

1 **Associations between Diet and Disease Activity in Ulcerative Colitis Patients using a**
2 **Novel Method of Data Analysis**

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20

20 **Abstract**

21 **Background**

22 The relapsing nature and varying geographical prevalence of ulcerative colitis (UC)
23 implicates environmental factors such as diet in its aetiology.

24

25 **Methods**

26 In order to determine which foods might be related to disease activity in UC a new
27 method of dietary analysis was developed and applied. Eighty-one UC patients were
28 recruited at all stages of the disease process. Following completion of a 7 d diet diary,
29 clinical assessment including a sigmoidoscopic examination (scale 0 (normal mucosa) to
30 6 (very active disease)) was conducted. Food weights for each person were adjusted
31 (divided) by the person's calorific intake for the week. Each food consumed was given a
32 food sigmoidoscopy score (FSS) calculated by summing the products of the (adjusted)
33 weight of food consumed and sigmoidoscopy score for each patient and occurrence of
34 food and dividing by the total (adjusted) weight of the food consumed by all 81 patients.
35 Thus, foods eaten in large quantities by patients with very active disease have high FSSs
36 and vice versa. Foods consumed by <10 people or weighing <1 kg for the whole group
37 were excluded, leaving 75 foods.

38

39 **Results**

40 High FSS foods were characterized by high levels of the anti-thiamin additive sulfite
41 (Mann-Whitney, $p < 0.001$), i.e. bitter, white wine, burgers, soft drinks from concentrates,
42 sausages, lager and red wine. Caffeine also has anti-thiamin properties and decaffeinated

43 coffee was associated with a better clinical state than the caffeine containing version.
44 Beneficial foods (average intake per week) included pork (210 g), breakfast cereals (200
45 g), lettuce (110 g), apples and pears (390 g), milk (1250 ml), melon (350 g), bananas (350
46 g), bacon (120 g), beef and beef products (500 g), tomatoes (240 g), soup (700 g), citrus
47 fruits (300 g), fish (290 g), yogurt (410 g), cheese (110 g), potatoes (710 g) and legumes
48 (120 g).

49

50 **Conclusions**

51 The dietary analysis method described provides a new tool for establishing relationships
52 between diet and disease and indicates a potentially therapeutic diet for UC.

53

54 Key words: ulcerative colitis, diet, sulfite, thiamin, resistant starch.

55

55 **Background**

56 Ulcerative colitis (UC) is a chronic, relapsing mucosal disorder that extends in
57 continuous fashion proximally from the rectum and is limited to the colon. The aetiology
58 of UC includes a genetic component possibly involving an abnormal cell-mediated
59 immune response to commensal enteric bacteria in the large intestine. The
60 relapse/remission pattern of the disorder and substrate driven nature of microbial
61 metabolism in the large bowel implicate environmental factors such as diet.

62 Apart from nutritional repletion, dietary measures do not play a role in the
63 management of UC. Nonetheless, attempts to link the cause of UC with specific foods
64 date back at least 50 years[1]. Many foods or food groups have been related to UC (table
65 4) [2-13] including sugar, eggs, soft drinks, fruit and vegetables, protein, carbohydrate
66 and fat. However none have been proven to be of significant benefit or to contribute to
67 the cause of UC. This may partly be because both the assessment of disease activity in
68 UC and dietary intake are difficult to measure, or because the actual dietary component
69 that is key to this relationship has not been measured.

70 It has been proposed that sulfide, produced in the large bowel from either amino
71 acid fermentation or sulphate reduction, may be a triggering factor in the inflammatory
72 process of UC [14-16]. Recently, in a prospective dietary study where foods rich in
73 sulfur compounds were quantitated, evidence that sulfur compounds may increase the
74 likelihood of subsequent relapse in UC was found[17].

75 The main source of inorganic sulfur, predominantly sulfate, in the diet are the S
76 (IV) family of additives; the sulfiting agents. Sulfites have been used as food
77 preservatives since the 17th century and are amongst the most widely accepted and

78 versatile of additives. Sulfiting agents, denoted by E220-228 in Europe and generally
79 recognized as safe (GRAS) substances in the USA, include sulfur dioxide, hydrogen
80 sulfites, sulfites and metabisulfites. Sulfiting agents are cheap, easy to use and extremely
81 effective at preventing microbial growth and reducing spoilage[18]. They serve as
82 antioxidants, inhibit enzymatic and non-enzymatic browning reactions and act as a
83 texture modifier in biscuit dough. Sulfites improve color extraction from, and
84 stabilization of grape must in winemaking and preserve lobsters and shrimps from
85 discoloration during iced storage.

