**Conclusions** CTC was useful guide to direct further management of the patients as regards any endoscopy being requested at all and type being mainly simple sigmoidoscopy in most of the cases. Combination of CTC with faecal occult blood in BSP could be a reasonable alternative for patient's assessment especially those elderly group with multiple comorbidities and challenging endoscopy.

## OWE-25 PATIENTS WITH MULTIPLE ADENOMAS IN BOWEL CANCER SCREENING PROGRAM ARE NOT REFERRED FOR GENETIC TESTING

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**Introduction** Approximately one in twenty cases of colorectal cancer (CRC) are associated with germline mutations that confer higher susceptibility to the disease. Guidelines recommend that patients with ten or more adenomas be referred for genetic testing[1], with evidence that suggests >9% of these patients with 1–9 adenomas have a highly penetrant CRC predisposition syndrome[2]. The primary aim of this study was to quantify patients with ten or more adenomas in the West London (WL) and North Central London (NCL) Bowel Cancer Screening program (BCSP) centres. The secondary aim was to determine what proportion of these multiple adenoma patients were referred for genetic screening.

**Methods** A retrospective cross-sectional study was performed of patients who underwent colonoscopy following a positive faecal occult blood test (FOBt) as part of the WL and NCL BCSPs between May 2007 &amp June 2018. All polyps were examined histologically and only confirmed adenomas were included. Clinicopathological data including age and gender was recorded from BCSP patient records. Referrals to regional clinical genetics services were ascertained. Statistical analysis was performed in Graphpad Prism.

**Results** 11,337 patients underwent colonoscopy following positive FOBt and 5,650 (49.8%) had 1 or more adenomas. 107 patients (0.94%) had 10 or more adenomas. The proportion of patients with 10 or more adenomas was higher in NCL BCSP (1.1%) than in WL BCSP (0.7%) ($p=0.02$; $\chi 2$ test). 42 patients presented with 10 or more metachronous adenomas at the index colonoscopy or following an early follow up procedure; the remaining 65 patients undergoing excision of a total of 10 or more metachronous adenomas after subsequent surveillance colonoscopy. An accurate family history was not routinely collected in this population. Of the 107 patients with 10 or more adenomas, only 3 (2.8%) were referred to a clinical genetics service.

**Conclusions** In two London BCSP centres, patients with ten or more metachronous adenomas are an uncommon finding. Despite guidelines advising genetic testing in this group, referral rates are low. A referral pathway and management strategies should be established to address this patient population.

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## OWE-26 INFLAMMATORY RESPONSES IN PATIENTS WITH COLORECTAL CANCER AND IRON DEFICIENCY ANAEMIATREATED WITH ORAL IRON

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**Introduction** Colorectal cancer (CRC), a major cause of cancer-related mortality, is associated with iron deficiency anaemia (IDA) and is typically treated with oral iron. Iron is an essential nutrient element that is required for a diverse range of biological activities including the immune function (1). However, studies in animal and cell line models have shown that accumulation of iron in the gut can generate toxic reactive oxygen species and proinflammatory cytokines that results in tissue damage and CRC progression (2). We studied, for the first time in humans, the effect of oral (OI) versus intravenous iron (IVI) treatment on iron distribution, immune cell infiltration and systemic cytokine profile in CRC patients with IDA.

**Methods** Patients with CRC and IDA received oral-ferrous sulphate (n=20) or intravenous ferric carboxymaltose (n=20) for 2 weeks. Tissue was analysed for iron loading using Prussian-Blue staining. Tissue cytokine and immune cell distribution was determined using immunohistochemistry. Systemic cytokine production was evaluated by multiplex-cytokine assays.

**Results** Iron expression was distributed to the epithelium of tumour tissues in OI treatment ($p<0.001$) and to the tumour stroma and the macrophages in IVI treatment ($p<0.01$). OI treatment significantly increased the production of the systemic pro-inflammatory cytokines; IL-6 ($p<0.02$), IL-12 ($p<0.03$), IL-17 ($p<0.04$), GM-CSF ($p<0.02$) and IFN-γ ($p<0.03$). Moreover, OI treatment decreased the anti-inflammatory cytokine IL-4 ($p<0.01$) and IVI increased the anti-inflammatory cytokine IL-10 ($p<0.003$) production in the serum. Immunoreactivity of IL-12 levels was higher in tissues from patients treated with OI and distributed to the epithelium. However, there were no significant differences in immunoreactivity of tissue IL-10 between the treatment groups. Inflammatory cell (lymphocytes and granulocytes) infiltration was higher in the OI treatment group.

**Conclusions** Intravenous iron is compartmentalised to non-epithelial cells indicating that iron is potentially less bio-available to the tumour cells. Oral iron treatment induces inflammatory immune responses both systemically and within the tumour microenvironment. Further functional studies are ongoing to evaluate the mucosal immune response to differential iron loading.

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