## LETTER TO THE EDITOR

## Barrett's Esophagus and Antireflux Surgery: Wraps, **Rings**, and **HALOs**

## To the Editor:

e read with interest the article by Reveiller et al,<sup>1</sup> which was published in a recent issue of the Annals of Surgery. The genetic studies compared the effects of bile and acid on squamous and columnar epithelial cells of the esophagus, thus remodeling gastroesophageal reflux disease at the cellular level. The authors found that the major compounds of reflux, that is, bile and acid, affect the genetic program of esophageal epithelial cells.<sup>1</sup> Most importantly, bile acids inhibited the genetic program required for the differentiation of normal squamous epithelial cells. As a consequence, the bile acid induced genetic changes favoring the activation of the CDx2 pathway and mediating the development of premalignant Barrett's esophagus.<sup>1</sup> Thus it seems that the bile-milieu formats the cellular type toward Barrett's esophagus. Remains to be questioned the clinical relevance of these striking and important findings.

The data of the present study confirm that the elimination of bile reflux seems to be the prerequisite for successful protection from Barrett's esophagus.<sup>2</sup> In contrast to medical

therapy, effective antireflux surgery eliminates acid and bile reflux and normalizes bile acid, which induces tissue stress, and mediates the regression of Barrett's esophagus.2-4 In line with this notion, effective fundoplication potentiates the success of radiofrequency ablation of Barrett's esophagus.5

Perhaps the data of the present study will serve as the basis for the development of a new anti-Barrett's esophagus drug. At present, the combination of radiofrequency ablation and effective antireflux surgery represents promising treatment options for the effective and durable eradication of Barrett's esophagus.<sup>2-5</sup> Future studies will have to elucidate, if the early implementation of less invasive antireflux procedures (ie, laparoscopic sphincter augmentation) before the reflux-induced inactivation of the normal genetic program will contribute to prevent the development of Barrett's esophagus and esophageal adenocarcinoma.6,7

The authors are to be congratulated for this outstanding work and kindly asked to address the above considerations.

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