**GIH-19**  Evaluation of an automated immunochemical faecal occult blood test for colorectal neoplasia screening in a Chinese population

WM. Wong1, S.K. Lam1, K.L. Cheung1, T.S.M. Tong, P. Rozen2, G.P. Young3, K.W. Chu1, J. Ho1, W.L. Law1, H.M. Tung1, H.K. Choi1, Y.M. Lee1, K.C. Lai1, W.H.C. Hu1, C.K. Chan1, M.F. Yuen1, B.C.Y. Wong1
Departments of 1Medicine and 1Surgery, University of Hong Kong, 2Department of Gastroenterology, Tel Aviv University, Israel, 3Department of Gastroenterology and Hepatology, Flinders University of South Australia, Adelaide, Australia.

**Background and Aim:** Most commercial fecal occult blood tests (FOBT) used for colorectal cancer (CRC) screening in western populations are guaiac based, developed manually, subjective and sensitive to dietary components. Preliminary studies showed their unsuitability for screening in Chinese population. The aim of this study is to evaluate the performance characteristics of a human hemoglobin specific automated immunochemical FOBT, Magstream 1000/Hem Sp, in a Chinese population referred for colonoscopy.

**Methods:** 250 consecutive patients referred for colonoscopy and met the study inclusion criteria took samples for the immunochemical FOBT, without dietary restrictions, from two successive stool specimens. Tests were developed by an automated machine having an adjustable sensitivity threshold. The sensitivity, specificity and positive predictive value for detecting colorectal adenomas and cancers were calculated according to manufacturer’s instruction, using a range of sensitivity levels.

**Results:** The sensitivity, specificity and positive predictive value for detecting significant colorectal neoplasia (adenomas $\geq 1.0$ cm and cancers) at the optimal threshold level were 62%, 93% and 44% respectively. The test is easy to use and not dependent on operator experience.

**Conclusion:** An automated immunochemical FOBT is a robust, convenient and useful tool for colorectal cancer screening in Chinese population.

---

**GIH-20**  Cytogenetic and fluorescence in situ hybridisation characterisation of esophageal carcinomas showed clonal chromosomal aberrations and $CCND1$ amplification

Y Jin1,2,3, C Jin1, S Law2, K-M Chu2, H Zhang2, B Strombeck3, PW Yuen2, Y-L Kwong1.
Departments of 1Medicine and 2Surgery, Queen Mary Hospital University of Hong Kong, Queen Mary Hospital, Hong Kong, 3Department of Clinical Genetics, University Hospital, Lund, Sweden.

**Introduction:** The genetic mechanisms underlying the development of esophageal carcinomas are not well defined. The aim of the study is to characterize the cytogenetic aberrations that may be important for esophageal squamous cell carcinoma (SCC) tumorigenesis.

**Material and Methods:** Tumour samples and their surrounding tissues of 6 patients with esophageal SCC were studied by cytogenetic and molecular cytogenetic techniques.

**Results:** Cytogenetic analyses of four squamous cell carcinomas (SCC) of the esophagus showed clonal chromosome aberrations involving numerous complex numerical and structural abnormalities. Chromosomal bands or regions preferentially involved were 11q13, 8q10, 21q10, 3p10-p11, 1p11-q11, 5p11-q11 and 14p11-q11. For the first time, recurrent aberrations were identified in esophageal SCC, including homogenous staining region (hsr), isochromosomes i(3q) and i(21q), and ring chromosome. Losses of chromosomal material dominated over gains. Recurrent imbalances included under-representation of 4p13-pter, 5q14-qter, 9p22-pter, 10p, 11p13-pter, 12p13-pter, 17p10-pter, 18p11-pter, 21p, and 22p; and over-representation of 1q25-qter, 3q, 7q, and 8q. Interestingly, hsr at different chromosomal regions occurred in three of four cases. With the application of fluorescence in situ hybridization (FISH) and multicolor COBRA FISH using specific DNA probes, it could be shown that in two cases, the hsr was derived from chromosome 11 material and that the amplicon included $CCND1$.

**Conclusion:** These observed chromosomal aberrations and imbalances provide important information for the further molecular genetic investigation of esophageal SCC. $CCND1$ might be an important target in 11q13 amplification, and that amplification of this gene might be crucial in the tumourigenesis of esophageal SCC.

**Acknowledgement:** This work was supported by the Kadoorie Charitable Foundation, and research grants from the University of Hong Kong, and the Swedish Cancer Society.