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GASTRIN IS A COMPLETE CARCINOGEN FOR THE OXYNTIC MUCOSA

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The steroid hormones, particularly sex hormones, play an important role in carcinogenesis. Surgical or drug treatment to reduce the effect of sex hormones on cancers have been used for decades. Peptide hormones, on the other hand, have not had such a position. Moreover, most experts have claimed that even sex hormones are not complete carcinogens, only having a growth promoting effect in cells where an initial hit (mutation) has started the process of malignancy. When long-term drug inhibition of gastric acidity induced malignant tumors originating from the gastrin target cell, the ECL cell, in rodents, the question of whether gastrin was a carcinogen became of utmost importance. Subsequent studies have shown that every condition with long-term hypergastrinemia in whatever species develop ECL derived tumors. We started to study the role of gastrin in gastric carcinogenesis around 1985 after we first had shown that gastrin stimulated gastric acid secretion solely by releasing histamine from the ECL cell. Parallel to the stimulation of histamine release, gastrin stimulates ECL cell proliferation by the same receptor and same concentration dependence. On the background of problems in distinguishing between adenocarcinomas and neuroendocrine tumors both in man and rodents, we started to examine human gastric carcinomas with regard to ECL cell differentiation with the help of the most sensitive and specific methods available at the time of the study. We detected ECL cell differentiation in many of the carcinomas, and especially those of the diffuse type according to Lauren, and among them those belonging to the signet ring subgroup. Recently we could show expression gastrin receptor on hyperplastic and neoplastic ECL cells including a proportion of tumors originally classified as adenocarcinomas. The gastrin antagonist netazepide reduces ECL cell hyperplasia and can eradicate gastric neuroendocrine tumours (NETs) (carcinoids). Netazepide may also have an effect on the growth of gastric carcinomas with gastrin receptor as well. The recent epidemiological studies showing that proton pump inhibitors predispose to gastric cancer further incriminate gastrin in gastric carcinogenesis.

Biography

Helge L Waldum fulfilled his MD at University of Oslo in 1971 with excellence (reported to the King). He became Specialist in Internal Medicine and Gastroenterology at the University of Tromsø in 1980, the same year as he defended his first thesis on Studies on Group I Pepsinogens and Secretin. Since then he has worked at the University Hospital of Trondheim, except for one year where he stayed in Paris at Hospital Bichat where he in 1993 defended his second thesis on Enterochromaffinelike cell (ECL) - a key cell in the gastric mucosa with the mention, Very Honorable with Felicitatons. He has supervised twenty candidates for PhD and published about 487 papers and 100 scientific letters. He was the Head of Department of Gastroenterology and Liver Diseases at Trondheim University Hospital for more than twenty years. In 2011 he was appointed "Knight of 1st degree of the order of Saint Olav" by the Norwegian king for his translational research. He is presently serving as Editor-in-Chief, Scandinavian Journal of Gastroenterology.

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