intact stomach', but this is well known and obvious because duodenogastric reflux is a physiological event, which takes place in all the subjects as well as in all the Hp pylori positive ones. In contrast, in my opinion, because of these methodological reasons the statement that 'data suggest that Hp pylori may induce DGR' is apodictical and needs to be proved by examining wider series and using more adequate methods.

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1 Keane FB, Dimagno EP, Malagelada JR. Duo-
denogastric reflux in humans: its relationship
to fasting antroduodenal motility and gastric,
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Schindbeck NE, Heinrich C, Staallard F,
Monforte MA, Maller-Lasser SA. Healthy
controls have as much bile reflux as gastric

Calcium and colorectal epithelial cell
proliferation

EDITOR.—There is still much debate whether
calcium can prevent colorectal cancer in
patients with an increased risk of the
development of such tumours. Calcium
intervention studies, using epithelial cell
proliferation as an intermediate end point,
have produced inconsistent results. Most
studies have focused only on the effect of
calcium on the rectal epithelium. Several open
uncontrolled studies have demonstrated a
reduction of rectal epithelial cell proliferation
during calcium supplementation, but small
placebo controlled studies are not as uniform
in their conclusions. Recently Weisgerber et
al (Gut 1996; 38: 396–402) considered this
aspect of sample size, as well as the fact that
studies were performed with biopsy specimens
from the rectum. With respect to the
small size of patients population, two
recent studies included a large number of
subjects. Bostick et al., performed a
randomised, double blinded study in sporadic
adenoma patients. Patients received placebo
(n=66), 1 g calcium/day (n=64) or 2 g
 calcium/day (n=63) for six months. Rectal
biopsy specimens were obtained at baseline,
and at one, two, and six months. In this study
no difference in proliferation was observed
between the three groups. However, calcium
normalised the distribution of proliferating
cells in the crypts, which is supposedly
beneficial with respect to cancer risk.
Rothstein et al (J Surg Oncology 1994; 60:
185–91) demonstrated a dose response
dependent increase in epidermal progenitor
cells in the crypts, which could be a
mechanism for fueling colorectal cancer.
Weisgerber et al (J Surg Oncology 1994;
60: 185–91) found a decreased 22% of
proliferating cells in the crypts.

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