

with psychiatric disorders, more often represented by eating disorders. The manifestations of these disorders could influence overall functioning, but also treatment adherence and disease outcomes before, during, and after bariatric surgery, as demonstrated by the higher prevalence of altered eating habits in subjects with psychiatric disorders even in the absence of a comorbid eating disorder. An early screening for psychiatric disorders in overweight patients should be essential. Therefore, early screening can be important to provide early intervention and treatment in this vulnerable population.

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NEUROSCIENCE APPLIED 2 (2023) 102441 102753 NEURONAL RESPONSES TO VISUAL FOOD CUES ACCORDING TO WEIGHT AND HUNGER STATUS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: The neuronal mechanisms underlying food preferences have yet to be fully elucidated. Central food cue reactivity (FCR) can vary considerably depending on weight and hunger status of individuals, or the calorie content of the food [1]. In functional magnetic resonance imaging (fMRI) literature on FCR, these key variables are sometimes disregarded, potentially explaining inconclusive findings. Moreover, the field often fails to consider the implications of positivity bias, small sample sizes, lenient statistical thresholding, and inadequate contrast conditions in both original studies and reviews [2, 3]. To overcome these limitations, we conducted a systematic review and meta-analysis to investigate the effects of weight status (lean, overweight, and obese), hunger status (fasted, slight hunger, and satiated), and food calorie content on brain responses while viewing or rating of food pictures.

Methods: Following a preliminary search, we conducted a systematic review in accordance with the PRISMA guideline in PubMed and Web of Science databases [4]. The search spanned from January 1, 2000, to October 31, 2022. The inclusion criteria were studies involving healthy adults with stated body mass index (BMI) who were exposed to visual food cues inside the MRI scanner. The main outcomes were a) fMRI-derived brain regions modulated in response to visual food cues according to weight (and hunger) status and b) specifications such as direction of β and contrasts, fMRI acquisition parameters, and task designs. For more details, please see PROSPERO preregistration #CRD42022365310. Quality was evaluated according to state-of-the-art fMRI analysis together with established scales. For meta-analysis, we defined "Brainmap GingerALE software" [5] and leave-one-out jackknife for sensitivity analysis.

Results: In our main search, we retrieved 2,451 papers (1,116 PubMed, 1,335 Web of Science). After removing 1,070 duplicates, two reviewers screened and excluded 1,174 papers and considered 207 papers eligible for full-text reading, of which a total of 62 papers with 1552 participants were included. Overall, stimuli, task instructions, and analyses varied considerably. Based on preliminary results, comparing food vs. nonfood viewing, 21 studies involving 448 lean individuals reported increased neural activity in regions including the insula, amygdala, nucleus accumbens, thalamus, and prefrontal including left orbitofrontal cortex. Meanwhile, five studies with 125 participants living with obesity, showed heightened brain activation in overlapping regions such as insula, amygdala, orbitofrontal cortex, and also in the putamen, caudate, occipital, inferior temporal, fusiform, and frontal gyrus. In addition, six studies with 198 participants showed greater activation in the temporal gyrus, hippocampus, and prefrontal cortex while comparing food viewing in obese versus lean. As next step, we will perform meta-analysis, in-depth quality assessment, and extraction of the results depending on food stimuli calorie content and hunger status.

Conclusion: In this systematic review, preliminary results indicate that central FCR differs depending on weight status. For instance, individuals with obesity recruit more widespread brain areas such as medial temporal and occipital

regions when viewing food compared to nonfood. The forthcoming meta-analysis will serve to provide a better understanding of neural correlates of FCR in health and disease and eventually eating behavior and enhance the validity of their findings.

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NEUROSCIENCE APPLIED 2 (2023) 102441 102754 THE USE OF LISDEXAMFETAMINE IN BINGE-EATING DISORDER – COULD IT WORK FOR BULIMIA AS WELL?

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Introduction: In 2015, Lisdexamfetamine (LDX), a stimulant drug mainly used in the treatment of Attention Deficit and Hyperactivity Disorder (ADHD) became the first Food and Drug Administration (FDA)-approved treatment for binge-eating disorder (BED), with evidence supporting its efficacy in reducing both bingeing episodes and body weight in BED patients. Both BED and Bulimia Nervosa (BN) share binge eating as a core feature. Moreover, a common neurobiological substrate involving inhibitory control, reward circuitry and dopaminergic neurotransmission has been proposed in recent studies. As such, significant overlap between the explanatory models of eating disorders and ADHD has been pointed out, as well as significantly higher ADHD symptom prevalence in ED, with the presence of inattentive ADHD symptoms predicting nonrecovery in ED patients. This supports the case for the use of stimulants in BN and BED.

Aims: To perform a narrative review of the available evidence regarding the use of Lisdexanfetamine in BED, its mechanism of action and potential use in Bulimia Nervosa.

Methods: Using the PubMed database, we performed a literature search in two phases: in the first one, we used the MeSH terms for BED and LDX, retrieving a total of 41 results, and in the second one we replaced BED with the BN MeSH terms obtaining only 9 results. Results were screened by assessing of titles and abstracts of the studies, including only the most relevant and recent ones.

Results: In five Randomized Clinical Trials (RCTs), LDX has showed effectiveness in reducing binge eating frequency, binge eating symptoms (measured through CGI and YBOCS-BE), body weight and triglyceride levels in BED patients. In studies using rat models, LDX also reduced food intake. The proposed mechanism of action of LDX involves the increase of dopaminergic, serotonergic and noradrenergic neurotransmission in the prefrontal cortex and striatum reducing appetite and hypersensitivity to food stimuli by acting upon reward pathways and reducing impulsivity in BED patients. This could also benefit BN patients, however, little research has focused on the use of LDX in BN, with no published RCTs. Nevertheless, a series of six case reports has found reductions on the number of binge/purge days per month with the use of LDX. Other case reports have reported similar findings with the use of other stimulants on BN cases, focusing on comorbidity with ADHD. There is also an 8-week open-label feasibility study with 23 participants, finding an association of LDX with reductions in