successful Dieters have Increased Neural Activity in Cortical Areas Involved in the Control of Behavior. *Int J Obes.* 2007; 31: 440-448.
11. Piacentini A, Schell AM, Vanderweele DA. Restrained and Nonrestrained Eaters' Orienting Responses to Food and Nonfood Odors. *Physiol Behav.* 1993; 53: 133-138.
12. Nederkoorn C, Jansen A. Cue Reactivity and Regulation of Food Intake. *Eat Behav.* 2002; 3: 61-72.
13. Bleichert J, Feige B, Hajcak G, Tuschen-caffier B. To Eat or Not to Eat? Availability of Food Modulates the Electrocortical Response to Food Pictures in Restrained Eaters. *Appetite* 2010; 54: 262-268.
14. Hachl P, Hempel C, Pietrowsky R. ERPs to Stimulus Identification in Persons with Restrained Eating Behavior. *Int J Psychophysiol.* 2003; 49: 111-121.
15. Hume DJ, Howells FM, Karpul D, Rauch HL, Kroff J, et al. Cognitive Control Over Visual Food Cue Saliency is Greater in Reduced-overweight/Obese but not in Weight Relapsed Women: An EEG Study. *Eat Behav.* 2015; 19: 76-80.
16. Polich J, Kok A. Cognitive and Biological Determinants of P300: An Integrative Review. *Biol Psychol.* 1995; 41: 103-146.
17. Sokolov EN. Perception and the Conditioned Reflex. Pergamon Press, 1963.
18. Nijs IM, Franken IH, Muris P. Food-related Stroop Interference in Obese and Normal-weight Individuals: Behavioral and Electrophysiological Indices. *Eat Behav.* 2010; 11: 258-265.
19. Matsuda I, Nittono H. Motivational Significance and Cognitive Effort Elicit Different Late Positive Potentials. *Clin Neurophysiol.* 2015; 126: 304-313.
20. Smith E, Hay P, Campbell L, Trollor JN. A Review of the Association between Obesity and Cognitive Function Across the Lifespan: Implications for Novel Approaches to Prevention and Treatment. *Obesity Reviews.* 2011; 12: 740-755.
21. Stuss DT, Benson DF. The Frontal Lobes. Raven Press, 1996.
22. Hume DJ, Howells FM, Rauch HL, Kroff J, Lambert EV. Electrophysiological Indices of Visual Food Cue-Reactivity. Differences in Obese, Overweight and Normal Weight Women. *Appetite.* 2015; 85: 126-137.
23. Polivy J, Herman CP, Howard KI. In Dict Behav Assess Tech. Pergamon Press. 1988; 377-379.
24. Yoshioka M, St-Pierre S, Drapeau V, Dionne I, Doucet E, et al. Effects of Red Pepper on Appetite and Energy Intake. *Br J Nutr.* 1999; 82: 115-123.
25. Sun SS, Chumlea WC, Heymsfield SB, Lukaski HC, Schoeller D, et al. Development of Bioelectrical Impedance

- Analysis Prediction Equations for Body Composition with the use of a Multicomponent Model for use in Epidemiologic Surveys. *Am J Clin Nutr.* 2003; 77: 331-340.
26. Stunkard AJ, Messick S. The Three-factor Eating Questionnaire to Measure Dietary Restraint, Disinhibition and Hunger. *J Psychosom Res.* 1985; 29: 71-83.
27. Stroop JR. Studies of Interference in Serial Verbal Reactions. *J Exp Psychol.* 1935; 18: 643.
28. Nijs IM, Franken IH. Attentional Processing of Food Cues in Overweight and Obese Individuals. *Curr Obes Rep.* 2012; 1: 106-113.
29. Nijs IM, Franken IH, Muris P. Enhanced Processing of Food-related Pictures in Female External Eaters. *Appetite.* 2009; 53: 376-383.
30. Brignell C, Griffiths T, Bradley BP, Mogg K. Attentional and Approach Biases for Pictorial Food Cues. Influence of External Eating. *Appetite.* 2009; 52: 299-306.
31. Münte TF, Urbach TP, Düzél E, Kutas M, Boller F, et al. Event-related Brain Potentials in the Study of Human Cognition and Neuropsychology. *Handbook of neuropsychology.* 2000; 1: 139-236.
32. Stockburger J, Schmälzle R, Flaisch T, Bublatzky F, Schupp HT. The Impact of Hunger on Food Cue Processing: An Event-related Brain Potential Study. *Neuroimage.* 2009; 47: 1819-1829.
33. Cuthbert BN, Schupp HT, Bradley MM, Birbaumer N, Lang PJ. Brain Potentials in Affective Picture Processing: Covariation with Autonomic Arousal and Affective Report. *Biol Psychol.* 2000; 52: 95-111.
34. Volkow ND, Wang GJ, Telang F, Fowler JS, Goldstein RZ, et al. Inverse Association Between BMI and Prefrontal Metabolic Activity in Healthy Adults. *Obesity.* 2009; 17: 60-65.
35. Walther K, Birdsill AC, Glisky EL, Ryan L. Structural Brain Differences and Cognitive Functioning Related to Body Mass Index in Older Females. *Hum Brain Mapp.* 2010; 31: 1052-1064.
36. Willeumier KC, Taylor DV, Amen DG. Elevated BMI is Associated with Decreased Blood Flow in the Prefrontal Cortex using SPECT Imaging in Healthy Adults. *Obesity.* 2011; 19: 1095-1097.
37. Yokum S, Ng J, Stice E. Relation of Regional Gray and White Matter Volumes to Current BMI and Future Increases in BMI: A Prospective MRI Study. *Int J Obes.* 2012; 36: 656-664.
38. Volkow ND, Wang GJ, Tomasi D, Baler RD. Obesity and Addiction: Neurobiological Overlaps. *Obes Rev.* 2013; 14: 2-18.
39. Svaldi J, Naumann E, Trentowska M, Schmitz F. General and Food-specific Inhibitory Deficits in Binge Eating Disorder. *Int J Eat Disord.* 2014; 47: 534-542.
40. Luck S. An Introduction to the Event-related Potential Technique. MIT press, 2005.
41. Murphy KJ, Brunberg JA. Adult Claustrophobia, Anxiety and Sedation in MRI. *Magn Reson Imaging.* 1997; 15: 51-54.
42. Hughes DM, Yates MJ, Morton EE, Smillie LD. Asymmetric Frontal Cortical Activity Predicts Effort Expenditure for Reward. *Soc Cogn Affect Neurosci.* 2015; 10: 1015-1019.
43. Blechert J, Meule A, Busch NA, Ohla K. Food-pics: An Image Database for Experimental Research on Eating and Appetite. *Front Psychol.* 2015; 26: 40.

#### ADDRESS FOR CORRESPONDENCE

David John Hume, Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Newlands, South Africa, E-mail: davidjohnhume@gmail.com

Submitted: Apr 01, 2016

Accepted: Apr 25, 2016