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

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25 **Short Title**

26 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

119 Reduced protein consumption and more prevalent disordered eating behaviour traits were

120 present in CD. Greater binge eating and decreased control of cravings may be attributed to

121 lower mood and higher anxiety observed. Patients may benefit from stronger psychological

122 support with firm dietetic advice for healthy eating.

123 **Keywords**

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

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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

168   drive of appetite regulation [11]. The cross-link between eating behaviour and active CD is poorly

169   understood. Disordered eating might be associated with a change in the homeostatic and

170   hedonic balance. The aim of this study is to examine free-living calorie and macronutrient

171   intake in patients with active CD compared to healthy volunteers and to determine the

172   prevalence and type of eating behaviour traits and disordered eating in CD patients with active

173   disease.

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190 **METHODOLOGY**

191 **Basic protocol and patient recruitment**

192 This was an open label, qualitative questionnaire-based study with a matched-pair design.

193 The study was conducted between July 2015 and January 2018 at the National Institute of

194 Health Research (NIHR) Nottingham Digestive Diseases Biomedical Research Centre (NDD

195 BRC) at the Queens Medical Centre Campus, Nottingham, UK. Participants were recruited

196 from The Inflammatory Bowel Disease Clinic, via the study flyer and social media. CD patients

197 (aged 16-75yrs) with active disease were recruited as well as age, BMI and gender-matched

198 healthy volunteers. Healthy volunteers (HV) were recruited form an existing participant

199 database in the Nottingham BRC and from the local healthy populations of Nottingham

200 University Hospitals and the University of Nottingham. This study was advertised through

201 study fliers and social media.

202 Disease activity was defined through objective markers of inflammation: faecal calprotectin of

203 >250µg/g or CRP of >5g/dl or through recent ileocolonoscopy, CT or MR enterography

204 showing active inflammatory and uncomplicated disease (not of a stricturing or penetrating

205 behaviour). CD clinical activity was measured with a Harvey Bradshaw Index[12] (HBI) score

206 recorded at inclusion. Potential participants with recent corticosteroid use (in the last 3

207 months), pregnancy or breast-feeding and patients with significant co-morbidities were

208 excluded from the study. Stable doses of immunosuppressive agents or anti-TNF agents were

209 permitted.

210 All CD patients and healthy volunteers gave their informed consent prior to recruitment.

211 Participants completed a single, spontaneously administered 24hr dietary recall either face-

212 to-face at the NDD BRC or by telephone, the Hospital Anxiety and Depression scale (HADS)

213 and psychometric eating behaviour questionnaires within the study period.

214 **Outcomes**

215   The primary outcome of this study was to compare total 24 hr calorie intake as measured by

216   a single face-to-face or telephone-administered 24-hour dietary recall[13] between CD with

217   active disease and age-, BMI- and gender-matched HV. Calories consumed were calculated

218   for the recall based on manufacturers' labels and the nutrition analysis tool Nutritics (Nutritics

219   v4.312 Academic Edition, Ireland). Dietary recall did not include caloric intake from weekends

220   or holidays but only days Monday to Thursday. The secondary endpoint for this study was to

221   measure eating behaviour traits through psychometric scales: Three Factor Eating

222   Questionnaire (TFEQ) [14]; the Binge Eating Scale [15]; the Power of Food Scale [16]; the Dutch

223   Eating Behaviour Questionnaire [17]; and the Control of Eating Questionnaire [18,19].

24-h dietary recall

225   The Automated Multiple-Pass Method (AMPM) was utilised to perform the single

226   spontaneously administered 24hr dietary recall. This five-step questionnaire can accurately

227   assess dietary consumption and may be administered face-to-face or by telephone [13,20] RW,

228   AN and GT conducted all interviews. A copy of the dietary assessment textbook Carbs and

229   Cals was provided to each participant to facilitate the dietary recall [21]. This book contains over

230   1700 food and drink photographs and was primarily used to assist in identifying the appropriate

231   food type and portion size consumed. Diet logs were analysed using Nutritics dietary analysis

232   software (Nutritics v4.312 Academic Edition, Ireland).

