**Conclusions:** The natural history of CD in elderly patients diagnosed over the age of 70 yr seems to be milder than that in those diagnosed between 60 and 70 yr. This needs to be taken into account when establishing therapeutic strategies.

## P665
## Familial aggregation of inflammatory bowel disease on the Faroe Islands: a Faroese inflammatory bowel disease cohort study

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**Background:** Over the last 2 decades, the incidence of inflammatory bowel disease (IBD) has increased dramatically on the Faroe Islands[1], an archipelago located in the North Atlantic Ocean populated by an endogamous population, and is today the highest in the world.2 This steep increase in IBD is mainly caused by UC, as the incidence of CD has remained stable over the last 5 decades. The reasons for these observations are unknown. In this study we aimed to investigate the genetic contribution component in IBD on the Faroe Islands.

**Methods:** The Faroese IBD cohort is a population-based, nationwide cohort of all patients diagnosed with Crohn's disease (CD),

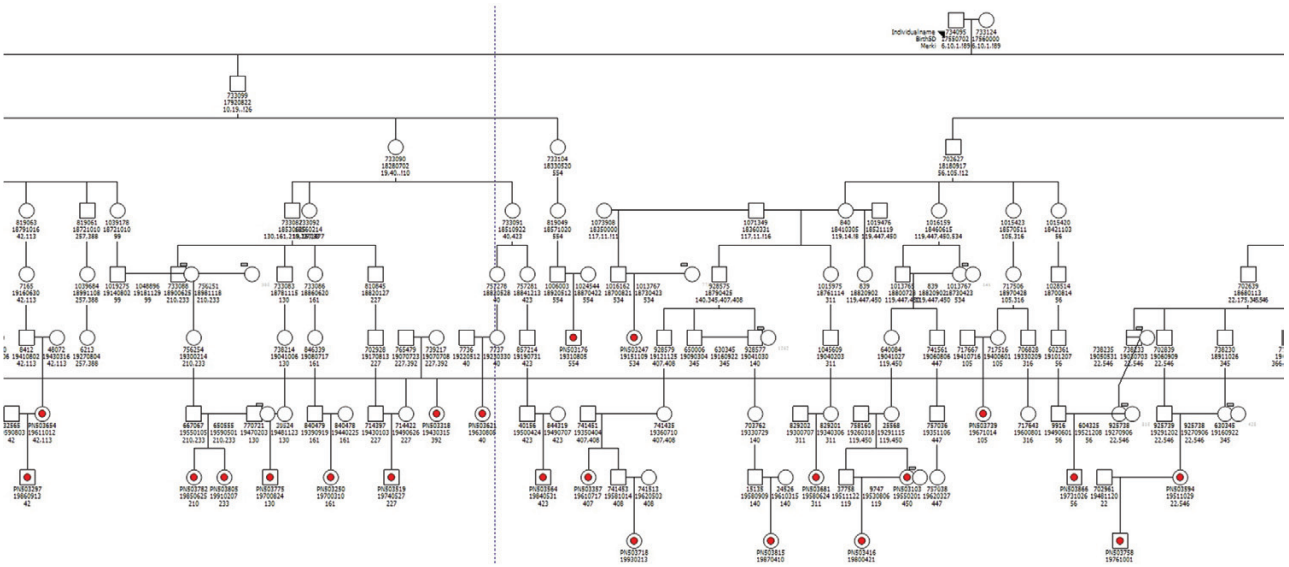

**Figure 1.** An ancestral pedigree of a family with high prevalence of UC patients (red dots).

**Table 1** Patient characteristics

| | Ulcerative colitis | Crohn's disease |
|---|---|---|
| Average age at onset, yr | 41 (0–86) | 41 (0–86) |
| Females | 47% | 46% |
| Smoking status | Current 20%, former 29%, never 51% | Current 36%, former 18%, never 46% |
| Extent (UC) | E1 21%, E2 39%, E3 40% | |
| Behaviour (CD) | | B1 65%, B2 25%, B3 8%<br>Perianal 2% |
| Location (CD) | | L1 22%, L2 65%, L3 10%, L4<br>(+/- L1-L3) 3% |

**Table 2** Relative risks (RR) of kinship for IBD patients and controls

| Family relation | Ulcerative colitis RR | Controls – ulcerative colitis (RR CI93%) | x times higher risk for UC vs upper bound in controls | Crohn's disease RR | Controls – Crohn's disease (RR CI 93%) | x times higher risk for CD vs upper bound in controls |
|---|---|---|---|---|---|---|
| 1st-degree relatives | | | | | | |
| Sibling | 2.74 | 1.46–2.08 | 1.3 | 0.99 | 0.00–0.57 | 1.7 |
| Child | 2.09 | 0.81–1.88 | 1.1 | 0.46 | 0.00–0.46 | 1.0 |
| 2nd-degree relatives | | | | | | |
| Uncle/aunt | 1.14 | 0.72–0.79 | 1.8 | 0.09 | 0.00–0.28 | 0.3 |
| Niece/nephew | 1.27 | 0.98–1.14 | 1.1 | 0.12 | 0.00–0.42 | 0.3 |
| Grandchild | 0.99 | 0.20–0.79 | 1.3 | 0.55 | 0.00–0.00 | 0.0 |
| 3rd-degree relatives | | | | | | |
| First cousins | 0.90 | 0.94–1.18 | 0.8 | 0,07 | 0.22–0.52 | 0.1 |

