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Original article

Perceived dietary intolerances, habitual intake and diet quality of patients with an ileoanal pouch: Associations with pouch phenotype (and behaviour)

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SUMMARY

Background and aims: Ileoanal pouch patients frequently attribute pouch-related symptoms and pouchitis with diet. We aimed to assess perceived food intolerance and habitual dietary intake and their relationship with pouch indication, symptoms and current or history of pouchitis.

Methods: In this cross-sectional study, patients with an ileoanal pouch completed a dietary intolerance and a food frequency questionnaire, that specifically quantifies habitual intake of FODMAPs. Perceived dietary intolerance rates, nutrient intake and diet quality, and their differences based on pouch indication, symptom, and current or history of pouchitis were assessed. Associations between intolerances and intake, and between dietary intake with pouchitis risk were analysed using univariable and multivariable regression analysis.

Results: Of the 58 (10 FAP and 48 UC) patients with complete data, 81% of UC and 80% of FAP patients reported dietary intolerances. Overall diet quality was good. Differences in dietary intake were limited to a few food groups. Patients with a history of pouchitis had a lower intake of fruits ($p = 0.03$) and nuts ($p = 0.004$). Patients with current pouchitis had a lower intake of nuts ($p = 0.02$). On multivariable logistic regression, intake of dietary fibre was associated negatively [OR 0.68(95%CI:0.51-0.92)] and of non-digestible oligosaccharides positively with pouchitis history [OR 5.5(95% CI:1.04-29.1)].

Conclusions: In patients with an ileoanal pouch, perceived dietary intolerances are common but had minimal impact on nutritional adequacy and diet quality. Negative associations of the intakes of fruits, nuts and dietary fibre and positive association with non-digestible oligosaccharides with a history of pouchitis require further study to inform dietary recommendations.

1. Introduction

Patients with ulcerative colitis (UC) and familial adenomatous polyposis (FAP) who undergo restorative proctocolectomy with ileal pouch–anal anastomosis, frequently report dietary intolerances [1] associated with pouch-related symptoms. The increased pouch emptying frequency, decreased stool consistency, urgency and bloating markedly affect quality of life and potentially lead to self-directed dietary restrictions [1]. Several studies have explored dietary intolerances and habitual dietary intake in pouch patients, but are beset by numerous methodologic limitations and heterogeneity. Conclusion derived from these studies are, therefore, tentative at best, and suggest that fruits and vegetables are associated with pouch-related symptoms and are consumed less amongst pouch patients compared with healthy controls [2]. Additionally, the limited and inconclusive findings do not permit the formulation of dietary recommendations for patients with a pouch, an area that is clearly lacking in clinical guidelines for the management of these patients.

There is a growing interest to understand the role of dietary factors on pouch function and its involvement in the pathogenesis of pouchitis. The pursuit of such knowledge is important to identify

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Abbreviations:

BCFA	branched-chain fatty acids
CNAQ	Comprehensive Nutrition Assessment Questionnaire
CQOL	Cleveland Global Quality of Life
DGI	Dietary Guideline Index
FAP	Familial Adenoma Polyposis
FODMAP	Fermentable oligo-, di- and mono-saccharides and polyols
FDR	False Discovery Rate
HRQOL	health-related quality of life
H2S	Hydrogen sulfide
IQR	Interquartile range
PDAI	Pouchitis Disease Activity Index
SCFA	short-chain fatty acids

avenues to direct dietary therapies for management and prevention of pouchitis. The current understanding of the pathogenesis of pouchitis is that it is an interaction of pouch dysbiosis with dysregulated pouch mucosal immunity, both of which can be influenced by diet [3–6]. Pouch dysbiosis is characterised structurally by lower microbiota density and diversity [3], lower relative abundance of putatively protective bacteria such as *Bifidobacteria* and butyrate-producing bacteria [3], and increased abundance of *Bilophila wadsworthia* [7], and functionally by lower short-chain fatty acids (SCFA) and higher hydrogen-sulphide (H_2S) faecal concentrations and production *in vitro* in those with pouchitis compared with those with healthy pouches [7–9]. Dysregulated pouch mucosal immune response is characterised by defective barrier function and active adaptive immunity in those with a history of pouchitis [10,11].

Studies investigating the relationship between diet and the risk of pouchitis are limited to those from one research group in Israel and report an inverse association between the consumption of fruits but not vegetables, and rates of pouchitis [12], and that good adherence to a Mediterranean diet has greater likelihood for lower faecal calprotectin levels and tends to protect against developing pouchitis [13]. If this represents a causal relationship, which food components are mechanistically responsible are not known. Additionally, it is unknown whether patients with active symptoms in these studies [3,13] tended to have poorer intakes of fruit or worse adherence to the Mediterranean diet due to dietary restrictions or food intolerances.

Studies investigating the relationship of diet with pouch function and the risk of pouchitis share several additional limitations. First, they have not assessed differences in dietary intake and intolerance rates between different groups of pouch patients. Secondly, studies have not correlated reported dietary intolerances with actual dietary intake. Thirdly, none of the studies assessing dietary intake have quantified FODMAP intake, relevant due to their possible association with symptoms [14]. Finally, given food or nutrients are not consumed in isolation and complex interactions exist that may underpin diet-pouchitis relationship, there is good rationale to explore whole-diet quality in patients with an ileo-anal pouch.

Therefore, the aims of this study are three-fold: (a) to investigate the perceived dietary intolerance in patients with ileoanal pouches and their association with clinical (FAP vs UC) and pouch-based characteristics (symptomatic vs asymptomatic and pouchitis status); (b) to define habitual dietary intake of pouch patients with a specific focus on FODMAPs [15] and diet quality and examine whether differences exist between pouch patient types; (c) to assess associations of dietary intolerance with dietary intake, and the association of diet intake and the history of pouchitis.

2. Methodology

2.1. Study population

Patients with an ileoanal pouch, created for UC or FAP, were consecutively enrolled to take part in this cross-sectional study over the course of 2.5 years from August 2018 and February 2021. They were recruited from various outpatient IBD clinics in metropolitan hospitals in Victoria, Australia and via word of mouth/advertisements at private gastroenterology/surgical clinics. Patients were eligible if they had an ileoanal pouch with bowel continuity for at least 12 months and were aged 18 years or older. Exclusion criteria included the inability to provide informed consent.

2.2. Study protocol

Patients were asked to complete two dietary questionnaires that were sent via email and completed electronically. Demographic and clinical data were collected by the study doctor over the telephone, telehealth or in a face-to-face interview. Patients with a UC-pouch also underwent a pouchoscopy as part of this cross-sectional study as described elsewhere in detail [16]. Symptomatic patients with FAP underwent a pouchoscopy with their treating specialist. This study was conducted according to the guidelines of the Australian National Statement on Ethical Conduct in Human Research National Statement on Ethical Conduct in Human Research and all procedures involving patients were approved by Alfred Health Ethics Committee (HREC 376/18). Written informed consent was obtained from all participants prior to study enrolment.

2.3. Pouch-specific dietary intolerance questionnaire

The dietary intolerance questionnaire was developed by Monash University in conjunction with two gastrointestinal dietitians, using REDCap® for the purposes of this study, and consisted of closed- and open-ended questions. Participants were asked questions regarding the presence of dietary intolerances before and after the creation of the pouch, were provided with a list of 27 food items commonly identified as culprit foods along with free text for listing other food triggers and were asked to associate symptoms with the individual food items. Patients were also asked whether they sought dietary information and what sources they used, whether they eat foods which they did not tolerate, and whether they overcame dietary intolerances and, if so, when in relation to pouch creation. A sample of the questionnaire is included in Supplementary Fig. 1.