86 However, there are some problems with sulfite use[19, 20]. In the early 1980s
87 ingestion or inhalation of sulfites was shown to cause bronchospasm in about 5 % of
88 asthmatics. Sulfite sensitivity can pose a particular threat in the workplace where
89 sulfiting agents are used, but may also occur with ingestion of sulfited foods such as
90 potato products and wine. In addition, skin sensitivity has been reported and there are
91 anti-nutritional effects, particularly the destruction of thiamin[21]. The mechanism
92 involves an initial nucleophilic substitution of thiamin by the sulfite ion. As a result of
93 this anti-nutritional effect the GRAS status for sulfites was reviewed in the USA and in
94 1986 the use of sulfites in fresh and frozen fruit and vegetables revoked and a declaration
95 on the label required[22, 23]. Earlier (in the USA) their use in meat had been prohibited,
96 because these foods are an important source of thiamin.

97 A study of diet and disease activity in UC using a 7 d dietary diary, a full
98 assessment of disease activity and a method of dietary data analysis that allows trends in
99 food consumption not apparent using customary dietary software was therefore
100 undertaken.

101

101 **Methods**

102 *Subjects.* Eighty-one UC patients were recruited and informed consent obtained. Ethical
103 permission was granted by Tayside Committee on Medical Ethics, Dundee, UK (ref
104 007/00). As it was important to have a range of disease activities present, recruitment
105 included patients at all stages of the disease. Patients were excluded if clinical
106 examination or histology indicated Crohn's disease or indeterminate colitis, if there was a
107 positive stool culture for pathogens or if the patient had antibiotic treatment within 3
108 months preceding the start of the study.

109 *Dietary Assessment.* All the UC patients were asked to complete a 7 d diet diary[24].
110 The diet diary used has been validated for use in the European Prospective Investigation
111 into Cancer study (EPIC). Following completion of the diet diary, subjects attended the
112 research clinic and a full clinical assessment (see below) was carried out. The time
113 interval between the first day of the diary and the clinical visit was on average 28 d. Thus
114 the dietary data is prospective.

115 7d diet diaries were coded and analyzed using Tinuviel, WISP v3.0 nutritional
116 analysis software (Warrington, UK). Due to the variation in the sulfiting protocols and
117 widespread use of sulfiting agents, current tables of food composition do not contain
118 inorganic sulfur values and cannot be used to quantify intake. Instead of quantitating the
119 intake of particular dietary components, foods and food groups were assessed in their
120 entirety using the method described in the dietary data analysis section (below).

121 *Clinical Assessment.* Clinical assessment included history, physical examination and
122 global clinical grading, plus full blood count, liver function tests and inflammatory
123 markers. Patients were examined by rigid sigmoidoscopy or flexisigmoidoscopy and

124 graded on a scale 0-6 (integers and half integers used) according to the macroscopic
125 appearances of the rectal mucosa at a distance 5-10 cm from the anal verge[25]. The
126 clinical assessment of disease activity was confirmed in each case by histological
127 examination, by a single histopathologist blinded to the clinical details, of a rectal biopsy
128 taken from the posterior rectal wall 5-10 cm from the anal verge[26]. A simple clinical
129 colitis score was assigned to patients on each visit following Walmsley's scoring
130 system[27], together with blood parameters of disease severity (Hb, plasma viscosity,
131 CRP, serum albumin).

132 *Dietary Data Analysis.* Patterns of dietary intake associated with disease activity became
133 apparent through the study of the dietary diaries, e.g. high intakes of sulphite containing
134 foods coupled with a modern processed, convenience diet was associated with a high
135 sigmoidoscopy score. Traditional dietary coding (WISP) did not show any such clear
136 associations between micro or macro nutrient intake and sigmoidoscopy score.
137 Traditional dietary analysis was therefore thought to be missing important patterns in
138 dietary data and a new method of dietary assessment was subsequently developed.

139 This new method used the following procedure. To calculate the association of a
140 particular food with clinical score, each food or food group consumed was given a food
141 sigmoidoscopy score (FSS) calculated by summing the products of food weight and
142 sigmoidoscopy score for each occurrence of the food or food group and dividing by the
143 total weight of the food or food group contained in all diaries. In order for each diary to
144 make equal contributions to the FSSs, the weight of each food was adjusted using the
145 calorific intake for each person. This procedure was carried out separately for every food
146 item recorded in the 7 d diet diaries but is explained below using the example of red

147 wine.

