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

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**Short Title**

Eating Behaviour In Crohn's disease: EBIC study

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## 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 matched healthy volunteers (HV) were recruited. 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

30 CD (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. Mean faecal calprotectin was 1032.5±176µg/g,C-reactive protein 83.8±47.1mg/L and HBI 4.8±1. There were no significant differences in calorific intake between groups. Protein intake was lower in the CD cohort (p=0.03). Hospital Anxiety and Depression score was higher (p=0.01) and CoEQ-Positive Mood (p=0.001) lower in CD. CD were characterised by higher BES (p=0.01) and lower CoEQ Craving Control (p=0.027) with greater craving for Sweet (p=0.043), Savoury (p=0.021) foods. PFS food present (p=0.005), DEBQ Emotional (p=<0.001) and External Eating (p=0.022) were significantly higher than HV.

Conclusions

Reduced protein consumption and more prevalent disordered eating behaviour traits were present in CD. Greater binge eating and decreased control of cravings may be attributed to lower mood and higher anxiety observed. Patients may benefit from stronger psychological support with firm dietetic advice for healthy eating.

**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 [3,4]. 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 [5]. Abnormal eating patterns have been described in IBD with qualitative studies unselectively describing eating behaviour irrespective of disease activity [6,7]. Approximately three-fourths of patients with IBD describe a decline in appetite when the disease is active [6] with up to 37% of CD patients showing abnormal eating patterns [8]. Malnutrition is more prevalent in CD than ulcerative colitis with up to 75% of hospitalised patients being malnourished with 50% in negative nitrogen balance [9]. 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 [10]. 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 [11]. 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 January 2018 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[12] (HBI) score recorded at inclusion. Potential participants with 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. 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 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[13] 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) [14]; the Binge Eating Scale [15]; the Power of Food Scale [16]; the Dutch Eating Behaviour Questionnaire [17]; and the Control of Eating Questionnaire [18,19].

**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 [13,20] 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 [21]. 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) [14-18].

<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 [22]. 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.

The Binge Eating Scale (BES)

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 [15,23,24].

The Control of Eating Questionnaire (CoEQ)

The CoEQ is a 21-item questionnaire designed to assess the severity and type of food cravings experienced over the previous seven days [18]. 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 [14]. 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 [25]. 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 [17].

**Statistical Analysis**

The sample size was based on published data [26] 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 participants had objective evidence of active disease with an elevated C-reactive protein (83.8±47.1mg/L), or faecal calprotectin (1032.5±176µg/g) or as assessed by colonoscopy or MR enterography or both (see supplementary table). These objective investigations have been undertaken as part of the participants standard of care within a mean of 52.9±14.1 days of recruitment. Mean HBI score was 4.8±1. None of the participants had any change in management prior to recruitment and data collection. Upon recruitment, 10 participants (33.3%) were being prescribed immunosuppressant therapy, 6 (20%) anti-TNF therapy and 7 (23.3%) CD participants a combination of anti-TNF therapy and immunosuppressant therapy. Eleven participants (36%) had a history of CD-related intestinal surgery with a mean of 0.4±0.1 CD-related operations per patient. Mean disease duration prior to recruitment was 8.1±1.5 years.

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 and HV cohorts 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, 70.3g±6.1; HV, 92.6g±7.8p=0.03). There was no significant difference in the consumption of all other macronutrients.

In a sub-analysis of this dataset aimed at investigating difference by gender, the 24hr calorie intake of male CD participants was not significantly different to male HV participants. In female participants, 24-hour calorie intake was significantly reduced in the CD cohort compared with HV participants (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), sugar (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) were significantly less than in HV participants.

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 | 2187.0± 193.7 | 1519.3± 136.5 | 2054.3± 110.7 | 2065.0± 167.0 | 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 | 112.0± 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 participants had significantly higher scores on the Hospital Anxiety and Depression scale compared to HV participants (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=<0.001) subscales.

