Wardle, R. A., Thapaliya, G., Nowak, A., Radford, S., Dalton, M., Finlayson, G., & Moran, G. W. (2018). An examination of appetite and disordered eating in active Crohn's disease. Journal of Crohn's and Colitis, 12(7), 819-825. https://doi.org/10.1093/ecco-jcc/jjy041

An examination of appetite and disordered eating in active Crohn's disease

Richard Anthony Wardle [a 1], Gita Thapaliya [a 2], Adam Nowak [a 3], Michelle Dalton [b 4], Graham Finlayson [c 5], Gordon W. Moran [a 6]

[a] National Institute for Health Research (NIHR) Biomedical Research Centre in Gastrointestinal and Liver Diseases at Nottingham University Hospitals NHS Trust & The University of Nottingham, Queens Medical Centre Campus, E Floor, West block, Nottingham, NG7 2UH, UK

[b] School of Social and Health Sciences, Leeds Trinity University, Leeds, UK

[c] Human Appetite Research Unit, School of Psychology, Faculty of Medicine and Health, University of Leeds, Leeds, UK

[1] Richardwardle@nhs.net

[2] Gita.thapaliya@nottingham.ac.uk

[3] Adam.nowak@nhs.net

[4] M.dalton@leedstrinity.ac.uk

[5] G.s.finlayson@leeds.ac.uk

[6] Gordon.moran@nottingham.ac.uk

Trial registration number was NCT02379117

Hard Copy Poster P105 Presented at 12th Congress of ECCO - Inflammatory Bowel Diseases 2017 February 15-18, 2017 in Barcelona, Spain

Short Title

Eating Behaviour In Crohn's disease: EBIC study

Corresponding author

G. W. Moran

NIHR Nottingham Digestive Diseases Biomedical Research Centre in Gastrointestinal and Liver Diseases, Nottingham University Hospitals NHS Trust & The University of Nottingham, Nottingham, United Kingdom

E-mail: Gordon.Moran@nottingham.ac.uk

Telephone no: +44 (0)115 9249924 ext 70608

Authors' contributions

Richard Anthony Wardle

Conception and design of study, acquisition of data, analysis and/or interpretation of data, drafting the manuscript, revising the manuscript critically for important intellectual content, approval of the version of the manuscript to be published.

Gita Thapaliya

Acquisition of data, analysis and/or interpretation of data, drafting the manuscript, approval of the version of the manuscript to be published.

Adam Nowak

Acquisition of data, approval of the version of the manuscript to be published.

Michelle Dalton

Analysis and/or interpretation of data, drafting the manuscript, revising the manuscript critically for important intellectual content, approval of the version of the manuscript to be published.

Graham Finlayson

Conception and design of study, analysis and/or interpretation of data, drafting the manuscript, revising the manuscript critically for important intellectual content, approval of the version of the manuscript to be published.

Gordon W. Moran

Conception and design of study, acquisition of data, analysis and/or interpretation of data, drafting the manuscript, revising the manuscript critically for important intellectual content, approval of the version of the manuscript to be published.

We have not received substantial contributions from non-authors.

Financial conflicts of interest

There are no financial conflicts of interest

This manuscript, including related data, figures and tables has not been previously published and is not under consideration elsewhere.

ABSTRACT

Background

Crohn's disease (CD) patients suffer from nutritional deficiencies when in active disease. We aim to examine calorific intake, macronutrient choice and disordered eating behaviour in patients with active CD.

Methods

CD patients with active disease were recruited with matched healthy volunteers (HV). Active disease was defined by faecal calprotectin >250ug/g, C-reactive protein >5mg/dl, or active disease seen on endoscopy or imaging. Symptoms were quantified by Harvey-Bradshaw Index (HBI). Calorific intake was assessed by 24-h dietary recall. Disordered eating was assessed using validated questionnaires [Binge Eating Scale (BES); Power of Food Scale (PFS); Control of Eating Questionnaire (CoEQ); Dutch Eating Behaviour Questionnaire (DEBQ); Three Factor Eating Questionnaire (TFEQ)].

Results

21 CD (14M:7F, Age:33.3±2.3, BMI:24.9±1.0) and 21 matched HV (14M:7F, Age:33.8±2.4, BMI:25.1±0.6) were recruited. Mean HBI was 5.1±0.8, faecal calprotectin 640±140µg/g and C-reactive protein 27±7.2mg/dl. There were no differences in calorific or macronutrient intake between groups. CD were characterised by higher BES (p=0.001), TFEQ-Disinhibition (p=0.006) and Hunger (p=0.014), DEBQ-Emotional (p=0.001) and External eating (p=0.022), PFS (p=0.007) and by lower CoEQ-Craving control (p=0.016) with greater craving for savoury compared to HV (p=0.005). There were no differences in dietary restraint measures. Hospital Anxiety and Depression score was higher (p=0.002) and CoEQ-Positive Mood (p=0.001) lower in CD.