Eating Behaviour traits

234   Eating behaviour traits were measured through five validated self-report questionnaires; the

235   Power of Food Scale (PFS); the Binge Eating Scale (BES); the Control of Eating Questionnaire

236   (COEQ); Three Factor Eating Questionnaire (TFEQ) and the Dutch Eating Behaviour

237   Questionnaire (DEBQ) [14-18].

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

240 The PFS is a 15-item questionnaire reflecting the psychological influence of the food

241 environment. It measures appetite for, rather than consumption of palatable foods and may

242 be a useful measure of the hedonic impact of food environments replete with highly palatable

243 foods [22]. Items are grouped into three domains according to food proximity; food available but

244 not physically present, food present but not tasted and food tasted but not consumed.

245 The Binge Eating Scale (BES)

246 The BES is a 16-item questionnaire that assesses the severity of binge eating tendencies.

247 Eight questions describe the behavioural manifestations of binge eating behaviour and eight

248 describe the feelings and cognitions associated with binge eating. Scores are summed to

249 produce a total score ranging from 0 to 46. Cut off points have previously been reported

250 denoting mild (≤17), moderate (18–26), and severe (≥27) binge eating behaviour [15,23,24].

251 The Control of Eating Questionnaire (CoEQ)

252 The CoEQ is a 21-item questionnaire designed to assess the severity and type of food

253 cravings experienced over the previous seven days [18]. The CoEQ has four subscales; Craving

254 Control, Craving for Savoury, Craving for Sweet and Positive Mood. Items on the CoEQ are

255 assessed by 100-mm visual analogue scales (VAS) with items relating to each subscale being

256 averaged to create a final score.

257 Three Factor Eating Questionnaire (TFEQ)

258 The TFEQ contains 51-items and measures three dimensions of human eating behaviour;

259 Cognitive Restraint of Eating, Disinhibition and Hunger [14]. Restraint refers to concern over

260 weight control and strategies which are adopted to achieve this. Disinhibition reflects a

261 tendency towards over-eating and eating opportunistically in an obesogenic environment.

262 Hunger is concerned with the extent to which hunger feelings are perceived and the extent to

263 which such feelings evoke food intake [25]. Each item scores either 0 or 1 point. The minimum

264 score for factors I, II and III is therefore 0, with the possible maximum scores being 21, 16

265 and14 respectively.

266   The Dutch Eating Behaviour Questionnaire (DEBQ)

267   The 33-item DEBQ assesses different eating styles that may contribute to weight gain;

268   emotional eating, external eating, and restraint. 'Emotional eating' occurs in response to

269   emotional arousal states such as fear anger or anxiety, 'external eating' in response to external

270   food cues such as sight and smell of food and 'restraint eating' is overeating after a period of

271   slimming when the cognitive resolve to diet is abandoned [17].

272   **Statistical Analysis**

273   The sample size was based on published data [26] where the 24hr self-reported calorie intake

274   in CD was 1978.7±169.7Kcal and that in HV was 1854.4 ±129.5Kcal. Assuming α of 0.05,

275   power of 80% and using 2-sided test, 30 participants in each group were needed to show a

276   significant difference in the primary outcome.

277   Data were analysed using SPSS version 20 for Windows. The parametric or non-parametric

278   nature of the data was determined by a normality test. Data is presented as mean ± standard

279   error of the mean (SEM). Continuous data was compared using paired t-test while categorical

280   data was compared with Chi-Squared test. Total 24hr Kcal intake, macronutrient intake

281   together with outcome data from the individual questionnaires administered to all participants

282   were compared between the groups. An exploratory sub-analysis was undertaken comparing

283   differences between gender. P values <0.05 were deemed significant.

284   **Ethical approval**

285   This study received research ethics committee approval from National Research Ethics

286   Service (NRES) Committee East Midlands (REC reference 15/EM/0142 as of the 27th April

287   2015). The protocol was registered with clinical trials.gov (NTC02379117).

