Comment. Gastric intestinal metaplasia and smoking

(Comentario. Metaplasia intestinal gástrica y tabaco)

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Although the pathogenesis of intestinal metaplasia remains unknown[1], it is well recognized that chronic gastritis and intestinal metaplasia are precursor conditions of gastric cancer. In intestinal metaplasia, the original gastric mucosa is replaced by glandular structures bearing an intestinal phenotype. Intestinal metaplasia is one of several steps in the prolonged precancerous process of gastric cancer, mostly of the intestinal histological type. There are 2 other types of intestinal metaplasia: the complite (tpe I) and the incomplite (tpe III) type. The incomplite type is more frequently associated with dysplasia and early carcinoma and is considered a more advanced stage of the process or of higher malignant potential[2].

On the other hand, there is enough evidence about the causal relationship between smoking and gastric cancer risk[3]. The study by Peleteiro and colleagues deals with a systematic review of prevalence of intestinal metaplasia among *Helicobacter pylori* infected individuals and the correlation with national tobacco availability by means of an ecological analysis[4].

Several case-control studies have shown the association between intestinal metaplasia and smoking. Although an ecological study is not the best appropiate epidemiological design to support the hypothesis of the role of smoking in the risk of intestinal metaplasia or in the progression from intestinal metaplasia to gastric cancer, there are no reviews published on the prevalence of intestinal metaplasia. This study provides a large, detailed, and useful information about the prevalence of intestinal metaplasia in 29 countries of five continents. Nevertheless, caution should be taken when the prevalence of intestinal metaplasia between studies and countries. The population source (clinical- or population-based), the age and the sex of participants, as well as the number of biopsies specimens, and the biopsy site, may influence the prevalence of intestinal metaplasia and therefore the comparability of results from different studies.

Information of tobacco availability in each country, expressed as the adult apparent tobacco consumption in cigarettes per day, were obtained from the Tobacco and Health Report of the WHO or from the National Tobacco Information Online System database, which provide realiable information at the population level. Despite the importance of these results, it is necessary to stress that causal relationships can be only derived from individual exposure data from studies to come.

References

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