Conclusions

Disordered eating behaviour traits were more prevalent in CD. Greater prevalence of binge eating may be attributed to lower mood and higher anxiety observed. Higher scores on measures of hedonic eating indicates that stronger psychological support with firm dietetic advice for healthy eating is warranted.

Keywords

Inflammatory Bowel Disease, Crohn's disease, eating behaviour, nutrition

INTRODUCTION

Patients with gastrointestinal disorders are at a greater risk of a disordered eating pattern compared to healthy volunteers with an increased prevalence of a wide range of abnormal eating patterns such as binge eating, meal skipping and food restriction [1,2]. Disordered eating behaviour applies to most patients with gastrointestinal disease and may include food restriction, meal skipping and over-eating rather than the more severe eating disorders where patients are diagnosed according to specific narrow criteria. A disordered eating behaviour may be described with a two-path theoretical model[1,2]. The first pathway concerns individuals who experience high levels of anxiety about unfamiliar foods and/or overestimate the negative consequences associated with their condition. These individuals may restrict their intake to self-prepared and familiar foods limiting their diet variety. The second pathway concerns individuals who gain weight when following their prescribed dietary regimen and subsequently employ techniques to reduce this weight gain.

In Inflammatory Bowel Disease (IBD), issues regarding food intake are felt to be either important or extremely important in 62.5% of patients, with virtually all Crohn's disease (CD) patients having had problems with unintentional weight loss [3]. Abnormal eating patterns have been described in IBD with qualitative studies unselectively describing eating behaviour irrespective of disease activity[4,5]. Approximately three-fourths of patients with IBD describe a decline in appetite when the disease is active[4] with up to 37% of Crohn's disease (CD) patients show abnormal eating patterns [6]. Malnutrition is more prevalent in CD than ulcerative colitis with up to 75% of hospitalised patients being malnourished with 50% in negative nitrogen balance [7]. To this effect, the IBD priority-setting partnership set up by the James Lind Alliance identified a research need to understand a role for diet in disease management[8]. The effect of disordered eating on the nutritional status in CD has never been investigated.

Appetite and satiety involve complex interactions between homeostatic and hedonic factors. The enteroendocrine-gut brain axis is central to the homeostatic control of food intake, whilst other neural circuits integrate environmental and emotional cues to constitute the hedonic drive of appetite regulation [9]. The cross-link between eating behaviour and active CD is poorly understood. Disordered eating might be associated with a change in the homeostatic and hedonic balance. The aim of this study is to examine free-living calorie and macronutrient intake in patients with active CD compared to healthy volunteers and to determine the prevalence and type of eating behaviour traits and disordered eating in CD patients with active disease.

METHODOLOGY

Basic protocol and patient recruitment

This was an open label, qualitative questionnaire-based study with a matched-pair design. The study was conducted between July 2015 and December 2016 at the National Institute of Health Research (NIHR) Nottingham Digestive Diseases Biomedical Research Centre (NDD BRC) at the Queens Medical Centre Campus, Nottingham, UK. Participants were recruited from The Inflammatory Bowel Disease Clinic, via the study flyer and social media. CD patients (aged 16-75yrs) with active disease were recruited as well as age, BMI and gender-matched healthy volunteers. Healthy volunteers (HV) were recruited form an existing participant database in the Nottingham BRC and from the local healthy populations of Nottingham University Hospitals and the University of Nottingham. This study was advertised through study fliers and social media.

Disease activity was defined through objective markers of inflammation: faecal calprotectin of >250µg/g or CRP of >5g/dl or through recent ileocolonoscopy, CT or MR enterography showing active inflammatory and uncomplicated disease (not of a stricturing or penetrating behaviour). CD clinical activity was measured with a Harvey Bradshaw Index[10](HBI) score recorded at inclusion. Potential participants with a BMI of <18 or >30kg/m^2, recent corticosteroid use (in the last 3 months), pregnancy or breast-feeding and patients with significant co-morbidities were excluded from the study. Stable doses of immunosuppressive agents or anti-TNF agents were permitted.

All CD patients and healthy volunteers gave their informed consent prior to recruitment. All participants were included in this study for a maximum of one week. Participants completed a single, spontaneously administered 24hr dietary recall either face-to-face at the NDD BRC or by telephone, the Hospital Anxiety and Depression scale (HADS) and five psychometric eating behaviour questionnaires within the study period.

Outcomes

The primary outcome of this study was to compare total 24 hr calorie intake as measured by a single face-to-face or telephone-administered 24-hour dietary recall [11] between CD with active disease and age-, BMI- and gender-matched HV. Calories consumed were calculated for the recall based on manufacturers' labels and the nutrition analysis tool Nutritics (Nutritics v4.312 Academic Edition, Ireland). Dietary recall did not include caloric intake from weekends or holidays but only days Monday to Thursday. The secondary endpoint for this study was to measure eating behaviour traits through psychometric scales: Three Factor Eating

Questionnaire (TFEQ) [12]; the Binge Eating Scale [13]; the Power of Food Scale [14]; the Dutch Eating Behaviour Questionnaire [15]; and the Control of Eating Questionnaire [16,17].