2.4. Dietary intake questionnaire

Habitual dietary intake over the previous six months was assessed using the Comprehensive Nutritional Assessment Questionnaire (CNAQ), a validated 297-item semi-quantitative food frequency questionnaire (FFQ) designed for assessment of FODMAPs, fibre and a range of macro- and micronutrients [15]. Incomplete FFQs were excluded from analysis. Dietary data were screened for implausible energy intakes by the study dietitian according to previously published protocol [15]. Briefly, a ratio of energy intake (EI) compared with estimated basal metabolic rate using the Schofield Equation (was used to determine those who substantially underestimated their intake as previously described

[15]. A cut-off in EI/BMR <1.35 for the FFQ based on 95% confidence intervals was applied [17] Patients who under- or over-reported total energy intake were asked to repeat the CNAQ and patients who consistently underestimated their energy intake were excluded from analysis. Nutrient intakes were then compared against the 2006 Nutrient Reference Values for Australia and the proportion of individuals meeting nutrient recommendations were calculated [18].

To calculate the number of food servings consumed, frequency responses from the CNAQ was first converted to a daily equivalent frequency using methodology from previously published studies [19,20] and subsequently, multiplied by portion sizes provided by the CNAQ questionnaire. If needed, food items were re-classified into food groups according to the 2013 Australian Guide to Healthy Eating [21]. Additional subgroups were also computed including 'high fat', 'high sugar', 'high fat with sugar' and alcohol within the discretionary food category. Furthermore, snacks, condiments, drinks and fats and oils not present in the 2013 Australian Guide to Healthy Eating were retained as separate categories.

2.5. Dietary guideline index (DGI)

The DGI is a scoring system of an individuals' diet quality that compares how closely their diet complies with the Australian Guide to Healthy Eating (see supplementary material) [22]. The DGI used in the present study comprised 11 components, omitting 2 components (total fluids and added salt) from the 13 components that make up the DGI-2013 published previously, as these could not be obtained from the CNAQ [23]. Each component is scored out of ten with the total DGI score ranging from 0 to 110; a higher score indicated greater compliance with the dietary guidelines, hence higher diet quality. The components are divided into two categories (1) those that reflect adequate intake of nutritious foods from the core food groups (vegetables, fruits, cereals, dairy and alternatives and meat and alternatives) as well as variety within these core food groups, and (2) those that reflect limited intake of discretionary foods such as food and drinks high in saturated fat and/or added sugar or alcohol [24]. Each component is scored out of ten, with zero indicating the guideline was not met and ten indicating the guideline was sufficiently met. Items with two subcomponents were scored out of five. The cut-offs used to obtain the maximum score for components were guided by the age- and sex-specific food-based daily recommendations outlined in the Australian Dietary Guideline(ADG) [22]. For the variety component, foods scored 1 point if consumed above a cut off (>15 g/d for beverages and >30 g/d for foods), and overall variety was estimated within each core food group by summing scores for each food group and dividing by the total number of foods within each core food group. Scores were summed across the five core food groups and multiplied by two to create a score out of 10 [25].

2.6. Clinical categorisation of pouch patients

Patients were categorised according to pouch indication (FAP *vs* UC) and symptoms associated with the pouch according to the clinical subscore of the Pouchitis Disease Activity Index (PDAI) [26] where <3 was considered asymptomatic *vs* ≥3 symptomatic. Patients with UC were further categorised according to their pouchitis history (present or absent) and whether they had current pouchitis (total PDAI was ≥7) or not. If symptomatic with PDAI < 7, they were considered to have irritable pouch syndrome.

2.7. Statistical analysis

Data were stored on an Excel spreadsheet [Microsoft] and analysed on SPSS® v27 statistical analysis software (IBM®; New York, USA) [27]. Continuous variables are presented as mean (SD) and median (IQR). Nominal variables are summarized in frequency tables and are presented as *n* [%]. Continuous variables were compared using the independent 2 sample t-test and Mann–Whitney U test according to the normality of their distribution. Nominal variables were compared using Chi-square test and Fisher's Exact test. Mean total DGI scores and mean scores for each component were calculated and compared between groups using independent 2 sample t-test. Correlation between intolerance and intake was assessed using univariate and multivariate linear regression analysis. Univariable and multivariable logistic regression analyses was used to assess the relationship of the intake of food groups, nutrients, and diet quality, and pouchitis history. Multivariable analysis included covariates of a priori importance (patient age, sex patient age at the time of UC onset, and pouch age), exploratory items (oligosaccharides and resistant starch), and covariates with a $p < 0.1$ in univariable analyses. All tests are two-sided and considered significant at $p \leq 0.05$. However, for multiple comparisons, a False Discovery Rate (FDR) was applied, and results considered significant at FDR ≤ 0.1.

3. Results

3.1. Participants

Supplementary Figure 2 shows the flow of participants. Of 80 patients invited to participate, 9 declined to participate, 10 did not complete ≥1 questionnaire, and 3 were excluded for significantly under-reporting their energy intake. Consequently, data from 58 patients were analysed, 48 with UC and 10 with FAP. The demographic and pouch characteristics of the patients are shown in Table 1. Despite a similar pouch age, none of the FAP patients had a history of pouchitis, whereas 37(77%) of UC pouch patients did. Clinical characteristics of UC pouch patients according to pouchitis status is shown in Table 1. Patients with a history of pouchitis were a mean of 17 years younger ($p = 0.002$) and had an onset of ulcerative colitis 16 years earlier than those without ($p = 0.002$).

3.2. Sources of dietary information

Of the 22 patients (38%) who had seen a dietitian at some point after their pouch was created, 20 were advised to follow a low FODMAP diet and 19 to avoid nuts. Twenty-one patients (37%) sought additional dietary advice from other sources, most commonly the internet (16%) and/or their gastroenterologist. There were no differences in the percentage of patients seeking dietetic advice, the type of advice given or additional sources for dietary information according to pouch indication or pouch-related symptoms. However, more UC patients with a history of pouchitis sought sources other than dietitians (16/37(43%) vs 1/11(9%); $p = 0.03$), the most common source being the internet.

3.3. Dietary intolerance

As shown in Table 2, perceived dietary intolerance to at least one food group was reported in 39(81%) UC pouch patients and 8(80%) FAP pouch patients. In two thirds of the UC pouch patients with dietary intolerances, onset was prior to pouch creation. Twenty-six

Table 1
Baseline characteristics of all ileoanal pouch patients, categories according to pouch induction and symptoms, and for ulcerative colitis pouch patients further categorised according to pouchitis history or current pouchitis.

