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Reply to Stachura

Dr. Stachura expresses doubts as to whether the risk index we propose would have any practical utility in surgical pathology. In response, we would first like to point out that this index was developed on the basis of routine gastric biopsies, our intention being to provide the physician awaiting the histopathological diagnosis of his endoscoped patient with prognostic information about the risk of gastric cancer associated with *H. pylori* gastritis (Meining et al. 1998). In addition, the index was intended to reduce the uncertainty as to which *H. pylori*-infected patients not suffering from peptic ulcer disease should be treated, with the aim of reducing the risk of gastric carcinoma (Meining et al. 1998). Dr. Stachura is, of course, right in remarking that the index is restricted only to patients currently infected with *H. pylori*. However, as is clear from the title of our paper and the Introduction and Discussion sections this was in fact what we intended; we made no attempt to establish a "general" gastric carcinoma risk index.

Dr. Stachura also expresses the opinion that "..patients with atrophic pangastritis and intestinal metaplasia are known to be in the higher risk group of gastric carcinoma anyway." We would point out here that high-risk atrophic gastritis, both in the Correa model [1] and as noted by Dr. Stachura himself, is always multifocal. It is thus obvious that the diagnosis of this type of gastritis on the basis of routine biopsies is associated with a high probability of sampling error, simply because the biopsy

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obtained by the endoscopist fails to include atrophic mucosa in the "bite" of the forceps. Considerably more information is provided by the inflammatory infiltrate induced by the *H. pylori* infection in the antrum and corpus, however, and this is always diffuse. Furthermore, even after the introduction of Dixon and Genta's "strict definition" of gastric mucosa atrophy [2], interobserver variability continues to be high – as very recently reported by Genta himself at the Congress of the AGA (New Orleans, May 1998). For these reasons, we believe that consideration of atrophy alone as a risk indicator for the development of gastric carcinoma is insufficient. Moreover, signet ring cell carcinoma is known to arise mostly in non-atrophic mucosa.

With regard to the third point of criticism, namely the choice of duodenal ulcer patients as controls, we refer to the discussion in our paper. This patient group was chosen advisedly, since such patients are known to have a reduced gastric carcinoma risk, reflecting the fact that gastritis of the duodenal ulcer phenotype has features associated with a reduced risk of carcinogenesis. Especially when the aim is to identify gastritis risk criteria it is, we believe, highly reasonable to compare patients having a high risk with those having a very low risk of developing gastric carcinoma.

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