24-h dietary recall

The Automated Multiple-Pass Method (AMPM) was utilised to perform the single spontaneously administered 24hr dietary recall. This five-step questionnaire can accurately assess dietary consumption and may be administered face-to-face or by telephone [11,18]. RW, AN and GT conducted all interviews. A copy of the dietary assessment textbook Carbs and Cals was provided to each participant to facilitate the dietary recall [19]. This book contains over 1700 food and drink photographs and was primarily used to assist in identifying the appropriate food type and portion size consumed. Diet logs were analysed using Nutritics dietary analysis software (Nutritics v4.312 Academic Edition, Ireland).

Eating Behaviour traits

Eating behaviour traits were measured through five validated self-report questionnaires; the Power of Food Scale (PFS); the Binge Eating Scale (BES); the Control of Eating Questionnaire (COEQ); Three Factor Eating Questionnaire (TFEQ) and the Dutch Eating Behaviour Questionnaire (DEBQ) [12-16].

<u>The Power of Food Scale (PFS)</u>

The PFS is a 15-item questionnaire reflecting the psychological influence of the food environment. It measures appetite for, rather than consumption of palatable foods and may be a useful measure of the hedonic impact of food environments replete with highly palatable foods [20]. Items are grouped into three domains according to food proximity; food available but not physically present, food present but not tasted and food tasted but not consumed.

<u>The Binge Eating Scale (BES)</u>

The BES is a 16-item questionnaire that assesses the severity of binge eating tendencies. Eight questions describe the behavioural manifestations of binge eating behaviour and eight describe the feelings and cognitions associated with binge eating. Scores are summed to produce a total score ranging from 0 to 46. Cut off points have previously been reported denoting mild (≤17), moderate (18–26), and severe (≥27) binge eating behaviour [13,21,22].

<u>The Control of Eating Questionnaire (CoEQ)</u>

The CoEQ is a 21-item questionnaire designed to assess the severity and type of food cravings experienced over the previous seven days [16]. The CoEQ has four subscales; Craving Control, Craving for Savoury, Craving for Sweet and Positive Mood. Items on the CoEQ are

assessed by 100-mm visual analogue scales (VAS) with items relating to each subscale being averaged to create a final score.

Three Factor Eating Questionnaire (TFEQ)

The TFEQ contains 51-items and measures three dimensions of human eating behaviour; Cognitive Restraint of Eating, Disinhibition and Hunger [12]. Restraint refers to concern over weight control and strategies which are adopted to achieve this. Disinhibition reflects a tendency towards over-eating and eating opportunistically in an obesogenic environment. Hunger is concerned with the extent to which hunger feelings are perceived and the extent to which such feelings evoke food intake [23]. Each item scores either 0 or 1 point. The minimum score for factors I, II and III is therefore 0, with the possible maximum scores being 21, 16 and14 respectively.

The Dutch Eating Behaviour Questionnaire (DEBQ)

The 33-item DEBQ assesses different eating styles that may contribute to weight gain; emotional eating, external eating, and restraint. 'Emotional eating' occurs in response to emotional arousal states such as fear anger or anxiety, 'external eating' in response to external food cues such as sight and smell of food and 'restraint eating' is overeating after a period of slimming when the cognitive resolve to diet is abandoned[15].

Statistical Analysis

The sample size was based on published data where the 24hr self-reported calorie intake in CD was 1978.7±169.7Kcal and that in HV was 1854.4 ±129.5Kcal. Assuming α of 0.05, power of 80% and using 2-sided test, 30 participants in each group were needed to show a significant difference in the primary outcome.

Data were analysed using SPSS version 20 for Windows. The parametric or non-parametric nature of the data was determined by a normality test. Data is presented as mean ± standard error of the mean (SEM). Continuous data was compared using paired t-test while categorical data was compared with Chi-Squared test. Total 24hr Kcal intake, macronutrient intake together with outcome data from the individual questionnaires administered to all participants were compared between the groups. An exploratory sub-analysis was undertaken comparing differences between gender. P values <0.05 were deemed significant.

Ethical approval

This study received research ethics committee approval from National Research Ethics Service (NRES) Committee East Midlands (REC reference 15/EM/0142 as of the 27th April 2015). The protocol was registered with clinical trials.gov (NTC02379117).

RESULTS

Demographic data

Thirty CD patients (18M:12F, Age:32.3±2.19, BMI:24.9±0.8) and 31 matched HV (19M:12F, Age:32.8±2.0, BMI:24.7±0.5) were recruited to this matched pairs cross-sectional study (see Table 1). There were no significant differences in gender ratio, mean age and mean BMI between the CD and HV. CD patients had clinically active disease with a mean HBI score of 7.2±0.7 and objective evidence of active disease with an elevated C-reactive protein (83.8±47.1mg/L), or faecal calprotectin (1009.2±192.9µg/g) or as assessed by colonoscopy or MR enterography or both (see supplementary table). These objective investigations have been undertaken as part of the patients' standard of care within a mean of 52.9±14.1 days of recruitment. None of the patients had any change in management prior to recruitment and data collection.