148

149 Red wine score = $(\sum v(i)s(i))/\sum v(i)$ for $i=1$ to 81 equation 1.

150 Where: -

151 i is the 7 d dietary diary number ($n=81$).

152 $v(i)$ is the volume (divided by calorific intake for patient (i) of red wine recorded in 7 d
153 dietary diary i .

154 $s(i)$ is the sigmoidoscopy score associated with 7 d dietary diary (i).

155

156 Thus foods eaten in large quantities by patients with high levels of disease activity will
157 have high scores and vice versa. The denominator in the above equation is the total
158 volume of the food in question from all diaries (corrected for calorific intakes) so the
159 food scores can be equated with the effect of a typical portion of the food in question on
160 the sigmoidoscopy scores of the patients. This procedure is repeated for every food item.
161 Foods or food groups were excluded from the analysis if 10 or fewer people consumed
162 them or if they made up less than 1 kg of the total intake of the entire population. The
163 decision as to where food group boundaries lay was made depending on the size of the
164 group and whether the differences between the foods were considered important for this
165 study.

166 *Statistics and Data Handling*. Dietary data was exported from WISP to Microsoft
167 EXCEL 98 (Macintosh version, 1998). A worksheet containing the core headings;
168 Patient ID, food description, weight and patient sigmoidoscopy score was completed.
169 The data was then sorted by food description and each food copied to a separate EXCEL

170 file. Equation 1 was then used to calculate food sigmoidoscopy scores for each food in a
171 manner similar to the example in table 2.

172 Correlation values for scatter plots were obtained using the linear regression
173 function in EXCEL. The equation $t=r \sqrt{(n-2)/(1-r^2)}$ combined with t tables provided
174 corresponding significance levels.

175

175 **Results**

176 Of the 81 patients recruited 43 were male and 38 female. The average age (range)
177 of the males and females were respectively 53 (26-78) y and 47 (19-74). The distribution
178 of sigmoidoscopy scores is shown in fig 1. One third of the patients had sigmoidoscopy
179 scores of 0, 0.5 or 1. The mean sigmoidoscopy score for all 81 patients was 2.09. The
180 correlation between the clinical activity indexes and sigmoidoscopy scores was $r^2=0.25$
181 (n=81).

182 Table 3 shows the foods and food groups with associated sigmoidoscopy scores
183 and average portion sizes. In total 75 foods (or food groups) were given FSS scores. The
184 higher the FSS value the greater the association with disease activity and vice versa. The
185 total weight of foods in all diaries was 1,681 kg. The average food sigmoidoscopy score
186 (i.e. a food sigmoidoscopy score calculated for the entire dietary intake data set was
187 2.127). Foods excluded from the FSS table (Table 3), by virtue of contributing <1 kg or
188 being consumed by <10 people, made up 8 % of the total weight of all foods and had a
189 score slightly lower (2.001) than that of an average food (2.127). Standard errors are not
190 quoted for the food scores as the data used to generate them (weight * sigmoidoscopy
191 score) was not normally distributed due to the number of sigmoidoscopy scores of 0.

192 The dietary diaries were assessed for completeness by comparing calorific intakes
193 with expected values for the sexes. Expected (calculated from dietary reference tables
194 using age and sex)[28] versus actual values for men and women were respectively 2481
195 kcal/d versus 2326 kcal/d and 1925 kcal/d versus 1887 kcal/d.

196 Foods for which regulations exist in the EU permitting sulfite addition are shown
197 in table 4[29]. Typically a manufacturer will add sulfite up to the maximum permitted

198 level in order to achieve the longest shelf life for the product. A report on sulfite usage in
199 the UK was produced in 2001[30]. Sweet wines, langoustines (prawns), dehydrated
200 potatoes and dried fruit were not given FSS scores because their data quantity fell below
201 the <10 people or <1 kg rule. Soft drinks were split into those known to contain sulphite
202 (drinks made from fruit squash concentrates and lucozade) and the rest. In terms of
203 intake (portion size*sulfite concentration), for this population, the major sources of
204 sulphite (FSS, FSS table position) were bitter beer (3.91, 75), white wine (2.87, 73),
205 burgers (2.84, 72), soft drink concentrates (2.79, 70), sausages (2.68, 68), lager (2.47, 64)
206 and red wine (2.00, 29). A Mann-Whitney test on the FSS positions of these foods gave a
207 significance of $p < 0.001$. The sulfite-containing, alcoholic beverages; wines and beers,
208 were associated with increased UC disease activity, but spirits were not, which suggests a
209 role for sulfite rather than alcohol in the disease process. A plot of alcohol consumption
210 from wine and beer against sigmoidoscopy score revealed a significant positive
211 correlation ($n=81$, $r^2=0.07$, $p < 0.02$).

