Correspondence

CEA Family Gene Expression and Ki67 Staining in Hyperplastic Polyps versus in Tubular Adenomas

To the Editor-in-Chief:

In a recent issue of *The American Journal of Pathology*, Scholzel et al¹ reported immunohistochemical studies on carcinoembryonic antigen family members in normal colon and in colonic polyps. They found that CEACAM6 and CEACAM7 were differentially expressed in normal tissues and oppositely deregulated in hyperplastic polyps and early adenomas.

One of their findings was that Ki67 localization moves toward the upper part of the crypts in hyperproliferative polyps. This observation was correlated with loss of CEACAM7 expression and a broader expression zone of CEACAM6 at the apical surface and extending into the cytoplasm. Their Figure 3i shows Ki67 expression moving toward the upper part of the crypts in a tubular adenoma. In our studies on colonic polyps (unpublished), we observed identical changes. In hyperplastic polyps, however, proliferation as investigated by Ki67 staining was most intense at the base of the crypts and moved upward, to some extent, toward the apical surface, particularly in serrated adenomas. Even in those, Ki67 localization was never seen predominantly in the upper part of the crypts, which is characteristic of tubular adenomas.

We therefore believe that the immunohistochemical and molecular changes in hyperplastic polyps are different from those in tubular adenomas, and that CEA family gene expression and Ki67 staining should be correlated separately in hyperplastic polyps and tubular adenomas.

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CEACAM6 and CEACAM7 are differentially expressed in normal tissues and oppositely deregulated in hyperplastic colorectal polyps and early adenomas. *Am J Pathol* 2000, 156:595–605

Authors' Reply:

In response to the letter by Ruebner et al to our recent publication in *The American Journal of Pathology*, we would like to make the following comments:

Our studies on the expression of Ki67 and their comparison to expression of CEA family members were limited to only two adenomas, as mentioned in the Results section of our publication.¹ Unfortunately, we did not look at Ki67 expression in hyperplastic polyps. We hope that there was not a misinterpretation of our results, wherein we describe the expression pattern of Ki67 in a hyperproliferative polyp, but were referring to the tubular adenoma shown in Figure 3i and not in a hyperplastic polyp.

We apologize if this led to any misunderstandings. Nevertheless, based on the interesting observation by Ruebner et al that Ki67 expression is different in hyperplastic polyps compared to adenomas, we agree that a comparison with the expression of CEA family members should also be made in hyperplastic polyps. Such studies should then be applied to a larger number of polyps to allow a statistical evaluation. This could help our understanding of the *in vivo* functions of CEA family members during hyperplasia, shedding more light on the molecular differences between hyperplastic polyps and tubular adenomas. It might also reveal molecular variability between different hyperplastic polyps, as indicated by the differences already seen for CEACAM6 expression in the eight hyperplastic polyps that were investigated and reported in our publication (see Table 3).

We would welcome the opportunity to coordinate such analyses with the Sacramento group.

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References

1. Scholzel S, Zimmermann W, Schwarzkopf G, Grunert F, Rogaczewski B, Thompson J: Carcinoembryonic antigen family members

References

1. Scholzel S, Zimmermann W, Schwarzkopf G, Grunert F, Rogaczewski B, Thompson J: Carcinoembryonic antigen family members CEACAM6 and CEACAM7 are differentially expressed in normal tis-