



Abstract O55 Figure 1

hour *versus* 30.0 [17.0] l.hour with no lactulose, $p=0.0078$) but had no effect on T1AC even after 36 hours treatment (0.74 [0.4]s *versus* 0.64 [0.28]s, $p=0.72$). Ondansetron did not significantly alter SBWC or T1AC, either after a meal alone or when combined with repeated doses of lactulose. Gut transit (median [IQR]), was unchanged by ondansetron compared to placebo (1.7 [0.5–5.8] versus 1.4 [0.5–6], $p=0.63$).

Conclusions Although lactulose increases SBWC by an amount close to that predicted by its osmotic load (130 ml) this did not significantly alter colonic water content. This may be due to its known rapid metabolism and suggests its laxative effect may be due to the stimulatory effects of products of fermentation. Ondansetron did not alter postprandial intestinal water nor reduce the effect of lactulose suggesting that its anti-diarrhoeal effect may be primarily due to altered colonic motility.

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RISK FACTORS FOR PROXIMAL COLON CANCER: HOW INFORMATIVE ARE POLYP FINDINGS IN DETERMINING FUTURE RISK?

¹Rhea Harewood*, ¹Kate Wooldrage, ²James Kinross, ³Christian von Wagner, ¹Amanda J Cross. ¹Cancer Screening and Prevention Research Group (CSPRG), Imperial College London, London, UK; ²Department of Surgery and Cancer, Imperial College London, London, UK; ³Research Department of Behavioural Science and Health, University College London, London, UK

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Introduction Early detection and removal of premalignant colorectal polyps with a high potential to progress to invasive cancer is important for incidence reduction. However, there is evidence that cancers in the proximal colon tend to be detected later than other subsites resulting in more advanced stage at diagnosis and lower survival. This study examined which polyp characteristics were independently associated with proximal colon cancer incidence.

Methods Data were used from the All Adenomas study, which examined endoscopy and associated pathology data on ~30,000 individuals with at least one adenoma identified. Eligible participants underwent colonoscopy between 1984 and 2010 in one of 17 UK hospitals. Polyp characteristics at baseline colonoscopy, including number, size, histology, grade and location were obtained from the database. Cox regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for incidence of proximal colon adenocarcinoma. Time since baseline colonoscopy was used as the underlying time scale. HRs were mutually adjusted for

polyp characteristics in addition to demographic- and colonoscopy-related confounders.

Results Of the 27,812 (42.4% female) participants included in the analysis, 227 (0.82%) developed proximal colon cancer during a median follow-up of 9 years. Cumulative incidence over 15 years was 1.4% (95% CI: 1.2% - 1.6%). Proximal colon cancer incidence was higher among participants with ≥ 1 adenoma in the proximal colon at baseline, either solely or in addition to distal adenomas, compared to patients with only distal adenomas (HR 1.95, 95% CI: 1.46 - 2.62). The risk was also higher among those with ≥ 3 adenomas compared to those with < 3 adenomas at baseline (HR 1.47, 95% CI: 1.04 - 2.08) and those with adenomas ≥ 10 mm compared to those with adenomas < 10 mm (HR 1.47, 95% CI: 1.07 - 2.01). Neither adenoma histology nor grade were independently associated with the outcome.

Conclusions Adenoma location, number and size are informative of subsequent proximal colon cancer. This study provides evidence needed to identify individuals at high risk for proximal colon cancer who would require post-polypectomy colonoscopy surveillance for the early detection and removal of cancer and precancerous lesions in this subsite.

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THE VALUE OF GERMLINE MUTATION TESTING IN SERRATED POLYPOSIS SYNDROME

¹Sujata Biswas*, ¹Michael Johnson, ¹Adam Bailey, ¹Elizabeth Bird-Lieberman, ¹Simon Leedham, ²Peter Risby, ²Joyce Solomons, ¹James East. ¹Translational Gastroenterology Unit, Oxford NIHR Biomedical Research Centre, University of Oxford, Oxford, UK, UK; ²Oxford Centre for Genomic Medicine, Nuffield Orthopaedic Centre, Oxford University Hospitals NHS Foundation Trust, UK

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Introduction Serrated Polyposis Syndrome (SPS) is now known to be the commonest polyposis syndrome. Previous analyses for germline mutations have shown no consistent positive findings¹. To exclude other polyposis syndromes, new 2019 BSG guidelines² advise gene panel testing if: the patient is under 50 years of age; if there are multiple affected individuals within a family; or if there is dysplasia within any of the polyps.

Methods A database of patients with SPS according to the WHO 2019 criteria³ was established at the Oxford University Hospitals NHS Trust. Data collection began in 2010 and in total there are 192 SPS patients. The results of any patients sent for genetic testing were analysed.

Results Out of 192 patients, 76 underwent genetic testing. The majority were tested for a hereditary colorectal cancer panel including MUTYH, APC, PTEN, SMAD4, BMP1A, STK11 and Lynch syndrome mismatch repair genes. Of these, 14 had a positive genetic test result. Table 1 characterises patient with positive results.

Conclusions 7% (14/192) of SPS patients were affected by heterozygous germline mutations, higher than in previous series¹, including previously unreported associations with CHEK2 and POLD1. This led to a change in management for patients or their families in seven cases. Only 57% (8/14) of these patients would have been recommended for gene panel testing in the current BSG guidelines². Detection of germline mutations could have significant impact on risk assessment and clinical management, including advice on extra-colonic surveillance in patients and their family members.