212 Decaffeinated coffee appeared better for the UC patient than the caffeine-
213 containing counterpart. Decaffeinated tea is not shown on table 3 because it was only
214 drunk by 9 people but had a FSS of 1.71 versus 2.01 for the caffeine-containing product.
215 Whole fruit consumption appeared better than the corresponding juice (e.g. fruit juice
216 scored 2.43 compared to citrus fruits at 1.96 and apples at 1.67).

217 An average thiamin concentration (mg / 100 g) (Holland, 1993 #9) for each food
218 or food group is also shown in table 3. There is a significant correlation ($p < 0.005$)
219 between this thiamin value and the food's sigmoidoscopy score.

220

220 **Discussion**

221 Ulcerative colitis is considered to have a genetic component. Twin studies[31]
222 have shown a 10% concordance of UC in monozygotic and 3% in dizygotic twins
223 suggesting about 90% environmental and 10% genetic contributions. The pool of
224 genetically susceptible individuals is therefore at least 10 times greater than those
225 diagnosed with the condition. A failure to date in identifying the gene(s) responsible
226 points to a complicated genetic component featuring multiple polymorphisms. The first
227 acute episode of UC must disrupt either, the ecology of, or the sensitivity and selectivity
228 of the immune system to, the commensal enteric microflora sufficiently to cause the
229 chronic condition. More extreme versions of the environmental conditions that lead to
230 subsequent relapses could conceivably lead to the first acute episode.

231 Of all the dietary components studied in relation to UC risk and disease severity,
232 milk has probably received the most attention. Andreson[1] was the first to postulate
233 that food allergy was the cause of UC in two-thirds of his patients, and by the use of
234 elimination diets claimed to identify the offending food and remove it. In Andreson's
235 experience, the most common provoking antigen was cow's milk. His views were
236 confirmed by Rowe[32] and later by Truelove[33]. They all postulated that milk protein
237 sensitivity was an aggravating cause of disease in up to 5% of colitic patients, who
238 benefited from a milk-free diet. While able to demonstrate circulating antibodies to milk
239 proteins more frequently and in higher titer than in matched controls, they were unable to
240 correlate the occurrence and titer of these antibodies with the extent, severity, or duration
241 of colitis, or with the response to a milk-free diet. Mishkin[34] concluded, in a review of
242 the subject, that IBD patients avoid dairy products to a much greater extent than the

243 prevalence of lactose malabsorption and/or milk intolerance in this population group
244 would justify. This observation was probably due to the incorrect perceptions of patients
245 and arbitrary advice of physicians and authors of popular diet books.

246 In order to ascertain whether dietary antigens may sustain the mucosal
247 inflammatory response, two prospective controlled trials have investigated the
248 effectiveness of bowel rest and total parenteral nutrition as primary therapy in the
249 management of acute UC[35, 36]. Neither study found any benefit over conventional
250 corticosteroid treatment alone and so the possibility of a dietary antigen driving the
251 chronicity of the disease seems unlikely. These results are in agreement with work
252 demonstrating[37] that a split ileostomy is of little benefit in the management of UC, but
253 the latter observations may have been confounded by the development of diversion
254 colitis[38].

