PWE-172 A NATIONAL BIOFEEDBACK PRACTITIONERS SERVICE EVALUATION

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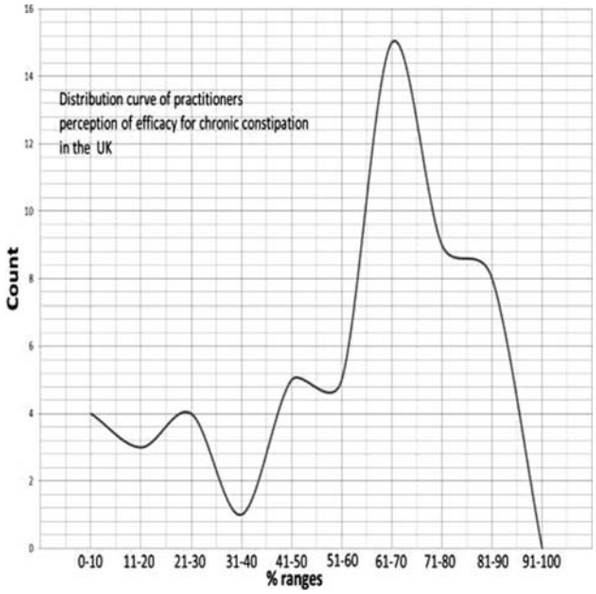
Introduction Within the UK, there is anecdotal evidence of disparity in the training, practice, supervision, and perception of efficacy amongst practitioners of biofeedback therapy for chronic constipation.

Methods Between October 2012 and October 2013, a prospective service evaluation was distributed to biofeedback practitioners in the UK through academic conferences or by invitation to complete an online assessment form.

Results 76 practitioners responded, consisting of nurses (47%), physiotherapists (35%), physiologists (12%) and others (7%). Only 45% described 'biofeedback' consistent with an accepted definition. 86% use equipment to provide sensory feedback. Methods of biofeedback varied: balloon catheter (54%), brace pump technique (78%), urge resistance (83%), irrigation (16%), and relaxation (12%). Only 65% of practitioners had attended formal training courses, and 52% considered themselves to be self-tau, UKght. 36% receive formal supervision and only 38% of those by a senior. Regular audit of outcomes is undertaken by 67%. UK-wide perception of treatment response for chronic constipation is markedly variable (mean response = 57% [IQR: 50–75%, SD 23%]); there were no differences in perception of treatment response between nurses or physiotherapists. Practitioners' free responses demonstrated strong positive themes of a holistic approach and an overall perception of effectiveness. Negative themes included service restrictions.

Conclusion There is marked variation in practice, training, and supervision of biofeedback therapists throughout the UK. Perceptions of efficacy vary greatly. Development of training and supervision standards is a priority as well as consensus to standardise therapy.

Disclosure of Interest None Declared.



Abstract PWE-172 Figure 1

PWE-173 PERIPHERAL AND CENTRAL EFFECTS OF A SINGLE DOSE OF THE GLUCAGON-LIKE PEPTIDE-1 RECEPTOR AGONIST EXENATIDE

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Introduction Administration of the glucagon-like peptide-1 receptor agonist (GLP-1RA) exenatide reduces food intake and induces weight loss. GLP-1 receptors are located throughout the body including the brain and vagal afferent neurones. Although exenatide slows gastric emptying (GE), it is believed the main mechanism behind exenatide-induced weight loss may be related to activation of central pathways that mediate satiety. However, research in humans is lacking. Brain activation can be indirectly measured by quantifying changes in the blood oxygen level dependent (BOLD) signal measured during functional magnetic resonance imaging (fMRI).

Methods To confirm a gastric inhibitory effect of exenatide, 12 healthy volunteers received a subcutaneous injection of 5 μg exenatide or placebo on separate occasions 60min prior to consumption of a 0.5 M carbohydrate test drink (500 mL). Over 45 min GE was non-invasively measured using breath tests. Then, to examine brain activation using fMRI, 15 healthy volunteers received 5 μg exenatide or placebo after ~10 h fast and underwent 70 min scanning (3T Philips scanner). Wholebrain analysis was conducted (data for the hypothalamus only are reported). For each volunteer the difference in mean%BOLD signal change from baseline between exenatide and placebo was extracted and analysed in 5min blocks using T-tests. Two volunteers were excluded due to excessive movement during scans.

Results Repeated measures ANOVA revealed that exenatide significantly slowed GE of the test drink with the area under the curve for exenatide being 31% lower than placebo ($p < 0.005$). fMRI analysis revealed bilateral increases in BOLD signal in the lower hypothalamus ($p < 0.05$), and a trend for increased BOLD ($p < 0.075$) in the right upper hypothalamus 45 min after exenatide. Conversely, BOLD signal was decreased in the left upper hypothalamus 40min after exenatide ($p < 0.05$).

Conclusion Our data confirms that a single dose of exenatide potently delays GE. Furthermore, most regions of the hypothalamus, a key appetite regulatory brain area, were progressively activated 45 min after exenatide administration. Those volunteers were fasted which suggests that the anorectic effects of exenatide in humans could be mediated via activation of central GLP-1 receptors, either directly or via vagal inputs. Further analysis will now be conducted to examine the effect of exenatide on areas in the brain associated with food reward such as the insula and orbitofrontal cortex.

Disclosure of Interest None Declared.

PWE-174 A PROOF OF CONCEPT ASSESSMENT OF NON-INVASIVE VAGUS NERVE STIMULATION (NVNS) WITH GAMMACORE® IN PATIENTS WITH GASTROPARESIS AWAITING ENTERRA® IMPLANTATION

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