288   **RESULTS**

289   **Demographic data**

290   Thirty CD patients (18M:12F, Age:32.3±2.19, BMI:24.9±0.8) and 31 matched HV (19M:12F,

291   Age:32.8±2.0, BMI:24.7±0.5) were recruited to this matched pairs cross-sectional study (see

292   Table 1). There were no significant differences in gender ratio, mean age and mean BMI

293   between the CD and HV. CD participants had objective evidence of active disease with an

294   elevated C-reactive protein (83.8±47.1mg/L), or faecal calprotectin (1032.5±176µg/g) or as

295   assessed by colonoscopy or MR enterography or both (see supplementary table). These

296   objective investigations have been undertaken as part of the participants standard of care

297   within a mean of 52.9±14.1 days of recruitment. Mean HBI score was 4.8±1. None of the

298   participants had any change in management prior to recruitment and data collection. Upon

299   recruitment, 10 participants (33.3%) were being prescribed immunosuppressant therapy, 6

300   (20%) anti-TNF therapy and 7 (23.3%) CD participants a combination of anti-TNF therapy and

301   immunosuppressant therapy. Eleven participants (36%) had a history of CD-related intestinal

302   surgery with a mean of 0.4±0.1 CD-related operations per patient. Mean disease duration prior

303   to recruitment was 8.1±1.5 years.

304   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

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306   **24-hour calorie intake**

307   The total self-reported 24-hour calorie and macronutrient intake for the CD and HV cohorts

308   are shown in Table 2. There were no significant differences observed in total energy intake

309   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

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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).

334 **Eating Behaviour traits**

335 Table 3 shows the outcomes from the psychometric eating behaviour questionnaires for CD

336 and HV. CD participants scored higher on BES compared to HV participants (CD, 10.9±1.9;

337 HV, 5.2±1.0; p=0.01) and a greater proportion of CD participants (29%) scored above the

338 clinical cut-off criteria for moderate levels of binge eating (>17 BES) compared to HV (3.3%)

339 $[\chi^2 (1) = 7.0, p=0.008]$.

340 CD participants reported lower levels of CoEQ Craving Control (CD, 56.16±3.5; HV, 66.4±2.9;

341 p=0.027) and greater craving for sweet (CD, 48.9±4.4; HV, 37.3±3.5; p=0.043) and savoury

342 (CD, 48.9±3.5; HV, 38.3±2.7; p=0.021) foods compared to HV participants. CD participants

343 scored significantly lower on the CoEQ Positive Mood subscale (CD, 50.8±3.3; HV, 64.8±2.5;

344 p=0.001).

345 CD participants had higher scores on the PFS food present (CD, 11.7±0.7; HV, 9.0±0.6;

346 p=0.005) subscale. No significant difference was seen however for overall PFS score or food

347 available or tasted subscales.

348 In addition, CD participants scored higher on the DEBQ Emotional Eating (CD, 36.4±3.7; HV,

349 20.0±1.7; p=<0.001) and External Eating (CD, 30.8±1.9; HV, 25.2±1.2; p=0.022) subscales

350 compared to HV participants. However, there was no difference in restraint assessed by either

351 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)

352 between CD and HV participants.

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354 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

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356 When analysed by gender, male CD participants showed significant difference in BES (CD,

357 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:

358 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;

359 p=0.005) and DEBQ: Emotional (CD, 31.4±4.2; HV, 18.9±1.9; p=0.02) when compared with

360 male HV participants.

361 Female CD participants showed significant difference in PFS: Present (CD, 12.8±1; HV, 9.6±1;

362 p=0.04), CoEQ: Mood (CD, 44.1±5.2; HV, 64.1±4.1; p=0.006) and DEBQ: Emotional (CD,

363 43.8±6; HV, 22±3.4; p=0.01) when compared with female HV participants.

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370 **DISCUSSION**

371 A poor nutritional status has always been associated with CD but a detailed analysis of eating

372 behaviour in this cohort compared to matched HV has never been undertaken. The primary

373   aim of this study was to compare the total self-reported 24 hr calorie intake in CD with active

374   disease and HV. The main secondary aim was to examine whether CD participants with active

375   disease had a greater prevalence of disordered eating patterns compared to HV. We found

376   no substantial difference in the 24-hour self-reported calorie intake between CD participants

377   with objective evidence of intestinal inflammation and age-, BMI- and gender-matched HV

378   participants. Analysing the data further by gender reveals that a significant decrease in calorie

379   intake is observed in female rather than male CD participants with this reduction in food intake

380   consisting mainly of a reduction in carbohydrates in females and protein in males. This finding

381   is novel and contrasts with observations made in previous studies that have showed no

382   difference in energy intake in CD patients with both active and inactive disease [27,28]. These

383   differences in food intake may be explained by the two-path theoretical model; with CD

384   patients experiencing high levels of anxiety to food intake, thus restricting food variety to

385   minimise symptom aversion [1,2].