			FAP (n = 10)	UC (n = 48)	*p*-value	Asymptomatic pouch (n = 38)	Symptomatic pouch (n = 20)	*p*-value	No Pouchitis history (n = 11)	Pouchitis history (n = 37)	*p*-value	Without pouchitis (n = 30)	With pouchitis (n = 18)	*p*-value
Pouch indication	Ulcerative colitis, n (%)		0	48 (100%)	NA	30 (79%)	18 (90%)	0.46[a]	11(33%)	37(77%)	1	30(100%)	18(100%)	1
	FAP, n (%)		10 (100%)	0	NA	8 (21%)	2 (10%)		0	0	NA	0	0	NA
Age (y), mean (SD)			38.5(15.2)	48.5(16.4)	0.06[b]	48.8(17)	48(15.1)	0.56[b]	60.9(16)	44.3(14.5)	0.002[b]	49(17.5)	47.6(14.8)	0.78[b]
Male sex, n (%)			4(40%)	29(60%)	0.9[c]	25(66%)	9(45%)	0.16[c]	8(73%)	21(57%)	0.48[a]	21(70%)	8(44%)	0.12[a]
Age(y), at ulcerative colitis onset, mean (SD)			NA	27.9(12)	NA	27.3(12.6)	27.6(12.5)	0.4[b]	40(12)	24.3(11)	0.002[b]	25.5(18.4–37)	22(18.7–36.4)	0.87[d]
Pouch age (y), mean (SD) r			15.7(10.7)	13.5(7)	0.42[b]	13.9(7.1)	12.9(6.9)	0.52[b]	14.5(11–20)	11(7.5–18.5)	0.45[d]	11.5(9.8–17.5)	11(6–21.5)	0.64[d]
Extra-intestinal manifestations, n (%)			0	35(73%)	<0.001[a]	23(61%)	12(60%)	1[a]	7(64%)	28(76%)	0.7[a]	23(77%)	12(67%)	0.5 [d]
Presence of auto-immune co-morbidities, n (%)			1	21(44%)	0.07[a]	13(34%)	9(45%)	0.57[a]	6(45%)	15(41%)	0.49[d]	14(47%)	7(39%)	0.76 [d]
Smoking status	Smoker, n (%)		0	3(6%)	0.66[a]	1(3%)	2(10%)	0.60[a]	0(0%)	3(8%)	0.11[a]	2(7%)	1(6%)	0.22[a]
	Ex-smoker, n (%)		3(30%)	8(17%)		8(21%)	3(15%)		4(36%)	4(11%)		7(24%)	1(6%)	
	Non-smoker, n (%)		7(70%)	37(77%)		29(76%)	15(75%)		7(64%)	30(81%)		21(70%)	16(89%)	
NSAID use	None or occasional use		4(80%)	41(87%)	0.5[a]	29(86%)	16(89%)	0.93[a]	10(90%)	32(86%)	0.23[a]	26(87%)	15(88%)	1[a]
	Regularly previously		1(20%)	5(11%)		4(12%)	2(11%)		0	5(14%)		3(10%)	2(11%)	
	Regularly currently		0	1(2%)		1(3%)	0(0)		1(8%)	0		1(4%)	0(0%)	
Pouchitis history, n (%)			0	36(75%)	<0.001[a]	20(67%)	16(89%)	0.16[a]	11(100%)	37(100%)	NA	19(63%)	18(100%)	<0.00[c]
Current pouchitis, n (%) %			0	21(44%)	0.009[a]	9 (30%)	12(67%)	0.01[c]	0	18(49%)	0.002[a]	30(100%)	18(100%)	NA
Clinical PDAI, median (IQR)			2(0.75–2.25)	2(1–3)	0.38[d]	1(1–2)	4(3–4)	<0.001[d]	2(1–3)	2(1–3)	0.32[d]	1(1–2)	3(2–4)	0.002[d]
Symptomatic pouch (Clinical PDAI ≥ 3)			2(20%)	18(38%)	0.47[a]	0	18(100%)		2(18%)	16(43%)	0.17[a]	6(20%)	12(67%)	0.002[c]
Pouch phenotype	No pouchitis history, n (%)		7(70%)	11(23%)	<0.001[a]	17(45%)	3(15%)	0.06[a]	11(100%)	0	<0.001 [a]	11(41%)	0	0.005[a]
	Acute recurrent pouchitis, n (%)		0	9(19%)		5(13%)	4(20%)		0	9(24%)		6(20%)	3(17%)	
	Chronic Pouchitis, n (%)		0	28(58%)		16(42%)	12(60%)		0	28(76%)		13(43%)	15(83%)	
	Isolated pre-pouch ileitis, n (%)		1(10%)	0		0	1(5%)		0	1(3%)		1(3%)	0	
	Irritable pouch syndrome, n (%)		2(20%)	6(11%)	NS	0(0%)	7(35%)	<0.001[a]	2(13%)	4(11%)	0.9[a]	6(33%)	0	<0.001[a]
Dietary advice	Dietitian		4(40%)	38%	1[a]	15(39%)	7(35%)	1[a]	5(45%)	13(35%)	0.7[b]	11(37%	7(38%)	1[b]
	Additional sources	All, n (%)	8(17%)	1[a]	6(16%)	4(20%)	0.72[a]	1(9%)	7(19%)	0.6[b]	5(17%)	3(17%)	1[b]	1[b]
		Internet, n (%)	3(30%)	13(27%)	0.7 [a]	11(30%)	5(25%)	0.8	1(9%)	12(32%)	0.2[b]	8(27%)	5(28%)	1[b]
		Gastroenterologist, n (%)	2(2%)	11(23%)	1[a]	7(19%)	6(30%)	0.5	1(9%)	10(27%)	0.4[b]	6(20%)	5(28%)	0.7[b]
		Other, n (%)	2(20%)	8(17%)	1[a]	6(16%)	4(20%)	0.72[a]	1(9%)	7(19%)	0.6[b]	5(17%)	3(17%)	1[b]

Table 2
Dietary intolerances of pouch patients according to sex, pouch indication, presence of absence of pouch-related symptoms, and in UC pouch patients according to history and current pouchitis.

Intolerable food item		All pouch patients	Sex			Pouch indication			Pouch-related symptoms (Clinical PDAI ≥3)			UC with pouchitis history			UC with current pouchitis (total PDAI≥7)		
N (%)		58	F 25	M 33	*p*-value	FAP 10	UC 48	*p*-value	No 38	Yes 20	*p*-value	No 11	Yes 37	*p*-value	No 30	Yes 18	*p*-value
Dietary intolerance		82%	73 %	95 %	0.07	80 %	81 %	NS	74 %	95 %	0.07	72 %	84 %	0.409	77 %	89 %	0.45
Fruits	All	41%	48 %	36 %	NS	20 %	46 %	0.17	58 %	42 %	0.4	36 %	49 %	0.5	43 %	50 %	0.76
	Fruits excluding citrus	26%	32 %	21 %	NS	10 %	29 %	0.42	21 %	35 %	0.34	18 %	32 %	0.46	27 %	33 %	0.75
	Citrus fruits	26%	28 %	24 %	NS	20 %	27 %	NS	26 %	25 %	NS	27 %	27 %	NS	23 %	33 %	0.51
Vegetables	All	48%	68 %	33 %	0.01	30 %	52 %	0.3	45 %	55 %	0.58	46 %	54 %	NS	50 %	56 %	0.77
	Green vegetables	10%	21 %	3%	0.07	12 %	12 %	NS	10 %	11 %	NS	9%	10 %	NS	10 %	11 %	NS
	Garlic and onion	26%	36 %	18 %	0.14	20 %	27 %	NS	16 %	45 %	0.026 [a]	27 %	27 %	NS	23 %	33 %	0.51
	Legumes	26%	36 %	18 %	0.14	10 %	29 %	0.423	29 %	20 %	0.54	36 %	27 %	NS	30 %	28 %	1
Dairy products incl. yoghurt		31%	40 %	21 %	0.15	30 %	29 %	NS	29 %	30 %	NS	36 %	27 %	NS	27 %	33 %	0.75
Carbohydrates	Unrefined carbohydrates	8%	8%	9%	NS	10 %	8%	NS	8%	10 %	NS	9%	8%	NS	7%	11 %	0.6
	Refined carbohydrates	19%	32 %	9%	0.04	0%	22 %	0.18	8%	40 %	0.005 [b]	18 %	24 %	NS	17 %	33 %	0.28
	Starchy foods	8%	12 %	6%	0.6	10 %	8%	NS	8%	10 %	NS	18 %	5%	0.22	10 %	6%	1
Meats and alternatives	Red meat and deli meats	12%	12 %	12 %	NS	0%	14 %	0.33	13 %	10 %	NS	9%	16 %	NS	13 %	17 %	0.45
	Chicken, fish and eggs	10%	12 %	9%	NS	10 %	11 %	NS	5%	20 %	0.16 [b]	9%	11 %	NS	10 %	11 %	0.6
	Nuts	29%	28 %	30 %	NS	10 %	33 %	0.25	24 %	40 %	0.23	18 %	38 %	0.29	27 %	44 %	0.17
Spicy foods		50%	56 %	45 %	0.45	40 %	52 %	0.7	47 %	55 %	NS	45 %	54 %	0.73	56 %	44 %	0.55
Fatty/oily meals		31%	36 %	27 %	0.57	30 %	31 %	0.5	18 %	55 %	0.007 [a]	18 %	35 %	NS	27 %	39 %	0.52
Drinks	Coffee	14%	12 %	15 %	NS	10 %	14 %	10	13 %	15 %	NS	9%	16 %	0.39	17 %	11 %	1
	Carbonated drinks	17%	20 %	15 %	NS	10 %	19 %	NS	11 %	30 %	0.08 [b]	9%	22 %	0.6	13 %	28 %	0.26
	Beer, wine and other alcohol	33%	36 %	30 %	NS	40 %	31 %	NS	37 %	25 %	NS	18 %	35 %	0.46	26 %	39 %	0.52

percent of UC pouch patients overcame intolerance to at least one food group at a median of 24(IQR 12–45) months in comparison to 40% of FAP pouch patients after 36(12–105) months. Eighty five percent of patients continued to eat at least one food item perceived to induce symptoms.