Both male (CD 13.5±2.1; HV, 4.3±1; p=0.001) and female (CD, 15.9±2.9; HV, 8.6±1.6; p=0.04) CD participants showed significant difference in HADS when compared with HV participants. Male CD participants scored significantly higher than HV participants in both

anxiety (CD 7.9±1.2; HV, 2.9±0.7; p=0.002) and depression (CD 5.5±1.2; HV, 1.3±0.4; p=0.005) subscales. Female participants however were only significantly different in the depression subscale (CD 6.5±1.5; HV, 2.5±0.6; p=0.02).

**Eating Behaviour traits**

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

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

CD participants 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 participants 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 participants. 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 participants.

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 | NS |
| PFS: Available | 12.1±1.2 | 10.5±0.9 | NS |
| PFS: Present | 11.7±0.7 | 9.0±0.6 | 0.005 |
| PFS: tasted | 11.7±0.8 | 11.7±0.8 | NS |
| 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 | NS |
| TFEQ: D | 6.1±0.8 | 4.5±0.6 | NS |

| | | | |
|---|---|---|---|
| TFEQ: H | 5.5±0.8 | 4.0±0.5 | NS |
| DEBQ: R | 23.7±2.7 | 21.6±1.9 | NS |
| 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 participants showed significant difference in 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=0.005) and DEBQ: Emotional (CD, 31.4±4.2; HV, 18.9±1.9; p=0.02) when compared with male HV participants.

Female CD participants showed significant difference in 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 participants.

## 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 participants 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 participants with objective evidence of intestinal inflammation and age-, BMI- and gender-matched HV participants. Analysing the data further by gender reveals that a significant decrease in calorie intake is observed in female rather than male CD participants 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 in previous studies that have showed no difference in energy intake in CD patients with both active and inactive disease [27,28]. 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 [1,2].

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 [8]. In the present study, it was demonstrated that CD participants scored significantly higher on measures of binge eating and hedonic responsiveness compared to HV participants. 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 participants 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. 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 [6], 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 [29] 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 participants 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 (data not shown). 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 [30]. For example, it is possible that psychological distress may serve as both a cause and a consequence of disordered eating behaviours [3]. 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 [31]. 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 [7] 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 our entire 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 [32].

When analysed by gender, female CD participants consumed significantly less calories than female HV participants 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 participants. In female CD participants, significantly higher PFS: present and DEBQ: Emotional with lower CoEQ: mood when compared with female HV participants might imply that female CD participants may be predisposed to emotional eating. These results may suggest that female CD participants have similar level of 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. 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 a prospective study aiming to compare calorific intake and the eating behaviour of CD patients with active disease to matched healthy volunteers. The BMI of the recruited cohort was BMI:24.9±0.8 in CD and 24.7±0.5 in HV participants. These values are at the upper limit of what the World Health Organisation considers as

normal weight. Nevertheless, these BMIs are representative of present world-wide trends making our cohorts more representative [33 34]. The sample size despite being relatively small was appropriately powered based on the group's previous pilot data [26]. Daily activity level is an important confounder that was not routinely measured to try and minimise participant research burden. Physical inactivity has already been shown in CD [35,36] and has been significantly correlated to disease activity but is still prevalent in remission [37]. 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. The effect of these variables on eating behaviour should be investigated in downstream studies. 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 [20]. 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 [6]. 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 [38].

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.