255 The dietary analysis procedure proposed here has the potential to highlight trends
256 in dietary data that would not be apparent using traditional dietary analysis software and
257 could be useful in the study of other diseases with dietary associations. This system
258 would highlight any possible dietary factors both positive and negative, not just sulfite.
259 The proposed method is less reductionist than traditional coding as it assesses the risk of
260 each food item or group rather than the risk from the foods' (quantitated) constituents.
261 Part of the power of this study derives from the availability of a sigmoidoscopic grading
262 (0-6) of the severity and extent of the disease. This grading provides the statistical
263 variable that is normally obtained from a non-UC control group. Other alternative
264 systems for analysis of disease risk for dietary components are; the use of disease
265 occurrence odds ratios between the top and bottom quartiles of intakes, and assessing the

266 correlation coefficients between disease activity and intakes. The odds ratio method loses
267 data and data accuracy by characterizing intakes as high, high middle, low middle and
268 low and then discarding the middle two quartiles. The correlation method is dependent
269 on spread. The proposed system has neither of these disadvantages. The food
270 sigmoidoscopy score calculation does rely on the assumption that the sigmoidoscopy
271 score is an approximately linear scale, i.e. a sigmoidoscopy score of 6 is caused by the
272 consumption of a double portion of a harmful food item of sigmoidoscopy score 3. This
273 could be argued to be reasonable. Both the sigmoidoscopy grading and dietary analysis
274 method are validated methodologies. The food sigmoidoscopy score is simply a
275 mathematical function of these two variables. As all data is transformed according to the
276 same simple rules any statistical treatment of the results is as valid as statistical treatment
277 of the raw data.

278 Whilst clinical activity indices were used to generate analogous scores to the food
279 sigmoidoscopy scores, the results from these measurements are not included in this paper.
280 Clinical activity index involves subjective measurements such as a feeling of well being.
281 Thus, the food orders generated by these measurements were not thought to be as
282 accurate as those generated by the sigmoidoscopy scores.

283 The consensus of previous studies on diet and UC pointed to the modern,
284 processed, highly refined, Western diets as being damaging. The results presented here
285 linking diet with disease activity are broadly in agreement with this. Additionally they
286 propose a new risk factor for UC, namely intake of sulfited foods.

287 The involvement of diet in UC is controversial. Differences in dietary intake
288 between patients and controls could be a result of changes in diet brought on by the

289 symptoms of the disease process[4]. While this explanation is possible it does not seem
290 likely that patients would increase their beer and wine intake as a consequence of feeling
291 unwell. The relationship between sulfite intake and sigmoidoscopy score in this study
292 was extremely strong and therefore an explanation for why sulfite should be a risk factor
293 for UC is required. Sulfite has a number of effects that may be relevant to this
294 discussion. Sulfite may be important because it is a precursor of sulfate. Sulfate can
295 potentially be reduced to sulfide by sulfate reducing bacteria in the colon. Sulfide is a
296 plausible metabolic toxin in UC. Supplementing patients with sulfate decreases the
297 microbial incorporation of hydrogen into methane (as measured by breath methane) and
298 increases the in vitro sulfide production rate of feces[39]. The end metabolic product of
299 both sulfite and protein is sulfate. Sulfate from both sources can be reduced to sulfide in
300 the gut. The absence of a significant relationship between protein intake and disease
301 activity in this study does not support a mechanism for UC that involves a common
302 pathway for sulfite and protein.

303 Alternatively, the relevance of sulfite to UC may be because of its ability
304 to degrade thiamin. Thiamin deficiency manifests itself in the nervous and
305 cardiovascular systems. It is unlikely that it is the status of the patient that is important,
306 but rather the amount of thiamin available to the gut microflora. An example of the
307 importance of thiamin to the gut microflora is the requirement of the probiotic bacteria,
308 lactobacilli, for thiamin. Thiamin status is influenced by a number of factors. Firstly,
309 thiamin intake; in foods such as pork, fortified cereals and legumes which are good
310 sources of thiamin, intakes were associated with improved clinical state. Traditional
311 dietary analysis did not reveal a significant correlation between thiamin intake and

312 sigmoidoscopy scores though no allowance is made in dietary coding software for the
313 reduction in thiamin content caused by sulfite usage. Secondly, carbohydrate intake;
314 Elmadfa *et al.* demonstrated that the thiamin status of adult humans depends on
315 carbohydrate intake[40]. Carbohydrate (and sugar) intakes have previously been
316 associated with UC relapse (table 1). Finally, thiamin status can be affected by caffeine's
317 anti-thiaminergic properties. For both coffee and tea intake, the decaffeinated version
318 was associated with better clinical state.

319 However, there was a sub group (n=8) of this population who recorded an intake
320 of either vitamin B complex or multivitamins. This sub group did not have a mean
321 sigmoidoscopy score significantly lower than the general UC population. It is likely that
322 vitamin B1 is a factor in the disease process but not the only nutritional one.

