PWE-082 ARE SCREEN TESTED FOBT +VE SUBJECTS FOUND TO HAVE COLORECTAL CANCER ASYMPTOMATIC?

doi:10.1136/gutjnl-2012-302514d.82

H A Griffiths,* I Fretwell, C Nickerson, L Coleman. *NHS Bowel Cancer Screening Programme, Sheffield, UK*

Introduction The National Bowel Cancer Screening Programme (NHSBCSP) aims to reduce mortality from bowel cancer in a defined population, by detecting cancer in asymptomatic individuals between 60 and 74 years. Current Government strategies are aimed at raising awareness of symptoms, to bring survival from cancer in England up to the average for Europe.[1] This study aims to identify whether individuals with a FOBt +ve screening test are asymptomatic?

Methods Data were extracted from the Bowel Cancer Screening System (BCSS) on FOBt +ve individuals diagnosed with cancer from commencement of the programme in 2006 to December 2011. This included all symptoms (one or more) reported at colonoscopy pre assessment, as captured by BCSS. The same data were extracted on a control group of FOBt +ve, colonoscopy negative individuals. In addition to examining trends in reported symptoms, data sets were also interrogated to look at those significant symptoms that if reported prior to the screening episode should have triggered a cancer two week wait (2WW) referral.[2]

Results 10 211 patient episodes with cancer (Male=6825, Female=3837) and 30 249 without cancer (Male=14 991, Female=15 259) were included in the analysis. Symptom data could not be verified with regard to recency, frequency or severity of symptoms or whether previously reported. Anxiety caused by a +ve screening test may itself heighten awareness of some symptoms however, the percentage reporting significant symptoms[i].

Conclusion Although the NHSBCSP aims to detect asymptomatic cases, a large proportion of individuals were symptomatic at the time of screening, some were eligible for referral under the DH guidance for high risk symptoms of colorectal cancer. The Government's drive to increase awareness of symptoms is necessary to prompt individuals to seek medical advice at an earlier stage. Standardising how data are captured at pre assessment will improve the quality and usefulness of the data strengthening future analysis on the impact of awareness campaigns on screening.

Abstract PWE-082 Table 1 Frequency of reported symptoms

Symptom	Cancer (%)	Normal (%)
None	926 (9.07)	4377 (14.47)
Frequency	2711 (26.55)	5083 (16.80)
Diarrhoea	2245 (21.99)	7049 (23.30)
Rectal bleeding	5636 (55.20)	13 266 (43.86)
Rectal bleeding without anal irritation*	3654 (35.78)	7534 (24.91)
Urgency	2551 (24.98)	7667 (25.35)
Mucus	1571 (1.54)	3751 (12.40)
Weight loss	1333 (13.05)	3801 (12.57)
Frequency and diarrhoea and urgency*	606 (5.93)	1321 (4.37)

*Significant symptoms.

Competing interests None declared.

[i]Making them eligible for referral as a cancer 2WW is higher in those FOBt +ve individuals found to have cancer at screening colonoscopy than in those with FOBt +ve negative colonoscopy.

REFERENCES

1. http://www.dh.gov.uk/en/Healthcare/Cancer/Earlydiagnosis/index.htm#jumpTo1 (accessed 10 Jan 2012).
2. **NICE**. *Referral Guidelines for Suspected Cancer*. London: NICE, 2011.

PWE-083 BUCCAL CELLS AS A POTENTIAL SURROGATE FOR DNA METHYLATION BIOMARKER TO IDENTIFY THOSE AT INCREASED COLORECTAL CANCER RISK

doi:10.1136/gutjnl-2012-302514d.83

[1,2]H Staley,* [2]D M Bradburn, [1]J C Mathers. [1]*Human Nutrition Research Centre, Institute for Ageing and Health, Newcastle University, Newcastle, UK;* [2]*Department of General Surgery, Wansbeck General Hospital, Northumberland, UK*

Introduction Colorectal cancer (CRC) screening programmes are an opportunity to alter the survival of patients with early CRC. Cells from the alimentary tract can be obtained from both the mouth and large bowel. If biomarkers of CRC risk could be identified in buccal cells, such assays could be more convenient and acceptable than those requiring rectal biopsies. One potential CRC risk biomarker is methylation of the WNT-related gene encoding the Secreted Frizzled Related Protein 4 (*SFRP 4*).

Methods DNA was extracted from macroscopically normal rectal biopsies and matched buccal cell swabs from volunteers at a relatively lower (healthy volunteers) and higher (patients with adenomatous polyps) CRC risk in the BORICC Study. Methylation of *SFRP4* was quantified by Pyrosequencing.

Results *SFRP4* promotor methylation was quantified in rectal biopsies and matched buccal cells in 233 lower and 89 higher CRC risk participants. For rectal biopsies, *SFRP4* promotor methylation was significantly (p=0.001) higher in those with polyps than in healthy controls. However, for buccal cells, the reverse was observed with significantly (p<0.001) higher *SFRP4* promotor methylation in the healthy controls. At CpG sites 1 and 4 only, *SFRP4* methylation was correlated significantly (p=0.001 and p=0.041 respectively) between the two patient groups.

Conclusion *SFRP4* promotor methylation in rectal biopsies is not the same as that in matched buccal cells. However, *SFRP4* methylation was significantly different between patient groups at each site, providing encouragement for further studies of the utility of buccal cells as a surrogate tissue for identification of those at increased CRC risk.

Competing interests None declared.

PWE-084 SECRETED FRIZZLED RELATED PROTEIN 4 (SFRP4) AS AN EPIGENETIC BIOMARKER OF COLORECTAL CANCER RISK

doi:10.1136/gutjnl-2012-302514d.84

[1,2]H Staley,* [2]D M Bradburn, [1]J C Mathers. [1]*Human Nutrition Research Centre, Institute for Ageing and Health, Newcastle, UK;* [2]*Department of General Surgery, Wansbeck General Hospital, Northumberland, UK*

Introduction Colorectal cancer (CRC) is the 3rd most common cancer in the UK. There is a lack of robust biomarkers of CRC risk which could act as surrogate endpoints for studies investigating modifiers of CRC risk. Epigenetic changes (aberrant DNA methylation marks) in the WNT-related Secreted Frizzled Related Protein 4 (*SFRP4*), a gene whose expression is down-regulated early in CRC development, may be a potential biomarker of CRC risk. Such epigenetic changes occur early in tumorigenesis and may contribute causally to CRC progression. In addition, they may respond to diet and other lifestyle determinants of CRC risk.

Methods DNA was extracted from macroscopically normal mucosal biopsies from the rectum of volunteers at a relatively lower (healthy volunteers) and higher (patients with adenomatous polyps) CRC risk in the BORICC Study. Methylation of *SFRP4* was quantified by Pyrosequencing and data were analysed by analysis of variance.

Results *SFRP4* promoter methylation was quantified in 253 biopsies from lower and 96 biopsies from higher CRC risk participants.