Table 1: Summary demographic data of participants

Group	Gender (n)	Age	BMI
CD	M (18)	31.1±2.7	24.1±1.1
	F (12)	34.1±3.8	26.1±1.2
HC	M (19)	32.6±2.3	24.7±0.6
	F (12)	33±3.9	24.8±1.0

24-hour calorie intake

The total self-reported 24-hour calorie and macronutrient intake for the CD cohort and HV are shown in Table 2. There were no significant differences observed in total energy intake between cohorts. Protein intake was significantly lower in the CD cohort (CD, 92.6g±7.8; HV, 70.3g±6.1; p=0.003). There was no significant difference in the consumption of all other macronutrients.

In a sub-analysis of this data setaimed at investigating difference by gender, the 24hr calorie intake of male CD patients was not significantly different to male HV. In female participants, 24-hour calorie intake was significantly reduced in the CD cohort compared with HV (CD, 1519.3±136.5; HV, 2039.4Kcal±133.8; p=0.01). In female participants consumption of carbohydrate (CD, 187.9g±19.9 HV, 270.1g±22.3, p=0.01), sugars (CD, 78.9±8.5; HV, 107.5±9.3; p=0.03) and fibre (CD, 15.9g±2.6; HV, 25.9g±3.8; p=0.04) was significantly less than in HV.

Table 2. 24-hour self-reported calorie and macronutrient intake in CD and HV. Data is presented as mean and Standard error of the mean

	CD (total)	CD (male)	CD (female)	HV (total)	HV (male)	HV (female)
Total (Kcal)	1900.9± 138.6	2187± 193.7	1519.3± 136.5	2054.3 ± 110.7	2065± 167	2039.4± 133.8
Carbohydrate (g)	248.4± 20.7	293.7± 28.5	187.9± 19.9	255.9± 17.3	245.9± 25.3	270.1± 22.3
Sugar (g)	97.8± 8.1	112± 11.6	78.9± 8.5	101.7± 7.4	97.6± 10.9	107.5± 9.3
Protein (g)	70.3± 6.1	74.0± 8.5	65.4± 8.8	92.6± 7.8	101.6± 12.2	79.9± 6.8
Fat (g)	69.7± 6.2	79.4± 8.2	56.9± 8.3	72.3± 5.3	73.8± 8.1	70.2± 6.2
Saturated Fat (g)	23.1± 2.1	26.2± 2.5	18.9± 3.3	23.1± 2.2	23.6± 3.4	22.5± 2.5
Fibre (g)	18.9 ± 2.1	21.2± 3.0	15.9± 2.6	23.4± 2.3	21.7± 2.8	25.9± 3.8
Alcohol (g)	3.5± 1.8	5.0± 2.9	1.5± 1.5	4.6± 1.9	5.5± 2.8	3.4± 2.2

Hospital Anxiety and Depression Scale

CD patients had significantly higher scores on the Hospital Anxiety and Depression scale compared to HV (CD, 13.4±1.6; HV, 7.4±1.5; p=0.01) (see Table 3). This was evident for both anxiety (CD, 8.6±0.9; HV, 4.2±0.7; p=0.001) and depression (CD, 6±0.9; HV, 1.8±0.3; p=0.00) subscales.

In addition, CD patients scored significantly lower on the CoEQ Positive Mood subscale (CD, 50.8±3.3; HV, 64.8±2.5; p=0.001).

Eating Behaviour traits

Table 3 shows the outcomes from the psychometric eating behaviour questionnaires for CD and HV. CD patients scored higher on BES compared to HV (CD, 10.9±1.9; HV, 5.2±1.0; p=0.01) and a greater proportion of CD patients (29%) scored above the clinical cut-off criteria for moderate levels of binge eating (>17 BES) compared to HV (3.3%) [χ^2 (1) = 7.0, p=0.008].

CD patients reported lower levels of CoEQ Craving Control (CD, 56.16±3.5; HV, 66.4±2.9; p=0.027) and greater craving for sweet (CD, 48.9±4.4; HV, 37.3±3.5; p=0.043) and savoury (CD, 48.9±3.5; HV, 38.3±2.7; p=0.021) foods compared to HV. CD patients had higher scores on the PFS food present (CD, 11.7±0.7; HV, 9.0±0.6; p=0.005) subscale. No significant difference was seen however for overall PFS score or food available or tasted subscales.

In addition, CD patients scored higher on the DEBQ Emotional Eating (CD, 36.4±3.7; HV, 20.0±1.7; p=<0.001) and External Eating (CD, 30.8±1.9; HV, 25.2±1.2; p=0.022) subscales compared to HV. However, there was no difference in restraint assessed by either or DEBQ (CD, 23.7±2.7; HV, 21.6±1.9; p=0.528) the TFEQ (CD, 6.4±0.9; HV, 8.4±0.9; p=NS) between CD and HV.

Table 3. Eating behaviour traits in CD participants and age-, BMI- and gender-matched HV.