323 An additional possible interpretation for the experimentally determined food order
324 is the carbohydrate nature and content of the foods. Carbohydrates, such as the α -
325 amylase resistant starch (RS) and prebiotics, escape digestion in the small intestine and
326 provide an energy substrate for the colonic microflora. Both prebiotics (found in chicory,
327 legumes, artichokes alliums, and in small amounts in cereals) and resistant starch
328 (potatoes, bananas, lentils and legumes) have been hypothesised to improve the colonic
329 health of the host. For RS, resistance to digestion is a function of the morphology of the
330 starch granules and their crystalline organisation, which is determined by the botanical
331 source of the starch and the processing it has undergone before being eaten[41].

332 Prebiotics are non-digestible carbohydrates that selectively stimulate the growth of
333 lactobacilli and bifidobacteria with benefit to health. Prebiotics are mainly fructose and
334 galactose polymers with a degree polymerisation of between 2 and 60. Of the prebiotic

335 sources; chicory and artichokes were not found in typical diets, legumes and cereals were
336 seen to have probable benefits in this study and alliums were not. This study therefore
337 provides only limited support for the use of prebiotics in UC. The foods containing RS
338 were all found to be of benefit in this study and therefore the role of RS in UC is strongly
339 supported.

340 Any dietary advice provided to ulcerative colitis patients should be based on the
341 FSS table. The table is of course imperfect because of experimental error, natural
342 variation and the associations between foods. For example, milk and cereal are coded
343 separately but are often consumed together. Thus the magnitude of the difference in the
344 FSSs for these two foods is less than if they'd been independent variables. Suggestions
345 have been made in this discussion as to the factors responsible for the FSS order and to
346 distill these factors into the advice given in Table 5. This table is speculation, as this diet
347 has not been formally tested in the UC population. It does however represent the only
348 comprehensive dietary advice available to ulcerative colitis patients at this time.

349 The list of dietary risk factors for colon cancer[42] bears a similarity to the dietary
350 risk factors presented here for UC. UC patients have an increased risk of colorectal
351 cancer and it is probable that factors responsible for inflammation in UC patients are also
352 responsible for neoplasia in the colon cancer population.

353

353

354 **Conclusion**

355 A dietary analysis method is described that provides a new tool for establishing
356 relationships between diet and disease. This method has been applied to the study of
357 ulcerative colitis and points to sulfite and caffeine as being harmful, with thiamin and
358 resistant starch being potentially therapeutic. For the first time, dietary guidelines for
359 ulcerative colitis patients, including food portion sizes have been developed.

360

360

361 **Abbreviations**

362 Ulcerative colitis (UC); Food sigmoidoscopy score (FSS); European Union (EU);

363 Odds ratio (OD) and Confidence interval (CI).

364

364

365 **Competing interests**

366 The authors declare they have no competing interests.

367

367 **Authors' contributions**

368 JHC, EAM and LME contributed to the study design and the writing of the
369 manuscript. EAM was the co-ordinator of the study and along with ST had responsibility
370 for managing the patients and coding the diaries. LME developed the dietary data
371 analysis protocol assisted by RC. CK and JHC performed the sigmoidoscopic
372 examinations of the patients. All authors read and approved the final manuscript.
373

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380 **Tables and figures legends**

381 Figure 1: Frequency distribution of sigmoidoscopy scores (n=81) for ulcerative colitis
382 patients recruited at all stages of disease.

383 Table 1: Review of studies of diet and ulcerative colitis (UC).

384 Table 2: Food sigmoidoscopy score (FSS) calculation example for red wine (NB
385 incomplete data set used). The final column value for patient 3 is $3.5 \times 750 / 2087 = 1.258$.
386 The values in the final column are summed and in this example the food sigmoidoscopy
387 score for red wine calculated as $2.614 / 0.883 = 2.96$.

388 Table 3: Foods consumed in order of food sigmoidoscopy scores (FSS). No foods
389 contribute to more than one category (with the exception of the field "all foods"). The
390 thiamin levels are the best estimate based on the distribution of foods within each
391 group[43].

392 Table 4: Permitted levels of sulfite in the UK.

393 Table 5: Proposed dietary advice for ulcerative colitis patients. The list of potentially
394 protective foods includes the average 7 d intakes for foods. The total calorific content of
395 the protective foods is approximately 30 % of that required for a whole week. Grams (g)
396 can be converted to ounces by dividing by 28.4. It is recommended that the majority of
397 the protective foods be consumed in the quantities listed. Provided sulfite containing
398 foods and coffee (except decaffeinated) are restricted all other foods can be eaten freely.
399 A varied, fresh, balanced diet is recommended.