386   An increased prevalence of disordered eating behaviour traits was observed in CD with a

387   greater prevalence of binge eating, food craving, lower mood and higher anxiety states

388   observed in this group. Patients with gastrointestinal disorders are reported to suffer from

389   disordered eating behaviour with more than a third of CD patients thought to be affected [8]. In

390   the present study, it was demonstrated that CD participants scored significantly higher on

391   measures of binge eating and hedonic responsiveness compared to HV participants. Binge

392   eating traits were more prevalent as revealed by a significantly higher BES together with

393   significantly stronger cravings with less ability of self-control. The CoEQ showed that CD

394   participants had less control of their cravings, with significantly greater cravings for both sweet

395   and savoury foods.

396   Significantly higher scores on the hedonic eating traits (i.e. BES, PFS, DEBQ-External) in CD

397   may be associated with increased food monitoring behaviour that occurs in patients with

398   dietary-controlled conditions. These findings are consistent with previous research that have

399   demonstrated a higher level of disordered eating patterns in individuals with gastrointestinal

400 disorders [1,2]. In a questionnaire-based study in 400 consecutive IBD patients in the UK [6],

401 approximately half of the patients felt that diet was the initiating factor in IBD and subsequent

402 relapses. The majority of patients' symptoms were triggered by food with two-thirds of the

403 patients depriving themselves of their favourite food to achieve symptom control. A case-

404 control study of 104 patients with an established diagnosis of IBD [29] concluded that avoidance

405 of meat, nuts, fruit and vegetables are more common among patients with IBD than healthy

406 controls. This corresponds with the findings of this study where the consumption of protein

407 was significantly reduced overall and carbohydrate, sugar and fibre intake were reduced in

408 females.

409 The current study also demonstrated that CD participants had lower levels of positive mood

410 as measured by the CoEQ and higher scores on the HAD scale. Greater levels of

411 psychological distress have been linked to increased binge eating prevalence and in the

412 current study we found that scores on the BES were negatively associated with positive mood

413 (data not shown). Similarly, we found a higher prevalence of emotional eating in the DEBQ.

414 These findings have important implications for the role of mood and psychological distress in

415 the aetiology of gastrointestinal disorders and their association with abnormal eating patterns

416 [30]. For example, it is possible that psychological distress may serve as both a cause and a

417 consequence of disordered eating behaviours [3]. Arigo et al suggested that fear and anxiety

418 surrounding gastrointestinal symptoms may lead to disordered eating practices of a restrictive

419 nature, as observed in this study [31]. This increased anxiety may link directly to the personal

420 attitudes and beliefs that patients hold about food. In a French survey of 244 IBD patients,

421 nearly half of the study patients reported that the disease had changed the pleasure of eating

422 [7] with only a quarter of the patients eating a normal diet when they relapse. Such a behaviour

423 influenced patients' social life in 25% of the cases. This might have a negative effect on mood

424 and depressive symptoms.

425 Disease activity has been quantified with objective markers of disease activity and intestinal

426 inflammation present in our entire recruited cohort. Clinical scores were quantified through

427 HBI. Gastrointestinal symptom severity may also play an important role in the development

428 of disordered eating patterns, with greater symptom severity correlating positively with the risk

429 of disordered eating [32].

430 When analysed by gender, female CD participants consumed significantly less calories than

431 female HV participants with reduced consumption of carbohydrate, sugar and fibre. This was

432 not observed in male participants. Male CD participants displayed greater hedonic

433 responsiveness with higher BES, lower CoEQ Control and TFEQ:Restraint compared with

434 male HV participants. In female CD participants, significantly higher PFS: present and DEBQ:

435 Emotional with lower CoEQ: mood when compared with female HV participants might imply

436 that female CD participants may be predisposed to emotional eating. These results may

437 suggest that female CD participants have similar level of self-control over dietary consumption

438 as HV. Consequently, females with CD may be less likely to binge eat during active disease,

439 being more likely to display inadequate calorie consumption as displayed by this study. Male

440 CD participants display greater hedonic responsiveness, with higher prevalence of binge

441 eating with the consequence of normalising calorie consumption. It is important to highlight

442 that this study was not powered to analyse the difference in eating behaviour by gender, so

443 such conclusions are hypothesis-generating.