High perceived intolerance rates (defined as ≥ 30% of the patients) were reported for fruits, vegetables, dairy products, spicy foods, fatty and oily meals, and alcoholic beverages, with no statistically significant differences between men and women, or indication for the pouch. The only differences in reported intolerances according to pouch phenotype were higher rates of intolerance to garlic and onion, fatty and oily meals, and refined carbohydrates ($p < 0.05$) in those with a symptomatic pouch.

Symptoms associated with dietary intolerances varied with food type and are described in Table 3. Diarrhoea was the most common symptom reported to most food groups, followed by looser stools, increased urgency and flatulence.

3.4. Dietary intake

3.4.1. Pouch patients overall

As shown in Fig. 1, 67% of pouch patients met (and exceeded) the recommended daily intake for fruits and 50% for vegetables, but less than a third met the recommended daily intake for cereals and grains, lean meat and alternatives, and dairy products. Mean dietary fibre intake was high with 69% of pouch patients meeting the

Table 3
Macro- and micronutrient intake of pouch patients.

			Pouch patients N = 58	NRV[a]
Macronutrients				
Energy Kcal/day, mean (SD)			2509.56 (789)	
Carbohydrates	Total g/day, mean (SD)		298 (103)	
	Percentage of total energy intake		48%	45–60%
	Sugar g/day, median (IQR)		134 (101–190)	
	Starch g/day, median (IQR)		126 (105–167)	
Fat	Total g/day, mean (SD)		100 (37)	
	Percentage of total energy intake, %		36%	<30%
	Saturated fat g/day, mean (SD)		37.9 (16)	
	Monounsaturated fat g/day, mean (SD)		40.7 (17.6)	
	Polyunsaturated fat g/day, mean (SD)		12.4 (5.4)	
Proteins	Total g/day, mean (SD)		111(40)	
	Percentage of total energy intake		18%	15–20%
Fibres	Total Fibre g/day, mean (SD)		35.3 (15)	25–30
	Met daily recommended target		69%	
	Resistant starch g/day, mean (SD)		4 (1.7)	
FODMAPs	Oligosaccharides	Total g/day mean (SD)	5.4 (2.4)	
		Fructans g/day, median (IQR)	4.1 (2.8–5.5)	
		GOS g/day, median (IQR)	1.1(0.6–1.5)	
	Lactose g/day, median (IQR)		15.5 (9.7–25.2)	
	Excess fructose g/day, median (IQR)		3.7(2–5.7)	
	Polyols g/day, median (IQR)		2.9 (1.2–5.2)	
Micronutrients				
Vitamins	Vitamin A mg/day, median (IQR)		384 (247–563)	500–625
	Vitamin C mg/day median (IQR)		160 (107–219)	30
	Thiamine mg/day, median (IQR)		2.3 (1.5–3.7)	0.9–1.0
	Riboflavin mg/day, mean (SD)		3.4 (1.7)	0.9–1.1
	Niacin mg/day, median (IQR)		28.6 (11.6)	11–12
	Folate mg/day, median (IQR)		1562 (1002–1887)	320
Minerals	Sodium mg/day, mean (SD)		2525 (1007)	460–920
	Potassium mg/day, mean (SD)		4574 (1872)	2800–3800
	Iron mg/day, mean (SD) median (IQR)		13.5 (11.4–16)	5–8
	Magnesium mg/day, mean (SD)		392 (144)	265–350
	Zinc mg/day, mean (SD)		12.6 (4.5)	6.5–12
	Calcium mg/d, median (IQR)		1093 (749–1428)	840–1100
	Phosphorous, median (IQR)		1748 (1351–2143)	580

NRV, Nutrient Reference Values. Estimated Average Requirements are used for comparisons where available, and Adequate Intake for fibre, potassium and sodium.

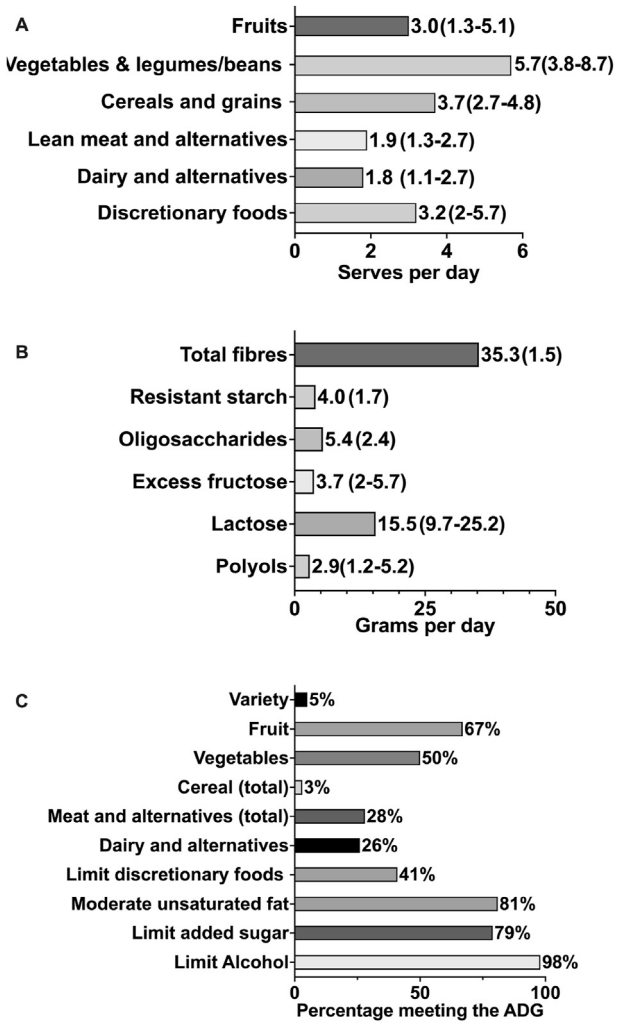

Fig. 1. A) Median (interquartile range) serves of each food group consumed daily by patients. B) The daily intake of total dietary fibres, resistant starch and FODMAPs. C) The percentage of patients meeting the recommended intake as per the Australian Dietary Guideline (ADG).

recommended daily intake and a similarly high level of intake was seen for oligosaccharides. Median lactose intake was moderate with 10% having less than 3 g a day. The daily consumption of the main food groups expressed as servings are shown in detail in Supplementary Table 1. In terms of nutrient intake, fat comprised a greater percentage of total energy intake than recommended in >50% (Table 3). Furthermore, intake of vitamin A was below the estimated average requirements, and around 69% met recommendations for calcium, whilst 98% met the recommendations for iron and zinc. Diet quality was good with an overall mean DGI score of 78(SD 12), out of a total of 110, but only 5% of the patients achieved a full score for food variety. As illustrated in Fig. 1, patients scored lowest on limiting discretionary food and having adequate intake of cereals and grains, and highest on moderating alcohol and unsaturated fat. The mean (SD) scores of the DGI components and subcomponents and the proportion meeting guidelines are shown in Supplementary Table 2.

3.5. Comparisons according to pouch phenotype

Details of the intake of food groups, nutrients and diet quality according to different aspects of pouch phenotype are shown in Figs. 2 and 3 and Supplementary Tables 3–5. Patients with UC as the underlying disease had no statistically significant differences in intake of daily servings of any of the food groups, but intake of sugar was 35 (21)% lower and carbohydrates 25 (9)% lower than those in patients with FAP. However, carbohydrates did not make more of total energy intake neither did it have an impact on total fibre or fibre/FODMAP types. In terms of diet quality, the overall mean (SD) DGI in the UC group was numerically higher than that of the FAP group [78.8 (10.3) vs 71.8 (17.4); p = 0.09], driven by higher scores in limiting added sugar and alcohol components.