400 *Carrageenan is to be avoided because of its link to ulcerative colitis in animal
401 models[44]. It is a type of seaweed that contains sulphur (used as a thickening agent in a

402 wide range of desserts, e.g. chocolate mousse). This study was not able to determine
403 whether or not this additive is harmful so advice is precautionary.

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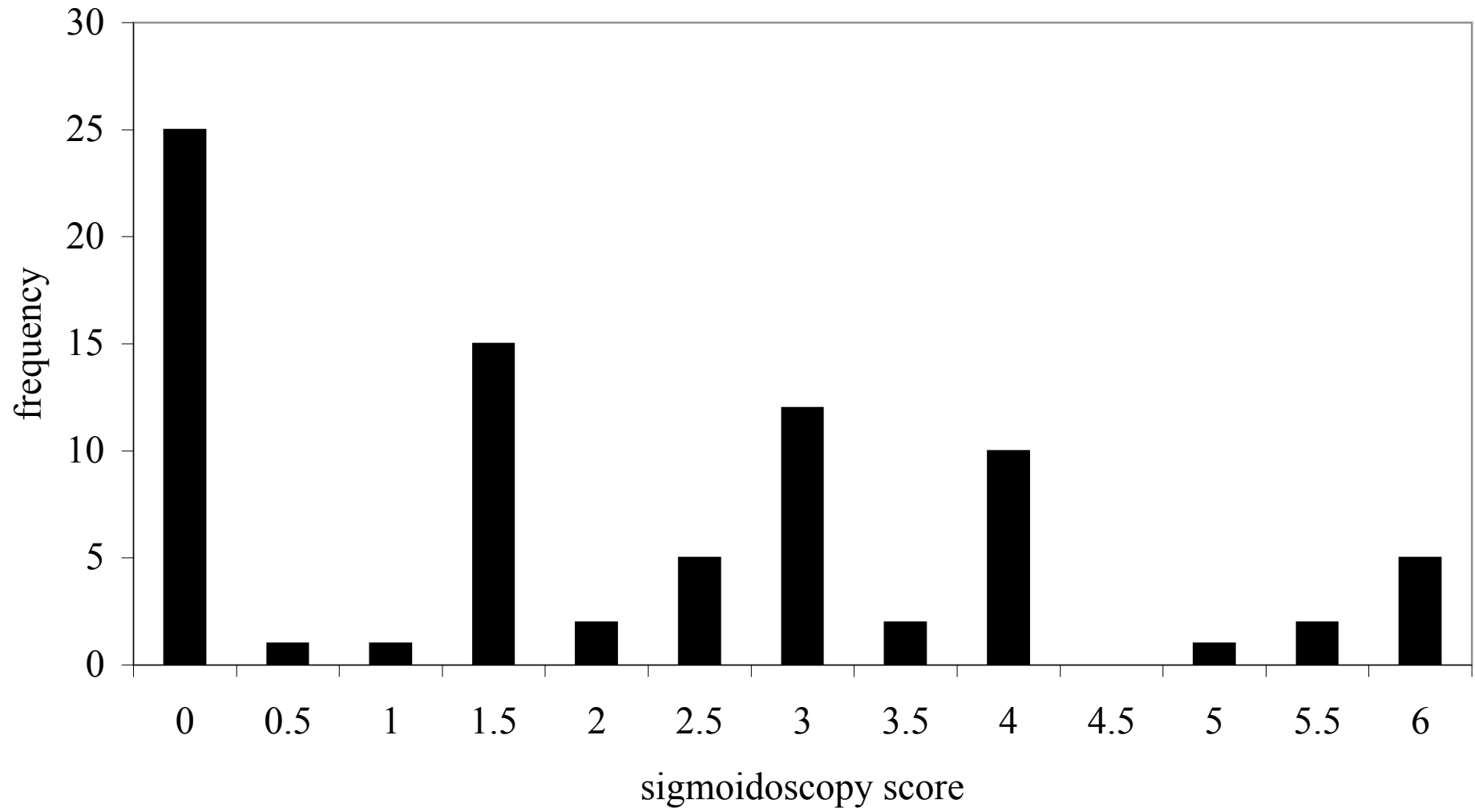


Figure 1

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