

Gastrointestinal endocrine cells and carcinoids

A histopathogenetic study with a comparison of silver-staining and immunohistochemical characteristics

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The principal aim of the present investigation was to study the histopathogenesis of mid-gut carcinoids, and, if possible, to obtain evidence for a not identical origin of appendiceal and other carcinoids of mid-gut type. Although carcinoids of the small-intestine, appendix and proximal colon usually are grouped together with the designation 'mid-gut' or 'classical' carcinoids, they are not in all respects identical, especially with regard to natural history. Further, the intestinal carcinoids mostly are considered to derive from the mucosal endocrine cells, but the origin of these tumours is still controversial. For this reason normal gastrointestinal endocrine cells and carcinoid tumours were characterized and compared by means of different silver-staining techniques, formalin-induced fluorescence (FIF) technique of Falck-Hillarp, immunohistochemistry, newly developed sequential staining methods for demonstration of peptide hormone/biogenic amine-containing cells and electron microscopically. The major immunological neuroendocrine markers used were antisera/antibodies to serotonin, neuron-specific enolase (NSE) and S-100 protein. A comparative examination of one fundic gastric carcinoid and a sample of intestinal adenocarcinomas was also performed because, it is well documented that these two types of tumours derive from epithelial endocrine and non-endocrine cells of the gastrointestinal mucosa. The results of this study suggest that the histopathogenesis of appendiceal and small-intestinal carcinoids is not identical. Evidence is presented indicating that appendiceal carcinoids develop from subepithelial neuroendocrine cells, in the mucosal stroma and are possibly related to the peripheral nervous system, and that small-intestinal carcinoids, like the fundic gastric carcinoids, are histo-genetically related to the intraepithelial endocrine cells of the gastrointestinal mucosa. Moreover, by means of a methodological piece of innovation, it became possible to accurately identify detectable amounts of serotonin and gastrin in the same cell in the antral mucosa from a patient with atrophic gastritis and multiple fundic gastric carcinoids. The method is considered to be of value for the further exploration of the gastrointestinal endocrine cells and tumours.

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Epidemiology of polyps in the rectum and sigmoid colon

A study of adenomas and hyperplastic polyps in a population of men and women aged 50–59 years

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The feasibility and acceptance of an endoscopic population screening examination with flexible fibersigmoidoscopy was demonstrated.

Reproducibility of size and localization of polyps at repeated endoscopy was good. A shortening of the sigmoid colon was observed at withdrawal of the endoscope during colonoscopy but not at sigmoidoscopy. This had no influence on the recovery of polyps in the sigmoid bowel segment.

Endoscopic population screening and histological examination of removed polyps showed that, in comparison to autopsy studies, the prevalence of adenomas and hyperplastic polyps was lower than expected for women, but not for men. This may be consistent with an overestimation of prevalence rates in younger age groups when based on autopsy studies. The prevalence of

adenomas was higher in men than in women, whereas women showed a trend towards larger and more dysplastic adenomas—consistent with previous autopsy results.

Two years follow-up results indicated both growth and regression of polyps—growth and occurrence of new lesions seemed to be more common in the colon and proximal rectum, whereas regression was seen more often in the distal rectum. This indicated that the present model for population screening with initial examination only of the distal parts of the large bowel may not automatically be transferrable to an older age group.

DNA distribution and LD and G-6-PD activity within colorectal polyps showed a limited correlation to polyp size or degree of dysplasia. Prospective studies will be needed to further elucidate the prognostic value of these parameters in regard to risk of colorectal carcinoma.

Breath methane cannot be used as an adjuvant method in the identification of individuals with adenomas or development of dysplasia within a group of persons with adenomas.

A discriminant analysis model used on multivariate clinical information failed to differentiate satisfactorily between polyp-bearing and polyp-free individuals unless combined with information from the dietary registrations. Consequently, population screening of this type (with an aim towards secondary prevention) ought to be based on the single most important parameter—age, with a prospect of finding asymptomatic colorectal carcinoma in 1% in the 50–59-year age group.

A double blind model for registration of diet in persons with and without polyps was in agreement with epidemiological and case/control studies of patients with colorectal cancer, giving further evidence for the significance of dietary factors in colorectal carcinogenesis.

The present results, based on epidemiological, endoscopic and histological data, lend further support to the adenomacarcinoma sequence theory.

Our results add to the accumulated knowledge during the past decades, indicating factors of importance for primary and secondary prevention of colorectal cancer.

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ERRATUM

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Röösér B., Pettersson H. and Alvegård T.: Growth rate of pulmonary metastases from soft tissue sarcoma.

On page 189, second column, line 16–17: 'between 6 and 99 months' should read 'between 1 and 99 months'. In the Table on page 190 it should be '(months)' instead of '(days)' in the last column.