There were no differences in food group consumption and nutrient intake according to the presence of pouch symptoms. For

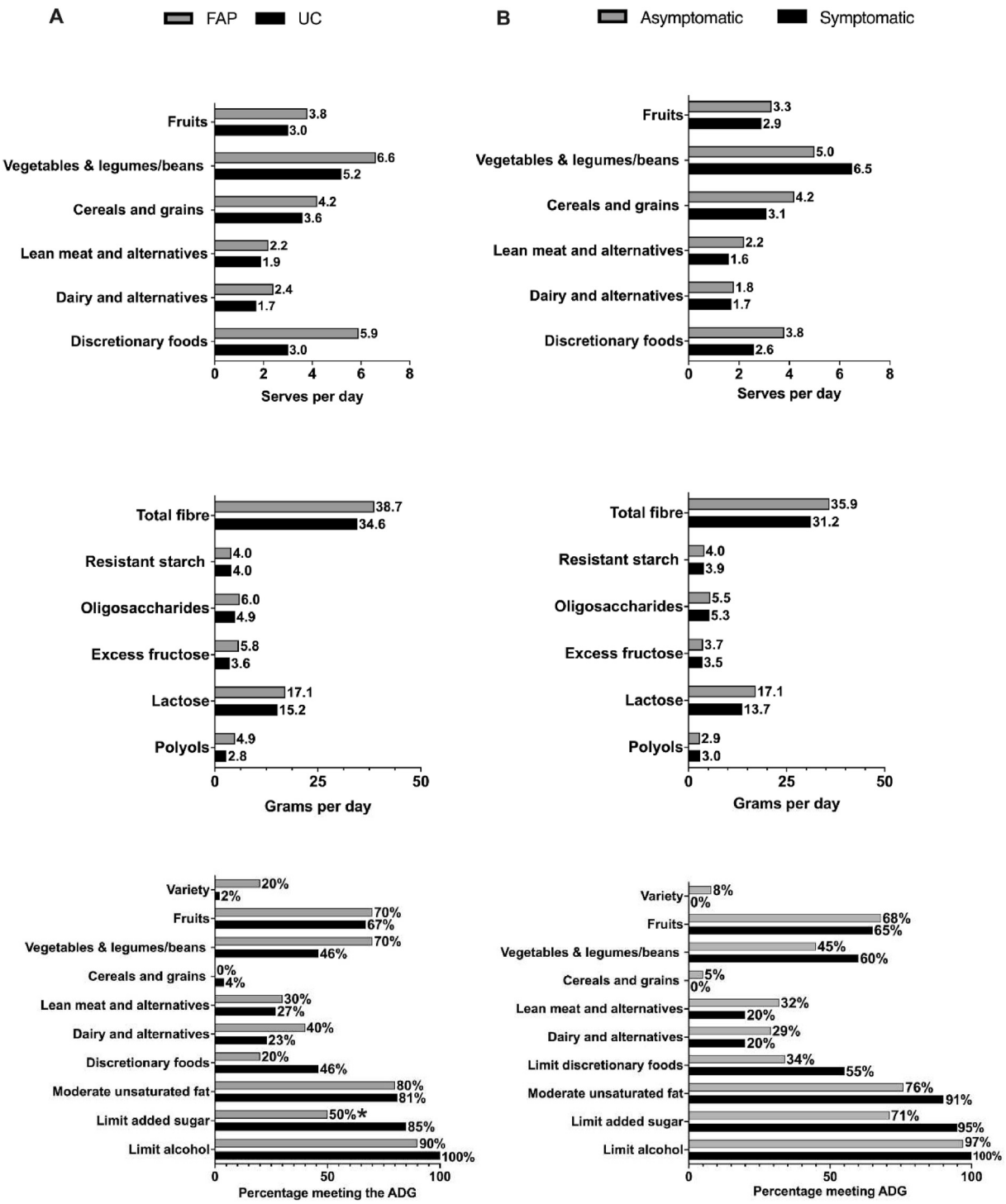

Fig. 2. Comparisons of the daily consumption of the core food groups, dietary fibres and FODMAPs and percentage of patients meeting the Australian Guide to Healthy Eating of pouch patients according to pouch indication (A) and pouch symptom status (B). *$p = 0.01$, FDR = 0.08. FAP, familial adenomatous polyposis; UC, ulcerative colitis. ADG, Australian Dietary Guidelines.

diet quality, symptomatic patients had a higher score in limiting added sugar [9.5(2.2) vs 7(4.6); $p = 0.01$, FDR = 0.08].

Patients with a history of pouchitis ate fewer median (IQR) daily servings of fruits [2.4(1.2–4.5) vs 3.8(3–8.3); $p = 0.03$, FDR = 0.1] and nuts ($p = 0.004$, FDR = 0.04) and a larger serving of condiments, sauces and spreads ($p = 0.002$, FDR = 0.01). There were no statistically significant differences in the daily intake of macro- and micro-nutrients or in the diet quality.

Patients with current pouchitis had a lower intake of lean meat and alternatives ($p = 0.006$, FDR = 0.06) specifically nuts ($p = 0.02$, FDR = 0.10). They also had 23(10)% lower overall energy intake from total fat (p = 0.011, FDR = 0.05) and lower intake of saturated and unsaturated fats. The intake of lactose and fructose (in excess of glucose), but not other FODMAP groups, was lower when pouchitis was present. Intake of micronutrients and diet quality did not differ if pouchitis was present.

