



Figure 1 Time between abstract presentation and publication.

audit. However, previous similar studies from other societies have suggested that their publication rates vary by as little as 5% from year to year. Thus assessing one meeting may be adequate.<sup>6</sup> In conclusion, acceptance of abstracts by the BSG meeting suggests more than a 2 in 3 chance of subsequent full publication. This compares favourably with similar studies of other societies.

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### Leptin in the human stomach

EDITOR.—After the report in 1998 by Bado and colleagues<sup>1</sup> describing the presence of leptin in rat stomach, we have recently reported the first evidence of leptin in the stomach mucosa of humans.<sup>2</sup> It was shown that the cells in the lower half of the stomach glands were clearly immunoreactive for leptin, and both leptin mRNA and leptin protein in the human gastric epithelium were detected. Western blot analysis showed the presence of a 16 kDa band corresponding to leptin and a 19 kDa band which, as suggested for rats,<sup>1</sup> could represent a leptin precursor. It was also shown that secretory granules of chief cells contain this hormone, suggesting that gastric leptin could function in the short term system control of feeding behaviour and that it is secreted (probably together with

pepsinogen) in the stomach lumen by chief cells. Confirmation of these findings was reported by Sobhani and colleagues.<sup>3</sup> They also showed the presence of leptin receptor in stomach epithelium, suggesting a possible paracrine pathway for leptin. Stomach leptin levels seem to be higher in humans than in rats.<sup>1,3</sup>

Interestingly, Sobhani *et al* have also shown<sup>3</sup> that gastric leptin is simultaneously released into the blood and into the gastric juice by pentagastrin and secretin. They suggested that secretin has a direct effect on gastric chief cells, an idea based on the presence of secretin receptors on these cells<sup>4</sup> and on the efficacy of secretin in stimulating pepsinogen secretion.<sup>5</sup>

However, by immunoelectron microscopy we observed<sup>2</sup> the presence of leptin not only in chief cells but also in endocrine cells exhibiting a distinctive morphology in the basal portion of the gland. These cells showed secretory granules labelled with many leptin-gold particles.<sup>2</sup> Its ultrastructure corresponded to the P cell type.<sup>6,7</sup>

Thus secretory granules of both endocrine and chief cells contain leptin.<sup>2</sup> It is probably secreted in the stomach lumen by chief cells and into the stomach circulation by a special type of endocrine cell. The observation<sup>3</sup> that intravenous infusions of pentagastrin or secretin caused an increase in circulating leptin levels and leptin release into gastric juice is in keeping with both endocrine and exocrine secretory sources. They could function in the short term system to control feeding behaviour and in the gastrointestinal lumen to regulate the availability of nutrients acting in the sites where a non-degraded form of hormone would approach.

Our observation of much lower levels of leptin immunostaining in a patient under postprandial conditions compared with five fasted patients<sup>2</sup> is in agreement with a likely functional response of human stomach leptin to food intake. The effects of cholecystokinin in the rat<sup>1</sup> and of pentagastrin and secretin in humans<sup>3</sup> stimulating emptying of stomach leptin are all strong arguments for a short term satiety role of leptin. There is also the observation that leptin interacts synergistically with other short term satiety peptides.<sup>8</sup>

There is a need for further investigation in humans, with difficulties arising from ethical limitations. However, taken together, both articles<sup>2,3</sup> on leptin in the human stomach and the previous report in rats,<sup>1</sup> we can conclude that three important pathways (endocrine, exocrine, and autocrine) for the action of leptin are present in human stomach, where the

main physiological role for this hormone is foreseen.

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### Histological and genetic heterogeneity in synchronous hepatocellular carcinoma

EDITOR.—The recent paper by Sirivatanakorn *et al* (*Gut* 1999;45:761-5) focused once again on the unresolved question as to whether (i) hepatocellular carcinoma (HCC) in human liver develops from a single clone or from multiple parallel clones and (ii) among multiple tumour nodules present in many patients, the smaller lesions represent intrahepatic metastases or "de novo" cancers. The authors correctly acknowledge that "information on the clonal origin of tumours will influence management strategies for prevention of recurrence after operation". They used arbitrarily primed polymerase chain reaction (AP-PCR)<sup>1</sup> to compare the DNA fingerprint of HCCs and regenerative nodules (RNs) removed from 13 cirrhotic explant livers. They found considerable genomic heterogeneity in 54 HCCs and 31 RNs that were microdissected. No two nodules (either RNs or HCCs) had identical electrophoretic