	CD	HV	Sig. (2-tailed)
HADS	13.4±1.6	7.4±1.5	0.01
HADS: Anxiety	8.6±0.9	4.2±0.7	0.001
HADS: Depression	6.0±0.9	1.8±0.3	0.001
BES	10.9±1.9	5.2±1.0	0.01
PFS	35.6±2.4	31.0±1.9	0.27
PFS: Available	12.1±1.2	10.5±0.9	0.28
PFS: Present	11.7±0.7	9.0±0.6	0.005
PFS: tasted	11.7±0.8	11.7±0.8	0.954
CoEQ: Control	56.2±3.5	66.4±2.9	0.027
CoEQ: Sweet	48.9±4.4	37.3±3.5	0.043
CoEQ: Savoury	48.9±3.5	38.2±2.7	0.021
CoEQ: Mood	50.7±3.3	64.8±2.5	0.001
TFEQ: R	5.9±0.9	8.4±0.9	0.114
TFEQ: D	6.1±0.8	4.5±0.6	0.135
TFEQ: H	5.5±0.8	4.0±0.5	0.142
DEBQ: R	23.7±2.7	21.6±1.9	0.528
DEBQ: Em	36.4±3.7	20.0±1.7	<0.001
DEBQ: Ex	30.8±1.9	25.2±1.2	0.022

When analysed by gender, male CD showed significant difference in HADS (CD 13.5±2.1; HV, 4.3±1; p=0.001), BES (CD, 7.3±1.6; HV, 3.4±0.8; p=0.04) CoEQ: Control (CD 58.9±4.4; HV, 70.5±3.3; p=0.04) CoEQ: Sweet (CD, 51.5±6.2; HV, 32.9±4.1; p=0.01), TFEQ: Restraint (CD, 4.1±0.8; HV, 8.3±1.1; p=0.00) and DEBQ: Emotional (CD, 31.4±4.2; HV, 18.9±1.9; p=0.01) when compared with male HV.

Female CD showed significant difference in HADS (CD, 15.9±2.9; HV, 8.6±1.6; p=0.04), PFS: Present (CD, 12.8±1; HV, 9.6±1; p=0.04), CoEQ: Mood (CD, 44.1±5.2; HV, 64.1±4.1; p=0.006) and DEBQ: Emotional (CD, 43.8±6; HV, 22±3.4; p=0.01) when compared with female HV.

DISCUSSION

A poor nutritional status has always been associated with CD but a detailed analysis of eating behaviour in this cohort compared to matched HV has never been undertaken. The primary aim of this study was to compare the total self-reported 24 hr calorie intake in CD with active disease and HV. The main secondary aim was to examine whether CD patients with active disease had a greater prevalence of disordered eating patterns compared to HV. We found no substantial difference in the 24-hour self-reported calorie intake between CD patients with objective evidence of intestinal inflammation and HV. Analysing the data further by gender reveals that a significant decrease in calorie intake is observed in female rather than male CD patients with this reduction in food intake consisting mainly of a reduction in carbohydrates in females and protein in males. This finding is novel and contrasts with observations made inprevious studies that have showed no difference in energy intake in CD patients with both active and inactive disease[2425]. These differences in food intake may be explained by the two-path theoretical model; with CD patients experiencing high levels of anxiety to food intake, thus restricting food variety to minimise symptom aversion.

An increased prevalence of disordered eating behaviour traits was observed in CD with a greater prevalence of binge eating, food craving, lower mood and higher anxiety states observed in this group. Patients with gastrointestinal disorders are reported to suffer from disordered eating behaviour with more than a third of CD patients thought to be affected [6]. In the present study, it was demonstrated that CD patients scored significantly higher on measures of binge eating and hedonic responsiveness compared to HV. Binge eating traits were more prevalent as revealed by a significantly higher BES together with significantly stronger cravings with less ability of self-control. The CoEQ showed that CD patients had less control of their cravings, with significantly greater cravings for both sweet and savoury foods.

Significantly higher scores on the hedonic eating traits (i.e. BES, PFS, DEBQ-External) in CD may be associated with increased food monitoring behaviour that occurs in patients with dietary-controlled conditions, something that should be examined in future research. These findings are consistent with previous research that have demonstrated a higher level of disordered eating patterns in individuals with gastrointestinal disorders [1,2]. In a questionnaire-based study in 400 consecutive IBD patients in the UK[4], approximately half of the patients felt that diet was the initiating factor in IBD and subsequent relapses. The majority of patients'

symptoms were triggered by food with two-thirds of the patients depriving themselves of their favourite food to achieve symptom control. A case-control study of 104 patients with an established diagnosis of IBD[26] concluded that avoidance of meat, nuts, fruit and vegetables are more common among patients with IBD than healthy controls. This corresponds with the findings of this study where the consumption of protein was significantly reduced overall and carbohydrate, sugar and fibre intake were reduced in females.