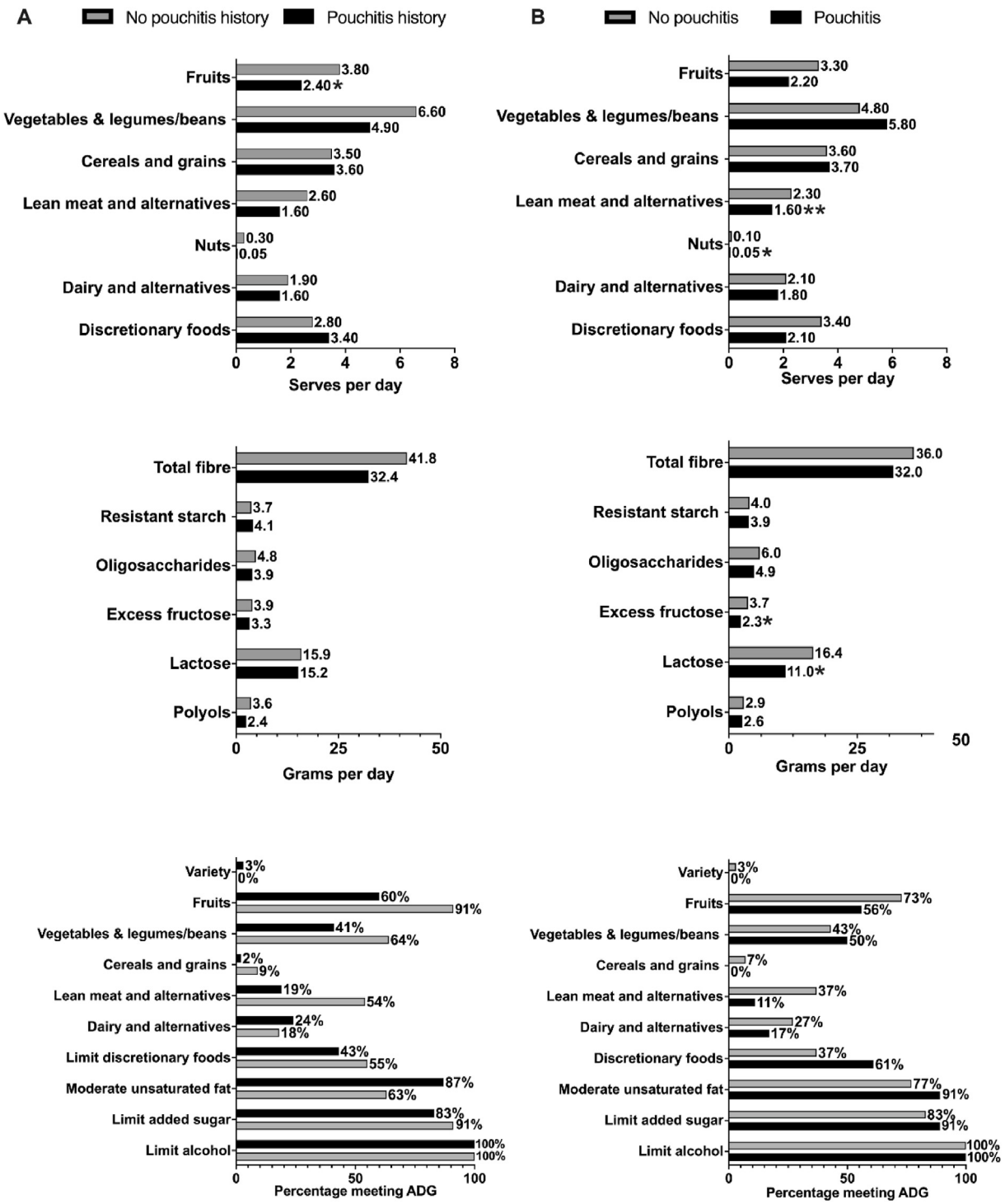

Fig. 3. Comparisons of the daily consumption of the core food groups, dietary fibres and FODMAPs and percentage of patients meeting the ADG of pouch patients according to pouchitis history (A)and current pouchitis status (B). Current pouchitis is based on a total PDAI ≥7). *p < 0.05 and FDR ≤ 0.1. ADG, Australian Dietary Guidelines.

3.6. Correlation of dietary intolerance and dietary intake

In order to determine if perceived dietary intolerances was negatively associated with the intake of the specific dietary factors, univariable and multivariable linear regression analysis of the correlation of intolerance and intake was performed Table 4. In univariable analysis, intolerances to dairy products [OR-0.77(−1.2 to −0.25); $p = 0.004$] and nuts [OR = −0.27(−0.02 to −0.52); $p = 0.03$] were negatively associated with intake of lactose-containing dairy products but not lactose-free dairy products. Multivariable analysis included the covariates sex, age, and any covariate with $p < 0.1$ in the univariable analysis (pouchitis history).

Table 4
Correlation of dietary food intolerance and intake.

		Univariable (beta coefficient 95% CI)	*p*-value	Multivariable (beta coefficient 95% CI)	*p*-value
Fruits		−1.2(-2.9–0.38)	0.14		
Vegetables & legumes/beans	All	0.94(-1.2–2.89)	0.41		
	Legumes	−0.01(-0.15–0.12)	0.86		
Cereals and grains including bread	All	−0.21(-2.0–1.6)	0.8		
	Whole grain bread	−0.38(-0.77–0.12)	0.06		
	Total bread	−0.74(-1.5–0.07)	0.07		
Lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans	All	−0.06(-0.6–0.46)	0.82		
	Nuts	**−0.27(-0.52–-0.02)**	**0.03**	−0.23(-0.48–0.01)	0.06[a]
Milk, yoghurt, cheese and/or alternatives (mostly reduced fat)	All	−0.67(-1.5–0.17)	0.133		
	Dairy (lactose-containing)	**−0.94(-1.7–-0.10)**	**0.029**	**−0.288(-1.8–-0.08)**	**0.032**[b]
	Dairy (lactose-free)				
Fats and oils		0.09(-0.56–0.76)	0.76		
Alcohol		−0.003(-0.26–0.25)	0.98		
Carbonated drinks		−0.21(-0.7–0.3)	0.4		

[a] Multivariable analysis included age, sex pouchitis history and current pouchitis. Pouchitis history was negatively associated with nut intake [OR -0.4(-0.76 to −0.11); p = 0.10).
[b] Multivariable analysis included age, sex, dairy intolerance, and pouch indication. UC pouch indication was also significantly negatively associated with dairy intake [OR -2.09(-2.1 to −0.4); p = 0.04].

In the multivariable analysis, intolerance to dairy remained significantly negatively associated with reduced lactose-containing dairy intake after adjusting for age and sex. However, intolerance to nuts did not remain significantly negatively associated with nut intake ($p = 0.067$) after adjusting for pouchitis history ($p = 0.01$).

3.7. Correlation of dietary intake and pouchitis history

In order to explore whether a particular dietary intake was associated with the risk of pouchitis, univariable and multivariable regression analyses of the relationship of food serving consumption

Table 5
Uni- and multivariable regression analysis of nutrient intake and pouchitis history. Multivariable logistic regression analysis included covariates of priori importance (age, sex, pouch age, age at diagnosis of ulcerative colitis) and covariates with p < 0.1 in the univariable regression analysis.

			Univariable (OR, 95% CI)	*p*-value	Multivariable (OR, 95% CI)	*p*-value
Age, y			**0.94(0.89–0.98)**	**0.01**	0.89(0.79–1.01)	0.084
Age UC dx, y			**0.93(0.88–0.99)**	**0.02**	1.012(0.89–1.15)	0.86
Pouch Age, y			0.97(0.88–1.07)	0.57		
Sex			0.49(0.11–2.1)	0.34	0.05(0.002–1.6)	0.09
Macronutrients						
Energy Kcal/day			1(0.99–1.11)	0.75		NA
Carbohydrates	Total g/d		0.97(0.993–1.008)	0.96		
	Sugar g/d		0.99(0.98–1.007)	0.46		
	Starch g/d		1.012(0.99–1.03)	0.2		
Fat	Total g/d		0.99(0.98–1.02)	0.88		
	Saturated fat g/d		1.012(0.97–1.06)	0.6		
	Monounsaturated fat g/d		0.98(0.95–1.02)	0.4		
	Polyunsaturated fat g/d		0.99(0.87–1.1)	0.89		
Proteins g/d			1.001(0.98–1.02)	0.92		
Fibres	**Total Fibre g/day**		**0.96(0.91–1005)**	**0.08**	**0.68(0.51–0.92)**	**0.013**
	Resistant starch g/d		1.2(0.76–1.86)	0.43	2.4(0.7–7.7)	0.12
FODMAPs	**Oligo-saccharides**	**Total g/d**	1.03(0.76–1.4)	0.83	**5.5(1.04–29.1)**	**0.045**
		Fructans g/d	1.04(0.74–1.46)	0.82		
		GOS g/d	1.03(0.34–3.1)	0.95		
	Lactose g/d		0.99(0.95–1.05)	0.95		
	Excess fructose g/d		0.90(0.74–1.09)	0.28		
	Polyols g/d		**0.8(0.71–1.01)**	**0.06**	1.1(0.71–1.9)	0.5
Macronutrients						
Vitamins	Vitamin A mg/d		1(0.99–1)	0.2		
	Vitamin C mg/d		**0.99(0.99–1.001)**	**0.12**	1(0.98–1.01)	0.98
	Thiamine mg/d		1.2(0.70–2.1)	0.49		
	Riboflavin mg/d		1.1(0.72–1.76)	0.58		
	Niacin mg/d		1.01(0.95–1.08)	0.67		
	Folate mg/d		**1(0.99–1)**	**0.06**	1(0.99–1.02)	0.38
Minerals	Sodium mg/d		1.001(1.001–1.002)	0.16		
	Potassium mg/d		1(0.99–1)	0.27		
	Iron mg/d		0.997(0.992–1.002)	0.5		
	Magnesium mg/d		0.997(0.992–1.002)	0.24		
	Zinc mg/d		0.99(0.83–1.14)	0.74		
	Calcium mg/d		1(0.998–1.001)	0.6		
	Phosphorous g/d		1(0.999–1.001)	0.4		

or nutrient intake with a history of pouchitis. As shown in Supplementary Table 6, univariable analysis identified several factors, but, on multivariable analysis, none of the demographic factors or food groups were significantly associated with pouchitis history. In contrast, multivariable regression analysis of nutrient intake identified that total dietary fibre intake was negatively associated with pouchitis history [OR 0.68 (95% CI 0.51–0.92); $p = 0.013$], and that intake of oligosaccharides were positively associated with pouchitis history (5.5 (1.04–29.1); p = 0.45 (Table 5); Multivariable analysis of the relationship of total DGI and DGI variety scores with a history of pouchitis showed no significant association.