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645

| | SEX | AGE (YR.) | BMI ($KG/M^2$) | MONTREAL | DIS. DUR. (YR.) | MED | HBI | CRP (MG/L) | FCP (µG/G) | MRI | COLONOSCOPY |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **P01** | F | 48 | 24.9 | A1L3B2 | 41 | Nil | 4 | - | - | - | Post op recurrence Rutgeerts i3 |
| **P02** | M | 22 | 21.8 | A2L1B3 | 3 | AZA | 2 | - | - | - | Post op recurrence Rutgeerts i3 |
| **P03** | M | 51 | 21.4 | A2L1B2 | 18 | HUM, MTX | 1 | - | 316 | multifocal active small bowel disease | |
| **P04** | F | 23 | 26.7 | A2L3B1 | 4 | AZA | 5 | - | - | - | Diffuse punched out ulcerations in terminal ileum |
| **P05** | M | 30 | 25.5 | A2L3B3 | 10 | Nil | 9 | - | - | - | Colonoscopy - Rutgeerts i2 |
| **P06** | M | 25 | 26.2 | A2L3B1 | 6 | HUM | 2 | - | 1763 | - | - |
| **P07** | M | 23 | 20 | A2L3B3 | 1 | HUM | 11 | - | - | 30cm of TI disease with an enter-enteric fistula | - |
| **P08** | F | 37 | 24.3 | A2L1B2 | 14 | Nil | 9 | - | - | Terminal ileitis | - |
| **P10** | F | 23 | 23.1 | A2L1B1 | 1 | MP | 5 | - | - | - | Diffuse punched out ulcerations in terminal ileum |
| **P11** | M | 35 | 33.7 | A2L3B1p | 2 | HUM | 9 | 52 | - | - | rectosigmoid inflammation with a perianal fistula |
| **P13** | F | 29 | 36 | A2L1B1 | 10 | Nil | 7 | - | - | 6cm terminal ileum inflammatory disease | - |
| **P14** | M | 32 | 29.6 | A2L3B3p | 14 | HUM, AZA,P | 5 | - | - | pancolonic inflammatory disease with distal sparing. Has a desc colon stricture. Distal 3cm TI inflamed | - |
| **P15** | M | 57 | 18.6 | A3L1B3 | 15 | AZA,P | 10 | - | - | mixed inflammatory and stricturing disease in the ileum | - |
| **P16** | F | 33 | 24.9 | A2L2B1 | 13 | INF, AZA | 6 | - | 449 | - | severe colonic disease with puynched out ulcers |
| **P17** | F | 40 | 27.6 | A3L1B3 | 0 | Nil | 5 | - | - | 30cm of terminal ileal inflammatory disease | - |
| **P19** | M | 49 | 25.7 | A3L3B1 | 1 | P | 2 | - | - | 15cm of terminal ileal inflammatory disease | - |
| **P20** | M | 33 | 22.5 | A2L3L4B2 | 8 | INF, AZA | 0 | - | 1800 | extensive jejunal disease | - |
| **P23** | M | 20 | 19.37 | A2L3B3 | 4 | MP | 7 | - | - | - | Post op recurrence Rutgeerts i2 |
| **P24** | M | 28 | 18.6 | A2L1B1 | 1 | Nil | 3 | - | - | - | Diffuse punched out ulcerations in terminal ileum |
| **P25** | M | 23 | 23.4 | A2L3B1p | 1 | AZA | 1 | - | - | Diffuse terminal ileal inflammatory disease | - |
| **P26** | M | 38 | 30.6 | A2L3B2 | 11 | AD, AZA | 8 | - | 785 | - | - |
| **P27** | F | 35 | 30.3 | A2L2B2 | 13 | HUM | 8 | - | 1226 | | |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **P28** | F | 22 | 23 | A2L1 B2 | 7 | HUM, AZA | 0 | 38 | | | Ruterts i2 |
| **P29** | M | 20 | 19.3 | A2L2B1 | 3 | AZA | 4 | - | 1027 | | |
| **P30** | F | 68 | 21.4 | A3L2B2/B3 | 1 | MTX | 8 | 224 | - | extensive transverse colonic disease with fistulisation | |
| **P31** | M | 31 | 25.5 | A2L1B1 | 9 | AD | 1 | - | 607 | chronic disease | |
| **P32** | F | 28 | 30 | A2L2B1 | 4 | INF, MP | 1 | - | 319 | | |
| **P33** | F | 24 | 22 | A2L2 B1 | 12 | HUM | 1 | - | - | | mild patchy colitis with loss of vascular pattern, erythema in R colon. |
| **P34** | M | 25 | 29.7 | A2L1B1 | 9 | MP | 2 | - | 1800 | Thickening of the terminal ilium | |
| **P35** | M | 18 | 22 | A2L2B1 | 6 | MP | 8 | - | 1266 | | |

Supplementary Table: CD Participant Demographic

**AD=ADALIMUMAB, AZA=AZATHIOPRINE, HUM=HUMIRA, INF=INFLIXIMAB, MP=MERCAPTOPURINE, MTX=METHOTREXATE, P=PENTASA**