Hunger and disinhibition but not cognitive restraint are associated with central norepinephrine transporter availability

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ARTICLE INFO

Article history:
Received 8 December 2016
Received in revised form 23 May 2017
Accepted 20 June 2017
Available online 21 June 2017

Keywords:
Norepinephrine transporter
Obesity
Eating behaviour
Three-Factor Eating Questionnaire
Position emission tomography
C-11-MRB

ABSTRACT

The relationship between food-intake related behaviours measured by the Three-Factor Eating Questionnaire (TFEQ) and in vivo norepinephrine transporter (NET) availability has not been explored yet. We investigated ten obese individuals (body mass index (BMI) 42.4 ± 3.7 kg/m²) and ten normal-weight healthy controls (HC, BMI 23.9 ± 2.5 kg/m²) with (S,S)-[11C]-O-methylreboxetine ([11C]MRB) positron emission tomography (PET). All participants completed the TFEQ, which measures cognitive restraint, disinhibition and hunger. Image analysis required magnetic resonance imaging data sets onto which volumes-of-interests were drawn. Tissue time activity curves (TACs) were obtained from the dynamic PET data followed by kinetic modeling of these regional brain TACs applying the multilinear reference tissue model (2 parameters) with the occipital cortex as reference region.

Obese individuals scored significantly higher on the hunger subscale of the TFEQ. Correlative data analysis showed that a higher degree of hunger correlated negatively with the NET availability of the insular cortex in both obese individuals and HC; however, this finding was more pronounced in obesity. Further, for obese individuals, a negative correlation between disinhibition and NET BPND of the locus coeruleus was detected.

In conclusion, these initial data provide in vivo imaging support for the involvement of the central NE system in maladaptive eating behaviors such as susceptibility to hunger.

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1. Introduction

Assessment of eating behaviors has been applied to obesity research to evaluate potential contributing factors to this major, rapidly developing epidemic (Witten, 2016). The Three-Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985), a widely used self-report questionnaire, has been extensively used in the study of eating behaviours, measuring the constructs of cognitive restraint, disinhibition and hunger. In short, cognitive restraint of eating is defined as the degree of cognitive control in daily food intake; disinhibition is the loss of control in food intake; and hunger is described as the susceptibility for internal or external hunger signs. Lately, in a growing body of neuroimaging literature, those behavioral aspects of eating have been further elucidated. Research on structural brain alterations demonstrated a close link between eating behaviors measured by the TFEQ and brain areas involved in homeostatic keeping, habitual learning and cognitive control of food intake (Yao, Li, Dai, & Dong, 2016). In fact, higher cognitive restraint correlated positively with the gray matter volume (GMV) of a cognitive-control associated brain region, the dorsolateral prefrontal cortex (DLPFC). Inversely, higher disinhibition has been linked to lower middle frontal gyrus volume (Yao et al., 2016), lower orbitofrontal cortex volume and executive dysfunction (Maayan, Hoogendoorn, Sweat, & Convit, 2011). Susceptibility to hunger has also been found to negatively correlate with the middle frontal gyrus volume (Yao et al., 2016), which suggests an impaired cognitive control element for higher levels of
inhibition and hunger) share a similar eating behaviours with an impaired cognitive control level (disinhibition). As such, we investigated whether made above, cognitive control may be a key element among the restraint, disinhibition and hunger. Following the observations central MRB) positron emission tomography (PET) to explore whether.

2. Subjects

Details of the participants have been reported (Bresch et al., 2017; Hesse et al., 2017). In this pilot study, ten obese (6 women, aged 34.4 ± 9.0 years, BMI 42.4 ± 3.7 kg/m²) and ten non-obese HC (6 women, aged 33.3 ± 10.0 years, BMI 23.9 ± 2.5 kg/m²) participated. Briefly, exclusion criteria were current or past neurological or psychiatric illness; positive family history for psychiatric illnesses; former psychotherapy; resistant hypertension; insulin-dependent diabetes, or other medical conditions that may alter brain function; the use of central-acting drugs; participation in weight loss programs during the last 6 months; past or present history of alcohol misuse and/or illicit drug abuse; contraindications for magnetic resonance imaging (MRI, e.g. implanted ferromagnetic devices, claustrophobia); pregnancy or breast feeding. The study was approved by the ethics committee of the Medical Faculty of the University of Leipzig (IFB PET K7-7, EC number 206-10-08032010) and by the Federal Office for Radiation Protection (number Z-22461-2-2011-002). It was conducted in accordance with the ICH Guideline for Good Clinical Practice (GCP) and the Declaration of Helsinki. After complete description of the study to the participants, written informed consent was obtained.

