Gastric carcinogenesis may now be understood by correct identification of cell of origin based on improved tumour classification and physiology

Helge L. Waldum

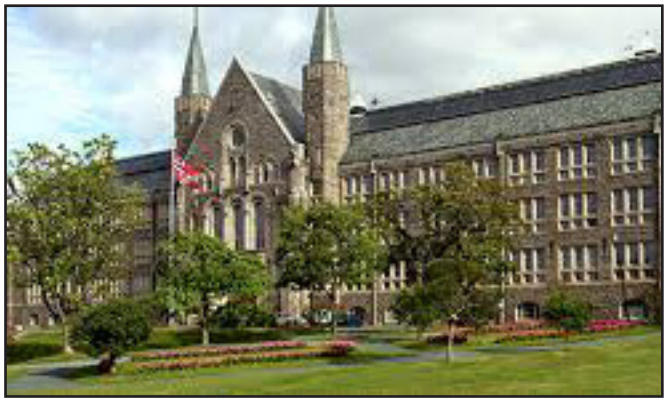
Norwegian University of Science and Technology, Norway

Abstract:

The ethology of gastric cancer like for most other malignancies has been incompletely understood. In the late forties it was recognized that gastric cancer was associated to reduced acid secretion, and in the fifties that gastric cancer seldom was found without accompanying gastritis. With the description of the central role of Helicobacter pylori (Hp) in gastritis , it was soon realized that Hp was the major cause of gastric cancer . However, the mechanism for this carcinogenic effect was not found. A major indication of how Hp predisposes to gastric cancer was given when Uemura described that Hp gastritis predisposes to gastric cancer first after having induced oxyntic atrophy. Moreover, it became evident that the carcinogenic process did continue even after Hp eradication by drugs or loss of Hp infection due to anacidity. Thus, in patients with Hp atrophic gastritis cancer can develop decades after loss of Hp clearly indicating that the carcinogenic effect of Hp was not a direct one. Furthermore, autoimmune atrophic gastritis and another condition with anacidity due to an inborn mutation of one of the genes coding for the proton pump (ATP4) (no inflammation) both predispose to cancer. All these conditions predisposing to gastric cancer thus have one thing in common, hypoacidity which necessarily leads to hypergastrinemia. By applying all the presently available methods we have shown that an important proportion of gastric carcinomas is neuroendocrine and more specifically ECL cell derived. The ECL cell is the target cell for gastrin. The therapeutic consequences of this knowledge are to perform Hp eradication before development of oxyntic atrophy, and in those with already established oxyntic atrophy as well as in those with autoimmune gastritis or genetic hypoacidity, treat with a gastrin antagonist. Moreover, inducing hypergastrinemia by inhibitors of gastric acid secretion should be reduces.

Biography:

Helge Waldum became M.D. in 1971 at the age of 25 years (University of Oslo, Norway, with a grade reported to the King) and completed two Ph.D.s (University of Tromsø, Norway, 1980 and Université de Paris, France, 1993) and is a specialist in Internal Medicine and Gastroenterology, 1980). He is a Professor at Norwegian University of Science and Technology, Trondheim Norway from 1986 and Head of Department of Gastroenterology and Hepatology, University Hospital of Trondheim, Norway for more than 20 years. He has published more than 350 papers and supervised 18 candidates to Ph.D. Research related to regulation of gastric acid secretion, gastrin and in its target cell, the ECL cell. The role of the ECL cell in physiology, patophysiology and carcinogenesis and the classification of gastric carcinomas have been of particular interest



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