The current study also demonstrated that CD patients had lower levels of positive mood as measured by the CoEQ and higher scores on the HAD scale. Greater levels of psychological distress have been linked to increased binge eating prevalence and in the current study we found that scores on the BES were negatively associated with positive mood. Similarly, we found a higher prevalence of emotional eating in the DEBQ. These findings have important implications for the role of mood and psychological distress in the aetiology of gastrointestinal disorders and their association with abnormal eating patterns [27]. For example, it is possible that psychological distress may serve as both a cause and a consequence of disordered eating behaviours [1]. Arigo et al suggested that fear and anxiety surrounding gastrointestinal symptoms may lead to disordered eating practices of a restrictive nature, as observed in this study [28]. This increased anxiety may link directly to the personal attitudes and beliefs that patients hold about food. In a French survey of 244 IBD patients, nearly half of the study patients reported that the disease had changed the pleasure of eating[5] with only a quarter of the patients eating a normal diet when they relapse. Such a behaviour influenced patients' social life in 25% of the cases. This might have a negative effect on mood and depressive symptoms.

Disease activity has been quantified with objective markers of disease activity and intestinal inflammation present in all our recruited cohort. Clinical scores were quantified through HBI. Gastrointestinal symptom severity may also play an important role in the development of disordered eating patterns, with greater symptom severity correlating positively with the risk of disordered eating[30].

When analysed by gender, female CD patients consumed significantly less calories than female HV with reduced consumption of carbohydrate, sugar and fibre. This was not observed in male participants. Male CD participants displayed greater hedonic responsiveness with higher BES, lower CoEQ Control and TFEQ:Restraint compared with male HV. In females CD participants, significantly higher PFS: present and DEBQ: Emotional with lower CoEQ: moodwhen compared with female HV might imply female CD patients may be predisposed to emotional eating. These results seem may suggest that female patients have similar level self-control over dietary consumption as HV. Consequently, females with CD may be less likely to

binge eat during active disease, being more likely to display inadequate calorie consumption as displayed by this study. Male CD participants display greater hedonic responsiveness, with higher prevalence of binge eating with the consequence of normalising calorie consumption to that of HV male participants. It is important to highlight that this study was not powered to analyse the difference in eating behaviour by gender so such conclusions are hypothesis-generating.

We believe that for the first time, this study highlights in detail the important behavioural differences that may be observed in patients with active CD. This study has some limitations that need to be considered. This was an 18-month long prospective study aiming to compare calorific intake and the eating behaviour of CD patients with active disease to matched healthy volunteers. The sample size despite relatively small was appropriately powered based on the group's previous pilot data. Daily activity levels are an important confounder that were not measured. Physical inactivity has already been shown in CD[31,32], and has been significantly correlated to disease activity but is still prevalent in remission[33]. Due to the small sample size, we did not investigate the effect of disease burden surrogates: disease duration, concomitant medication and surgical history in CD patients on eating behaviour. Nevertheless, the CD cohort recruited is representative of a CD cohort with moderate disease burden, making our findings generalizable to world-wide healthcare systems.

The use of the AMPM as a single administered 24-hour recall is limited, and accuracy may have been improved if this was performed on three consecutive days rather than one day. However, this method has been used successfully in previous research [18]. The 24-hour recall technique is also memory dependent and participants' potential bias in reporting "good/bad" foods may affect the accuracy of the outcome. In this study, the 24-hour recall data was collected by three interviewers, which may have introduced inter-rater variability in the data collected. Additionally, during dietary recall, if a manufacturer's nutritional label was not available, portion size was obtained using the Carbs and Cals textbook as a visual aid, which may have affected the estimation of portion size. When assessing eating behaviours, the use of multiple behavioural questionnaires may have introduced an element of participant fatigue that may have decreased the specificity of the responses given. The order of these questionnaires was administered randomly to all participants throughout the study to mitigate this risk. Future studies should use additional methods such as weighed food records, and laboratory test meals to measure food intake in patients with active CD and to confirm the caloric intake findings of the present study.

Biochemical, endoscopic and radiological objective measures of disease activity have been acquired as part of routine standard of care rather than as a specific screening process for

this study. For this reason, there was a variable lag between the dates of these assessments and recruitment to this study. None of these patients changed their maintenance therapy after these investigations and prior to recruitment within this study.

In conclusion, this study has highlighted the significantly higher prevalence of emotional eating and food monitoring behaviour in CD. Clinically these results imply that stronger psychological and firm dietetic education may be of benefit in CD. Nearly half of the IBD patients have never received dietetic advice and two-thirds feel they need more support[4]. Questioning patients on their attitudes and beliefs through counselling or psychotherapy may alter these behaviours. Firm dietetic advice for healthy eating should also be advocated. Additionally, combating underlying anxiety and depression in these patients may improve disordered eating traits. The UK IBD standards in 2013 highlighted the need for formal psychological support in IBD teams with only 24% of adult IBD services have defined access to a psychologist with an interest in IBD[34].

This study has provided new evidence regarding the complexity of disordered eating behaviour traits in active CD. A more objective understanding is needed regarding the fine balance between homeostatic and hedonic control of food intake in intestinal inflammation.