4. Discussion

Patients and health professionals alike request information on what dietary advice should be given to patients after an ileal pouch is created. Understanding patients' dietary intolerances, estimated intake and whole-diet quality, and their relationships to pouch phenotype and behaviour provide important insights. In this detailed evaluation of 58 patients with long-standing ileal pouches, several key findings were made. Perceived dietary intolerances were reported in 8 out of 10 patients independently of the pouch phenotype and behaviour. Despite this, whole-diet quality was good overall, with limited saturated fat intake, although it was poor for variety and for cereal intake. Phenotype-associated differences in dietary intake were mainly restricted to pouchitis where a history of pouchitis was associated with lower intake of fruits and nuts, and current pouchitis was associated with lower intake of nuts and lean meat and alternatives. Nutritional intake of most macro- and micronutrients was met in the majority of patients, but intake of core food groups only met recommended intake in the minority of patients.

The first key findings were that dietary intolerance to several food groups were frequently reported and that these were largely independent of sex or pouch phenotype and behaviour. Not surprisingly, the food groups with the highest intolerance rates (fruits, vegetables and dairy products, spicy foods and alcoholic beverages) and the symptoms they most commonly induced were similar to those reported in other studies on pouch patients [1,28,29] and in those with ulcerative colitis per se [30]. A common denominator in these food groups is their FODMAP content, which, by virtue of their ability to distend the small bowel via an increase in its water content, the increased liquid volume delivered to the pouch and their subsequent fermentation in the pouch with gas-induced distension, may lead to looser bowel actions, urgency, abdominal pain and flatulence [31–33]. Of particular interest are mono- and disaccharide FODMAPs that exert a larger osmotic effect than the oligosaccharides, which, by virtue of their being non-digestible, primarily cause issues via their fermentation [34]. Hypolactasia is common in patients with UC and hence pouches [34,35], but it is interesting that few of the patients (10%) ate less than 4 g/d, above which lactose maldigestion occurs [36]. Malabsorption of a high dose of pure fructose (35 g) was also frequently found in patients with UC [31], but whether that has clinical implications when much lower amounts of fructose in excess of glucose are consumed in a normal diet is contentious [37]. Spicy foods contain capsaicin and are often eaten in the presence of onions and other high FODMAP ingredients (such as in curries) and how they induce perianal burning is easy to understand (they 'burn twice!). The effects of chilli and alcohol might also relate to their ability to increase small intestinal transit [38,39].

Poor diet quality scores have previously been associated with increased disease risk in other clinical settings and reduced quality of life [40]. Hence, assessing diet quality using the DGI enables comparisons to be made with that exhibited by Australian adults in general, even though we used two fewer categories than the full DGI. Compared with the overall DGI score of 79 out of a total 130 from the 2011–2013 cross-sectional Australian Health Survey [25], a mean score of 79 out of a total 110 from the pouch patients is be considered favourable since a higher score relative to the total score represents better dietary quality. The total score was also comparable to another cohort of Australian adults (n = 3468; aged 55–65 years) [40]. Furthermore, component scores that were rated highly (moderating alcohol and unsaturated fat), showed poor compliance rates (cereals, total lean meat and alternatives and food variety), and were the least compliant (intake of discretionary foods), mimicked the patterns in that Australian cohort [40]. Intake of nutrients was comparable to those of healthy Australian adults in the CNAQ validation cohort [15] in terms of energy, carbohydrate, fat, protein and dietary fibre, although the intake of lactose and excess fructose were lower [15]. Similar intakes of energy, protein, carbohydrates, fat and dietary fibre were reported from the 80 UC pouch patients from Israel [41]. These findings highlight the notion that habitual dietary intake and patterns are more likely to be shaped by the dietary habits of adults in their society or household than their own dietary intolerances or by the presence of a pouch itself.

When dietary intake of symptomatic and asymptomatic pouch patients was compared, there were no differences in intake of lactose or excess fructose. A third of the symptomatic group did not have pouchitis (total PDAI < 7) and were categorised as irritable pouch syndrome, which is consistent with studies reporting a prevalence of irritable pouch syndrome in symptomatic pouch patients of 18%–43% [42,43]. In irritable bowel syndrome, malabsorption of lactose, excess fructose and other FODMAPs has been linked to symptoms of diarrhoea, flatulence and abdominal discomfort [44]. In patients with ileoanal pouches, an open-labelled dietary study showed high rates of fructose malabsorption (7/8) and lactose malabsorption (4/8), and that a low FODMAP diet reduced the frequency of pouch emptying by 50% ($p < 0.001$) [14]. Hence, it is possible that some of the symptomatic patients had lactose and/or fructose malabsorption and would benefit from a lactose-reduced diet. In this cohort, the difference in daily lactose intake between symptomatic and asymptomatic patients was marginal. In a double blind randomised controlled study on 13 participants with lactose maldigestion with intact colons, symptoms of lactose intolerance were more likely with doses of ≥12 g, but the proportion did not increase with a dose of 20 g. Furthermore, dose differences of <6 g resulted in similar symptom response [36]. Therefore, the higher PDAI in the symptomatic pouch group is potentially due to the lack of meaningful differences in lactose and excess fructose intake despite a higher likelihood of symptomatic patients having irritable pouch syndrome, and lactose and/or fructose malabsorption.

Another key finding from this study was that patients with a history of pouchitis had a lower intake of fruits and nuts compared with those without pouchitis despite no difference in intolerance rates. Other studies have similarly shown a lower intake of fruits in patients with a history of pouchitis [41]. Whether low fruit intake is a cause or a consequence of pouchitis cannot be determined from this cross-sectional study. It is possible that patients with a history of pouchitis link symptoms of pouchitis (loose stools) with foods that induce similar symptoms. Consequently, they are more likely to develop stronger negative food-related emotions and to avoid certain foods in an attempt to improve pouch-related symptoms. This could also explain the lower intake of lactose, excess fructose, fat, and lean meat and alternatives including nuts in those with current pouchitis. Such themes of negative food-related emotions and dietary restrictions to control symptoms were shown in a study

of 28 inflammatory bowel disease patients that included ileoanal pouch patients [45]. Furthermore, in this study, patients with a history of pouchitis were more likely to seek dietary advice from non-dietetic sources. Most of the available information online is centred on dietary advice in the first 12 months following stoma closure with a focus on a low fibre diet. In addition, the quality of online diet information for pouch patients varies greatly, risking exposing patients to potentially misleading information [46].

