

Abstract OC-119 Table 1 Relative quantity of inflammatory genes from SDD patients pre and post treatment with Placebo (P) or Mesalazine (M) (Median [inter-quartile range])

Median (IQR) Genes	P group			p Value	M group		
	Pre	Post			Pre	Post	p Value
NOD2	0.8938 (0.76–1.57)	1.021 (0.60–1.55)		0.6632	1.359 (0.79–1.96)	0.5925 (0.40–0.94)	0.0002
PAR2	1.841 (1.40–2.34)	1.571 (1.20–2.08)		0.4080	1.944 (1.49–3.37)	1.056 (0.77–1.25)	0.0007
TNF α	0.6611 (0.47–1.05)	0.6341 (0.34–1.15)		0.2977	0.9170 (0.61–1.40)	0.4435 (0.36–0.69)	0.0034
IL1B	0.7416 (0.47–1.40)	1.092 (0.37–1.49)		0.2575	0.9633 (0.37–1.96)	0.6220 (0.37–1.01)	0.0024
TLR4	1.377 (0.94–2.49)	1.465 (1.05–1.94)		0.3604	1.537 (1.01–1.96)	0.8932 (0.70–1.52)	0.0479
TLR9	0.4506 (0.34–0.73)	0.5264 (0.30–0.68)		0.4488	0.7991 (0.39–1.17)	0.4976 (0.32–0.64)	0.0215
MYD88	1.765 (1.51–2.66)	1.678 (1.22–2.44)		0.6013	1.780 (1.15–2.99)	1.134 (0.90–1.76)	0.0105

Methods Patients with confirmed SDD underwent an unprepared flexible sigmoidoscopy and biopsies at baseline and after 12 weeks treatment, completing diaries of pain and bowel habit. They were randomised to receive 3 g per day of mesalazine (M) or identical placebo (P) for 12 weeks with follow-up visits at 2 & 4 weeks. RNA from sigmoid biopsies was analysed using a custom made gene card. Gene expression and changes in symptoms were assessed between baseline and final visits using Wilcoxon signed-rank test.

Results 43 volunteers were recruited (F:M; 24:19), but 11 withdrew during the study resulting in 18 and 14 participants in the P and M groups respectively. M significantly reduced important inflammatory and pain genes including those involved in response to bacterial ligands (Abstract OC-119 table 1), changes not seen with P. M but not P significantly reduced the duration of abdominal pain (Median (range) (hrs/day) P group Pre 1.0 (0–5), Post 0.65 (0–4) p0.1919; M Group Pre 3.0 (0–20), Post 0.125 (0–5.5) p0.0413).

Conclusion This pilot study suggests that mesalazine significantly alters many of the pathways mediating immune activation by bacteria thought to contribute to pain in SDD and may provide benefit for patients. However further larger studies are required to confirm these mechanisms and efficacy of mesalazine in this group.

Competing interests J Smith grant/research support from: Wellcome Trust Training Fellowship, Conflict with: £17 000 Gift from Dr Falk Pharma for manufacture of medication for the study, D Humes: None declared, K Garsed: None declared, C Lam: None declared, A Zaitoun: None declared, A Bennett: None declared, J Scholefield: None declared, R Spiller: None declared.

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OC-120 THE "NUTRITION SUPPORT PYRAMID"—COMPOSITION AND TRENDS IN A REGIONAL PAEDIATRIC COHORT FROM SOUTH EAST SCOTLAND

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Introduction Within nutrition support (NS) there is a spectrum of complexity ranging from oral supplements via enteral tube feeding (ETF) to parenteral nutrition (PN) for intestinal failure (IF). Nutrition support teams (NST) are involved with those children with chronic or complex nutrition needs on home ETF (HETF) and home PN (HPN). There is no current robust data available on the prevalence of children on each level of what we newly describe as the nutrition support pyramid (NSP).

Aims To introduce the concept of the NSP and to describe the composition and temporal trends in the NSP in a UK regional paediatric cohort.

Methods We performed a retrospective cohort study (database/clinical note review) of all children (<16 yrs) in SE Scotland requiring NS over a 7-year period (2004–2010). We divided all

children having NS into levels of the NSP, which comprises four levels of nutrition support; a 5th level is children with IF who have required transplantation (Tx) to achieve intestinal adaptation. The NSP base comprises children receiving HETF, the second level is children with severe upper GI dysmotility requiring jejunal HETF, the penultimate level is children with type II IF (prolonged hospital PN), and the top level is type III IF (children who receive HPN). Poisson regression models and Fisher's exact testing were then used to compare the period prevalence (per calendar year) of children <16 yrs requiring NS and the proportions of each level of the NSP between the two epochs of 2004–2006 and 2008–2010.

Results There were a total of 780 NS episodes in 702 children (51% male); 69 (10%) had multiple episodes of NS. Median (IQR) age at commencing NS was 1.0 (0.2–4.7) yrs. There was a significant increase in the period prevalence of children requiring NS between the two epochs (p=0.004). However, although the number of children requiring HETF (level 1) rose from 372 to 422 between the two epochs, there was a non-significant change in the shape of the NSP (determined by the relative size of each level) between the two epochs (p=0.736). During the entire study period a total of 715 NS episodes were located on the base level (HETF); 31 on the second level (jejunal HETF); 21 on the penultimate level (type II IF); and 14 on the top level (type III IF), with four children requiring Tx to achieve enteral autonomy.

Conclusion We have introduced the concept of a NSP, not previously described, and shown that the NSP has significantly increased in size (number of children requiring NS) without a significant change in the shape (relative distribution of complexity). The NSP is a simple tool which allows us to show a significant increase in NST workload without any decrease in complexity over just 7 years.

Competing interests None declared.

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OC-121 FIBROBLAST ACTIVATION IN THE TUMOUR MICROENVIRONMENT PROMOTES TUMOUR CELL INVASION AND RESISTANCE TO CHEMOTHERAPY IN OESOPHAGEAL ADENOCARCINOMA

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Introduction Stromal and other non-malignant cells have the potential to undergo modifications that can synergistically create a supportive microenvironment for tumour growth, invasion and metastasis. Oesophageal adenocarcinoma (EAC) is characterised by early invasion, leading to metastatic disease and therefore only 20% of patients are suitable for treatment with curative intent. Cancer associated fibroblasts (CAFs) have an activated, myofibroblastic phenotype and have been recognised as mediators of tumour