FUNDING

This work was supported by National Institute for Health Research (NIHR) Biomedical Research Centre in Gastrointestinal and Liver Diseases at Nottingham University Hospitals NHS Trust & The University of Nottingham, Queens Medical Centre Campus

ACKNOWLEDGEMENTS

We acknowledge the support from the National Institute of Health Research Nottingham Biomedical Research Centre in the support in conducting this study.

REFERENCES

1. Satherley R, Howard R, Higgs S. Disordered eating practices in gastrointestinal disorders. *Appetite* 2015;**84**:240-50.
2. Satherley R-M, Howard R, Higgs S. The prevalence and predictors of disordered eating in women with coeliac disease. *Appetite* 2016;**107**:260-7.
3. Prince A, Whelan K, Moosa A, Lomer MC, Reidlinger DP. Nutritional problems in inflammatory bowel disease: The patient perspective. *Journal of Crohn's & colitis* 2011;**5**:443-50.
4. Limdi JK, Aggarwal D, McLaughlin JT. Dietary practices and beliefs in patients with inflammatory bowel disease. *Inflammatory bowel diseases* 2016;**22**:164-70.
5. Zallot C, Quilliot D, Chevaux JB*, et al.* Dietary beliefs and behavior among inflammatory bowel disease patients. *Inflammatory bowel diseases* 2013;**19**:66-72.
6. Addolorato G, Capristo E, Stefanini GF, Gasbarrini G. Inflammatory bowel disease: A study of the association between anxiety and depression, physical morbidity, and nutritional status. *Scandinavian journal of gastroenterology* 1997;**32**:1013-21.
7. Lochs H, Dejong C, Hammarqvist F*, et al.* Espen guidelines on enteral nutrition: Gastroenterology. *Clinical nutrition (Edinburgh, Scotland)* 2006;**25**:260-74.
8. Hart AL, Lomer M, Verjee A*, et al.* What are the top 10 research questions in the treatment of inflammatory bowel disease? A priority setting partnership with the james lind alliance. *Journal of Crohn's & colitis* 2017;**11**:204-11.
9. Berthoud H-R. Metabolic and hedonic drives in the neural control of appetite: Who's the boss? *Current Opinion in Neurobiology* 2011;**21**:888-96.
10. Harvey RF, Bradshaw JM. A simple index of crohn's-disease activity. *Lancet* 1980;**1**:514.
11. Moshfegh AJ, Rhodes DG, Baer DJ*, et al.* The us department of agriculture automated multiple-pass method reduces bias in the collection of energy intakes. *The American journal of clinical nutrition* 2008;**88**:324-32.
12. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research* 1985;**29**:71-83.
13. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. *Addictive behaviors* 1982;**7**:47-55.
14. Lowe MR, Butryn ML, Didie ER*, et al.* The power of food scale. A new measure of the psychological influence of the food environment. *Appetite* 2009;**53**:114-8.
15. van Strien T, Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. The dutch eating behavior questionnaire (debq) for assessment of restrained, emotional and external eating behavior. *International Journal of Eating Disorders* 1986;**5**:295-315.
16. Dalton M, Finlayson G, Hill A, Blundell J. Preliminary validation and principal components analysis of the control of eating questionnaire (coeq) for the experience of food craving. *European journal of clinical nutrition* 2015;**69**:1313-7.
17. Dalton M, Finlayson G, Walsh B*, et al.* Early improvement in food cravings are associated with long-term weight loss success in a large clinical sample. *Int J Obes* 2017;**41**:1232-6.
18. Dalton M, Blundell J, Finlayson GS. Examination of food reward and energy intake under laboratory and free-living conditions in a trait binge eating subtype of obesity. *Frontiers in psychology* 2013;**4**:757.
19. Cheyette C BY. *Carbs & cals: Count your carbs & calories with over 1,700 food & drink photos*: Chello Publishing Limited; 2013.
20. Cappelleri JC, Bushmakin AG, Gerber RA*, et al.* Evaluating the power of food scale in obese subjects and a general sample of individuals: Development and measurement properties. *International journal of obesity (2005)* 2009;**33**:913-22.