On the other hand, reduced intake of fruits and/or nuts could be a cause of pouchitis. In a study by Godny et al., a cohort of pouch patients with an initial healthy pouch had higher rates of pouchitis at one year if they had fewer than 1.45 servings of fruits daily [12]. In a subsequent study, greater adherence to Mediterranean diet was associated with higher dietary fibre intake and lower rates of developing pouchitis [13]. Multivariable logistic regression analysis in the current study highlights the importance of defining what is included or excluded in the assessment of fibre intake. Thus, intake of dietary fibre that did not include oligosaccharides (as is the case with most databases) was associated negatively with a history of pouchitis, of FODMAP oligosaccharides was associated positively with a history of pouchitis, and of resistant starch (inaccurately assessed by the CNAQ) had no significant association. Increased intake of fermentable fibres - whether rapidly or more slowly fermented, whether oligosaccharides, non-starch polysaccharides or resistant starch, or by modification of the diet or supplementation – may be associated with changes in the microbiome and metabolome [47]. The major changes in the microbiome include an increased relative abundance of *Bifidobacteria* and decreased relative abundance of *B. wadsworthia*, a sulphate-reducing genus [7,48]. The impact of these on health are at present more emotive than based upon actual health outcomes [49]. The major changes on microbial metabolic output include increased production and concentration of SCFA, consequent fall in luminal pH and reduction in protein fermentation as evident by reduced production of branched chain fatty acids (BCFA) [49,50]. The impact of changes in intestinal microenvironment are double-edged and concentration dependent. SCFA, especially butyrate is a key energy substrate with potential anti-inflammatory effects, but at high concentrations, it is potentially toxic to the epithelium as shown in animal and *in vitro* human studies [49,51]. Similarly H_2S at low concentrations may help stabilise mucus layers, prevent adherence of microbiota biofilm to the epithelium and resolve inflammation, but at higher concentrations, can cause mucus disruption and inflammation [52]. In animal studies, low caecal luminal pH is associated with impaired barrier function and increased epithelial cell turnover [53,54]. The underlying mechanisms could explain the evidence, albeit weak in humans, that readily fermentable fibres consumed in doses that exceed those in the average diet induce subtle increases in colonic mucosal inflammation in the colon [49]. In ileoanal pouches, rapidly-fermentable carbohydrates such as oligosaccharides are ideal candidates to increase carbohydrate fermentation in the pouch due to their extensive and rapid fermentation. In a 3-week placebo-controlled cross-over study in healthy pouch patients, 83% of the supplemented fructan was fermented in the pouch compared with only 43% of supplemented resistant starch. Both fibres resulted in a trend for lowering faecal pH and increasing the faecal excretion of total SCFA [33]. Other readily fermentable fibres such as inulin have been shown to have similar effects on metabolome without meaningful impact on pouch inflammation [16,55]. Therefore, the rapid and extensive fermentability could explain the positive association oligosaccharides and pouchitis history in a dose-dependent manner.

If oligosaccharides or resistant starch are not the component in fibres that is protective, how can we explain the negative association of dietary fibres and pouchitis history. One possibility is that the effect is due to fibre components not assessed by the CNAQ. Pectin, for example, is abundant in certain fruits and can theoretically have beneficial effects on dysbiosis, mucosal immune system and epithelial barrier function [56,57]. Like other fermentable fibres, the effects could be concentration-dependent, but in an animal study, supplementation of 7.5% of inulin or fructan exacerbated colitis, whereas similar amount of pectin did not [58]. Another plausible explanation for the negative association with fruits and/or nuts may not be solely due to their fibre content, but the rich supply of micronutrients (minerals, vitamins, and polyphenols) that coexists with fibres in fruits [59]. These can also have a beneficial role via modulation of microbial community structure and/or anti-inflammatory effects [60,61]. This could not be fully assessed in this study as the CNAQ does not assess of all dietary components such as polyphenols.

There are clear practical messages from the current food compositional data that would assist in dietetic counselling of patients with an ileal pouch to improve diet in general, to improve or optimise pouch function, potentially to protect against pouchitis, and help them deal with potentially negative food-related emotions. First, dietetic counselling should focus on nutritional gaps in intake of core food groups, such as those identified in this study, to correct for poor food variety in this cohort. Secondly, targeted dietary advice for specific pouch phenotypes may also be applied but more research is required to verify such recommendations. FODMAP foods appear to be commonly poorly tolerated and may physiologically worsen pouch function [1,28,29]. Patients with a suboptimal pouch with function not due to pouchitis might benefit from a trial of low FODMAP diet as shown in the study by Croagh et al. [14] However, the reduction of small intestinal water content might comprise both flow of contents in those prone to adhesion-related small bowel obstruction [62], and adequate fermentation in the pouch, possibly increasing the risk of pouchitis [63]. Furthermore, given the high prevalence of lactose malabsorption, patients with suboptimal pouch function would benefit from a low lactose diet (<4 g/day) under supervision of a dietitian to ensure calcium intake is not further compromised. Thirdly, associations of risk of pouchitis with low fibre intake might suggest avoiding fibre (non-starch polysaccharide) restriction although there are no data upon which a target level of fibre intake can be recommended. It is important to remember that currently-recommended intake of fibre (30 g/d for men and 25 g/day for women) was designed for those with an intact large bowel [18]. Further, the risk of high fibre intake in those with recurrent adhesion-related small bowel obstruction must be considered in any recommendation. Because of the negative association of a fruit consumption with the development of pouchitis [12] and the perceived health benefits of fruits beyond the pouch issues, it seems reasonable to recommend a daily intake according to the dietary guidelines [22]. However, selection of fruits should to take heed of their FODMAP intake. Finally, it would be reasonable to explore patients' food-related emotions and consequent reduced intake of certain foods to avoid unnecessary dietary restrictions, as clearly demonstrated int the current study, and its impact on health-related quality of life [1].

This study had several key strengths. First, it included patients with UC and FAP whose pouch behaviour and phenotype were assessed using a rigorous methodology [16]. Secondly, dietary assessment was unique in that it comprehensively examined FODMAP intake, potentially important in the genesis of symptoms, diet quality, and the relationship between dietary intolerance and intake. Thirdly, assessment tools applied were commonly used and relevant in the Australian population.

However, several limitations should be acknowledged. First, the dietary methodologies used all have inherent weakness such as being prone to recall bias [64] and the issue of overestimation of

energy intake [15]. Secondly, the cross-sectional nature of this survey precludes the ability to reach cause-effect conclusions on the observed diet-pouchitis associations. Thirdly, two of the DGI components (total fluid and added salt) were not included as the data were not provided by the CNAQ, but as such did not affect conclusions from the findings. Fourthly, the numbers of patients studied were limiting in terms of the power of subanalyses to define phenotypic associations.

5. Conclusions

In conclusion, pouch patients have a high rate of dietary intolerances due to the physiologic impact dietary factors have on pouch function. Despite this, pouch patients' overall diet quality is similar to Australian adults with intact colons, and not significantly different across different pouch phenotypes. In symptomatic pouch patients, particularly those without current pouchitis, fructose and lactose malabsorption could partially explain symptoms when intake is above a tolerable cut-off value. Patients with a history of pouchitis may have anxiety about dietary intolerance, with a consequent reduction of intake in a few food groups and nutrients associated negatively with pouchitis risk and their health in general. Therefore, pouch patients troubled by symptoms or pouchitis, would benefit from dietary education centred around moderating FODMAP intake, trialling lactose restriction, and ensuring adequate fibre intake. More prospective, longitudinal studies are needed to determine whether dietary intake plays a key role in the pathogenesis of pouchitis and if so, which nutrients are responsible.

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Author contributions

ZSA, KML, MS, PRG and CKY conceived the concepts and designed the study.

ZSA: Designed the study, recruited participants, collected and analysed data for the study, wrote the manuscript.

KML: Designed the DGI, contributed to the analysis of data and critically revised the manuscript.

MPS: Designed the study and edited the manuscript.

LP, JA and FR contributed to the data categorisation and analysis.

PRG: Designed the study, critically revised the manuscript for important intellectual content.

CKY: Designed the study, involved in data collection and analysis, edited and critically revised the manuscript.

All authors approved of the final manuscript.

Conflicts of interest

Zaid S Ardalan has no disclosures to declare.

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KML: has no disclosures to declare.

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MPS: Has (i) received educational grants or research support – Ferring, Orphan, Gilead; and (ii) received speaker's fees – Janssen, Abbvie, Ferring, Takeda, Pfizer, Shire, and (iii) Serves as a consultant and advisory board member for Janssen, Takeda, Pfizer, Celgene, Abbvie, MSD, and Emerge Health.

PRG has served as consultant or advisory board member for Anatara, Atmo Biosciences, Immunic Therapeutics, Janssen, Novozymes, Falk Pharma and Takeda. He has received research grants for investigator-driven studies from Atmo Biosciences. He holds shares in Atmo Biosciences. He has published two educational/recipe books on the low FODMAP diet.

ZSA, PRG and CKY also work in a department that financially benefits from the sales of a digital application and booklets on the low FODMAP diet. Funds raised contribute to research of the Department of Gastroenterology and to the University. No author receives personal remuneration.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clnu.2023.07.023.

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