444 We believe that for the first time, this study highlights in detail the important behavioural

445 differences that may be observed in patients with active CD. This study has some limitations

446 that need to be considered. This was a prospective study aiming to compare calorific intake

447 and the eating behaviour of CD patients with active disease to matched healthy volunteers.

448 The BMI of the recruited cohort was BMI:24.9±0.8 in CD and 24.7±0.5 in HV participants.

449 These values are at the upper limit of what the World Health Organisation considers as normal

450 weight. Nevertheless, these BMIs are representative of present world-wide trends making our

451 cohorts more representative [33] [34]. The sample size despite being relatively small was

452 appropriately powered based on the group's previous pilot data [26]. Daily activity level is an

453 important confounder that was not routinely measured to try and minimise participant research

454 burden. Physical inactivity has already been shown in CD [35,36] and has been significantly

455 correlated to disease activity but is still prevalent in remission [37]. Due to the small sample size,

456 we did not investigate the effect of disease burden surrogates: disease duration, concomitant

457 medication and surgical history in CD patients on eating behaviour. The effect of these

458 variables on eating behaviour should be investigated in downstream studies. Nevertheless,

459 the CD cohort recruited is representative of a CD cohort with moderate disease burden,

460 making our findings generalizable to world-wide healthcare systems.

461 The use of the AMPM as a single administered 24-hour recall is limited, and accuracy may

462 have been improved if this was performed on three consecutive days rather than one day.

463 However, this method has been used successfully in previous research [20]. The 24-hour recall

464 technique is also memory dependent and participants' potential bias in reporting "good/bad"

465 foods may affect the accuracy of the outcome. In this study, the 24-hour recall data was

466 collected by three interviewers, which may have introduced inter-rater variability in the data

467 collected. Additionally, during dietary recall, if a manufacturer's nutritional label was not

468 available, portion size was obtained using the Carbs and Cals textbook as a visual aid, which

469 may have affected the estimation of portion size. When assessing eating behaviours, the use

470 of multiple behavioural questionnaires may have introduced an element of participant fatigue

471 that may have decreased the specificity of the responses given. The order of these

472 questionnaires was administered randomly to all participants throughout the study to mitigate

473 this risk. Future studies should use additional methods such as weighed food records, and

474 laboratory test meals to measure food intake in patients with active CD and to confirm the

475 caloric intake findings of the present study.

476 Biochemical, endoscopic and radiological objective measures of disease activity have been

477 acquired as part of routine standard of care rather than as a specific screening process for

478 this study. For this reason, there was a variable lag between the dates of these assessments

479 and recruitment to this study. None of these patients changed their maintenance therapy after

480 these investigations and prior to recruitment within this study.

481   In conclusion, this study has highlighted the significantly higher prevalence of emotional eating

482   and food monitoring behaviour in CD. Clinically these results imply that stronger psychological

483   and firm dietetic education may be of benefit in CD. Nearly half of the IBD patients have never

484   received dietetic advice and two-thirds feel they need more support [6]. Questioning patients on

485   their attitudes and beliefs through counselling or psychotherapy may alter these behaviours.

486   Firm dietetic advice for healthy eating should also be advocated. Additionally, combating

487   underlying anxiety and depression in these patients may improve disordered eating traits. The

488   UK IBD standards in 2013 highlighted the need for formal psychological support in IBD teams

489   with only 24% of adult IBD services have defined access to a psychologist with an interest in

490   IBD [38].

491   This study has provided new evidence regarding the complexity of disordered eating

492   behaviour traits in active CD. A more objective understanding is needed regarding the fine

493   balance between homeostatic and hedonic control of food intake in intestinal inflammation.

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	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		

	Sex	Age	BMI	Montreal		Medication				Comments
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