21. Marcus MD, Wing RR, Hopkins J. Obese binge eaters: Affect, cognitions, and response to behavioural weight control. *Journal of consulting and clinical psychology* 1988;**56**:433-9.
22. Freitas SR, Lopes CS, Appolinario JC, Coutinho W. The assessment of binge eating disorder in obese women: A comparison of the binge eating scale with the structured clinical interview for the dsm-iv. *Eating behaviors* 2006;**7**:282-9.
23. Bryant EJ, King NA, Blundell JE. Disinhibition: Its effects on appetite and weight regulation. *Obesity reviews : an official journal of the International Association for the Study of Obesity* 2008;**9**:409-19.
24. Aghdassi E., Wendland B.E., Stapleton M., Raman M., Allard J.P. Adequacy of Nutritional Intake in a Canadian Population of Patients with Crohn's Disease. *Journal of the American Dietetic Association* 2007. 107 (9) (pp 1575-1580), 2007.
25. Filippi J, Al-Jaouni R, Wiroth JB, Hebuterne X, Schneider SM. Nutritional deficiencies in patients with crohn's disease in remission. *Inflammatory bowel diseases* 2006;**12**:185-91.
26 Chen T.-C., Cruz G., Sellin J., Hou J. Food avoidance and use of dietary supplements among patients with inflammatory bowel disease. *American Journal of Gastroenterology.*2014 Conference: 79th Annual Scientific Meeting of the American College of Gastroenterology Philadelphia, PA United States. Conference Start: 20141017 Conference End: 20141022. Conference Publication: (var.pagings). 109 (pp S507)
27. Peat CM, Huang L, Thornton LM*, et al.* Binge eating, body mass index, and gastrointestinal symptoms. *Journal of Psychosomatic Research* 2013;**75**:456-61.
28. Arigo D, Anskis AM, Smyth JM. Psychiatric comorbidities in women with celiac disease. *Chronic illness* 2012;**8**:45-55.
29. Pariente B, Mary JY, Danese S*, et al.* Development of the lemann index to assess digestive tract damage in patients with crohn's disease. *Gastroenterology* 2015;**148**:52-63.e3.
30. Tang TN, Toner BB, Stuckless N*, et al.* Features of eating disorders in patients with irritable bowel syndrome. *Journal of psychosomatic research* 1998;**45**:171-8.
31. van Langenberg DR, Della Gatta P, Hill B*, et al.* Delving into disability in crohn's disease: Dysregulation of molecular pathways may explain skeletal muscle loss in crohn's disease. *Journal of Crohn's & colitis* 2014;**8**:626-34.
32. van Langenberg DR, Papandony MC, Gibson PR. Sleep and physical activity measured by accelerometry in crohn's disease. *Alimentary pharmacology & therapeutics* 2015;**41**:991-1004.
33. Vogelaar L, van den Berg-Emons R, Bussmann H*, et al.* Physical fitness and physical activity in fatigued and non-fatigued inflammatory bowel disease patients. *Scandinavian journal of gastroenterology* 2015;**50**:1357-67.
34. group Is. Ibd standards. http://www.ibdstandards.or.uk, 2013.

	SEX	AGE	BMI	MONTREAL	HBI	CRP	FCP	MRI	COLONOSCOPY
P01	F	48	24.9	A1L3B2	-	-	-	-	Post op recurrence Rutgeerts i3
P02	M	22	21.8	A2L1B3	-	-	-	-	Post op recurrence Rutgeerts i3
P03	M	51	21.4	A2L1B2	-	-	316	multifocal active small bowel disease	
P04	F	23	26.7	A2L3B1	5	-	-	-	Diffuse punched out ulcerations in terminal ileum
P05	M	30	25.5	A2L3B3	-	-	-	-	Colonoscopy - Rutgeerts i2
P06	M	25	26.2	A2L3B1	-	-	1763	-	-
P07	M	23	20	A2L3B3	-	-	-	30cm of TI disease with an enter-enteric fistula	-
P08	F	37	24.3	A2L1B2	9	-	-	Terminal ileitis	-
P10	F	23	23.1	A2L1B1	5	-	-	-	Diffuse punched out ulcerations in terminal ileum
P11	M	35	33.7	A2L3B1p	-	52	-	-	rectosigmoid inflammation with a perianal fistula
P13	F	29	36	A2L1B1	7	-	-	6cm terminal ileum inflammatory disease	-
P14	M	32	29.6	A2L3B3p	-	-	-	pancolonic inflammatory disease with distal sparing. Has a desc colon stricture. Distal 3cm TI inflamed	-
P15	M	57	18.6	A3L1B3	10	-	-	mixed inflammatory and stricturing disease in the ileum	-
P16	F	33	24.9	A2L2B1	-	-	449	-	severe colonic disease with puynched out ulcers
P17	F	40	27.6	A3L1B3	-	-	-	30cm of terminal ileal inflammatory disease	-
P19	M	49	25.7	A3L3B1	-	-	-	15cm of terminal ileal inflammatory disease	-
P20	M	33	22.5	A2L3L4B2	-	-	1800	extensive jejunal disease	-
P23	M	20	19.37	A2L3B3	7	-	-	-	Post op recurrence Rutgeerts i2
P24	M	28	18.6	A2L1B1	-	-	-	-	Diffuse punched out ulcerations in terminal ileum
P25	M	23	23.4	A2L3B1p	-	-	-	Diffuse terminal ileal inflammatory disease	-
P26	M	38	30.6	A2L3B2	-	-	785	-	-
P27	F	35			-	-	1226		
P28	F	22			-	38			Ruterts i2

P29	M	20	-	-	1027		
P30	F	68	-	224	-	extensive transverse colonic disease with fistulisation	
P31	M	31	-	-	607	chronic disease	
P32	F	28	-	-	319		
P33	F	24	-	-	-		mild patchy colitis with loss of vascular pattern, erythema in R colon.
P34	M	25	-	-	1800	Thickening of the terminal ilium	
P35	M	18	8	-	-		

SUPPLEMENTARY TABLE: CD PARTICIPANT DEMOGRAPHIC STUDY INCLUSION CRITERIA