ulcerative colitis (UC), and IBD unclassified (IBDU) according to the Copenhagen diagnostic criteria from 1960 until 2014. The relationship amongst CD and UC patients was studied using a unique computerised nationwide genealogy registry covering pedigrees from the whole Faroese population from approximately 1 650. Relative risk (RR) of kinship for patients were compared with controls (simulated as if they were probands and shown as CI 93%). Controls were matched for age, gender, and approximate sib-ship size. Pedigrees for control subjects were randomly and reiteratively sampled from the Faroese genealogic database.

**Results:** Pedigrees (ie, Figure 1) were available for the whole cohort consisting of 664 incident IBD patients, 113 with CD, 417 with UC, and 134 with IBDU. Patient characteristics are shown in Table 1. The RR of kinship in UC and CD patients compared with controls are shown in Table 2. Overall, UC patients were more closely related than controls were (RR for UC was higher than upper CI 93% limit for controls), whereas no hereditary component in CD patients was found.

**Conclusions:** In this population-based, nationwide cohort of Faroese IBD patients a hereditary component was found for UC but not for CD patients. Further analyses on the effect of the genetic contribution on disease course, as well as genetic and microbial analysis, are ongoing.

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## P666
## Fruit consumption may protect against the development of intestinal inflammation via modification of microbial composition

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**Background:** Patients with ulcerative colitis (UC) undergoing proctocolectomy with ileal pouch-anal anastomosis commonly develop pouchitis. As pouchitis is inflammation developing in a previously normal small bowel. We hypothesise that it may represent the small intestinal inflammation characterising Crohn's disease (CD). CD pathogenesis involves environmental factors. We thus asked whether diet and the microbiome had a role in pouch inflammation.

**Methods:** Patients recruited at the Comprehensive Pouch Clinic were prospectively followed-up. Pouch behaviour was determined clinically and defined as normal pouch (NP) or pouchitis. All patients completed food-frequency questionnaires (FFQs). Faecal samples were collected and analysed for microbial composition (16S rRNA gene pyrosequencing). Microbial diversity was calculated using Shannon diversity index. *P*- values were corrected for multiple comparisons using false discovery rate (FDR < 0.1).

**Results:** In total, 172 pouch patients (89 [52%] females, with average age 44.9 ± 14 years and mean time since ileostomy closure 9.1 [range 0–30.4] years) were recruited. At the beginning of follow-up, 39 (22.6%) patients had NP. Within 1 year of follow-up, 5 (12.8%) of these developed pouchitis. Higher (> 1.45 servings/day), compared with lower fruit consumption was associated with significantly less development of pouchitis (3.8% vs 30.8%, respectively, log-rank test, $p = 0.03$). Faecal microbial analysis was performed in 81 patients (NP [$n = 22$], pouchitis [$n = 53$], and familial adenomatous polyposis [$n = 7$]). Fruit consumption correlated with microbial diversity (r = 0.37, $p = 0.001$), and with the abundance of several microbial groups. After adjustment for pouch behaviour and use of antibiotics, fruit consumption remained positively correlated with *Faecalibacterium* (r = 0.27, $p = 0.01$), *Lachnospira* (r = 0.31, $p = 0.005$), and 2 un-annotated genera from the *Lachnospiraceae* and *Ruminococcaceae* families (r = 0.24 $p = 0.03$; r = 0.28, $p = 0.01$, respectively). Significant decrease in fruit consumption was noticed in 10 patients who developed active disease over time ($\Delta = -1.4 \pm 1.7$ s/d, $p = 0.02$). Corresponding decrease in microbial diversity was noticed, as well ($\Delta = -0.6 \pm 1.2$, $p = 0.17$).

**Conclusions:** Fruit consumption of pouch patients significantly modified microbial composition, favouring expansion of Firmicutes, specifically *Faecalibacterium*, *Lachnospiraceae*, and *Ruminococcaceae*. Significant decrease in fruit consumption was associated with the development of active disease and with a decrease in microbial diversity. Thus, fruit consumption may be protective against intestinal inflammation, possibly by altering microbial composition. Therefore, dietary intervention may contribute to prevention of intestinal inflammation.

## P667
## The clinical features of patients with newly diagnosed moderate-to-severe ulcerative colitis in Korea: a population-based inception cohort study (the MOSAIK cohort)

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