3. Measures

3.1. Three-Factor Eating Questionnaire

We applied the German version of the Three-Factor Eating Questionnaire (Pudel & Westenholfer, 1989). It is a well-established 51-item self-report questionnaire for the assessment of three constructs: cognitive restraint (21 items), disinhibition (16 items) and hunger (14 items). Responses are scored 0 or 1 and summed; herein higher scores denote higher levels of the respective construct. This measure demonstrates adequate internal consistency (Cronbach’s alpha ranging from 0.79 to 0.92), and good convergent and discriminant validity (Allison, Kalinsky, & Gorman, 1992). Further, a study investigating a community-recruited sample of overweight individuals (N = 101) and obese individuals (N = 101) and normal weight matched controls concluded that self-report eating disorder measures (including the TFEQ) are valid and reliable among weight groups (Bohrer, Forbush, & Hunt, 2015).

4. PET and MR imaging

Detailed methods have been described previously (Hesse et al., 2017). In short, all participants underwent dynamic PET after intravenous bolus injection (90 s) of 359 ± 11 MBq [11C]MRB using the ECAT EXACT HR-2 scanner in three-dimensional acquisition mode (Siemens, Erlangen, Germany; intrinsic resolution at the centre 4.3 mm full-width at half maximum). Scans were not performed in fasting state. Participants were encouraged to have a light breakfast on the day prior to PET scanning. MRI scans were obtained using a 3T scanner (Magneton Verio, Siemens, Germany; T1-weighted 3D magnetization prepared rapid gradient echo; time of repetition 2300 ms, time of echo 2.98 ms, 176 slices, field of view 256 × 240 mm, voxel size 1 × 1 × 1 mm). Individual MR data sets were spatially reoriented onto a standard brain data set similar to the Talairach space using the image processing software PMOD version 3.4 (PMOD Technologies Ltd, Zurich, Switzerland). Volumes of interest were drawn by hand on three consecutive transversal slices of the reoriented individual MRI data sets (slice thickness: 2.5 mm). 13 different brain regions were further investigated (frontal cortex right and left (r/l), dorsolateral prefrontal cortex r/l, orbitofrontal cortex r/l, insular cortex r/l, locus coeruleus (LC), thalamus r/l, hypotalamus r/l). The regions were drawn manually by one independent researcher and experienced reader after sufficient training period. Tissue time activity curves (TACs) were obtained from the dynamic PET data using PMOD. Kinetic modeling of these regional brain TACs was performed using the nonlinear reference tissue model (Breteler et al., 1989). It is a well-established model of a target region with rate constants and the 1-tissue reference tissue model (Hesse et al., 2017; Hesse et al., 2018) with the occipital cortex as reference region (Hannestad et al., 2010) and simultaneously by applying the 2-tissue compartment model of a target region with rate constants and the 1-tissue compartment model of the reference region. Regional NET binding potential (BPND) values were then calculated (Hesse et al., 2017). SPSS 20 software (SPSS Inc., Chicago, IL, USA) was used to analyze the data. All data are presented as mean and standard deviation, unless otherwise stated. The data were tested for normal distribution using the Shapiro-Wilk test. All our data were normally distributed. Differences in normally distributed data were tested using the Student’s t-test. Pearson product moment correlation was

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<th>Definition</th>
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<td>body mass index</td>
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<td>BPND</td>
<td>binding potential</td>
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<td>DLPFC</td>
<td>dorsolateral prefrontal cortex</td>
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<td>fMRI</td>
<td>functional magnetic resonance imaging</td>
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<td>GMV</td>
<td>gray matter volume</td>
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<td>HC</td>
<td>healthy controls</td>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
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<td>MRTM</td>
<td>multilinear reference tissue model</td>
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<td>NE</td>
<td>norepinephrine</td>
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<td>NET</td>
<td>norepinephrine transporter</td>
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<td>TFEQ</td>
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