

## CONFERENCE REPORT

# Meeting report of the British Society for Gastroenterology Meeting, March 2003

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The 2003 meeting of the British Society for Gastroenterology (BSG) was held at the ICC Birmingham. The range of subjects covered reflected the diversity of the speciality, and high quality clinical papers were in abundance. This contrasted with an interesting survey showing an apparent decline in the number of publications achieved by SpRs at the time of appointment to consultant grade from a median of 19 in 1993 to only five in 2001.<sup>1</sup> Our ability to collect large amounts of clinical data are ever improving and current clinical practice came under scrutiny from a number of national and regional surveys. The BSG-blue card surveillance scheme was used to survey mortality from inflammatory bowel disease.<sup>2</sup> This suggested that these diseases account for about 1% of all gastrointestinal deaths, and highlighted surgery and sepsis as key factors. The BSG also acted as a conduit for data collection for a national assessment of hepatitis C care in the UK.<sup>3</sup> The survey demonstrated regional variation in both healthcare providers and treatment practice, and estimated that only about 5–10% of hepatitis C virus infected individuals are in secondary care. The theme of service provision was developed in a prospective audit from Scotland of 3293 upper gastrointestinal cancers.<sup>4</sup> In this series, the provision of services in large specialist centres appear to have little impact on the outcome of the disease. The 53 hospitals surveyed did however experience relatively high mortality rates from both oesophageal (9–15%) and gastric (13–15%) cancers.

The diagnosis of upper gastrointestinal and mediastinal masses was reported to be facilitated by endoscopic ultrasound in combination with fine needle aspiration and trucut biopsy.<sup>5</sup> Following chemotherapy for oesophageal cancer, however, in one study no single imaging modality (computed tomography, endoscopic ultrasound, positron emission tomography) accurately predicted response to treatment.<sup>6</sup> In this case prevention is better than cure, and surgery is an alternative to lifelong acid suppression for those with gastro-oesophageal reflux. However, a Finnish study demonstrated a 1 in 200 risk of postoperative life threatening or fatal complications in the 11 974 patients surveyed.<sup>7</sup>

At the other end, the macroscopic findings of colonoscopy were suggested to have both negative and positive predictive value in the diagnosis of dysplasia in individuals with ulcerative colitis.<sup>8,9</sup> This may have implications for both the frequency of colonoscopic surveillance and the sites of sampling for histology. Conversely, when screening for colorectal polyps, severely dysplastic flat adenomas may require indigo carmine spray for accurate diagnosis and are frequently (95%) out of reach of the flexible sigmoidoscope.<sup>10</sup> The “bottom-up” model of colorectal adenoma morphogenesis was supported by the finding of very early adenomatous change involving entire crypts. In this elegant study, tissue from an XO/XY individual was used to demonstrate that there were no instances of XO or XY adenomatous tissue migrating down to involve crypts of the other genotype.<sup>11</sup> The genetic simplicity of this study contrasts with the genetic complexity of inflammatory bowel disease (IBD). In individuals stratified for known genetic variants associated with Crohn’s disease

(IBD5 and CARD15/NOD2), a genome wide scan of 228 IBD families suggested yet another susceptibility locus on chromosome 16.<sup>12</sup> A further layer of complexity is that due to the heterogeneity of these loci and their potential for epistatic interaction. Approximately 10% of individuals with one of the common CARD15/NOD2 mutations have a further coding mutation which in half of the cases could potentially compromise function of the mature protein.<sup>13</sup>

Despite the advent of novel therapies for IBD, azathioprine is still the subject of interesting research. Susceptibility to azathioprine toxicity is linked to thiopurine methyl transferase (TMPT) deficiency. One group described the successful use of very low (<5%) dose azathioprine in two individuals with IBD and absence of TPMT and also, at a dose of 1 mg/kg, in 20 of 26 individuals with low TMPT levels.<sup>14</sup> A pragmatic approach to individuals with normal TMPT levels and azathioprine intolerance using a stepwise reintroduction of therapy was also reported.<sup>15</sup>

Basic science was represented in the liver section, with a mechanistic study of stellate cell apoptosis during recovery from experimental liver fibrosis.<sup>16</sup> The authors suggested that one of the key steps might be cleavage of N-cadherin by matrix metalloproteinase 2. Successful recovery from fibrosis is accompanied by hepatocyte regeneration. The origin of these recovering cells could be from stem cells expressing the markers CD90 and CD34. These were shown to differentiate into cells expressing hepatic and biliary cytokeratins.<sup>17</sup> Clinical practice was also challenged with a recalculation of the variables that define mortality in acute alcoholic hepatitis.<sup>18</sup> The authors’ variables performed better than Maddrey’s discriminant function, and we await the prospective validation of their system.

Nutrition is a problem affecting hepatologist and luminal gastroenterologist alike. Simple approaches to access problems can be effective and one group had tackled the common problem of rapid closure of gastrostomy sites following PEG displacement by using relatively cheap vascular dilators to enlarge tracts to facilitate transcatheter replacement.<sup>19</sup> Ghrelin is one peptide regulating both appetite and body composition. Based on an interesting dataset, in which plasma ghrelin levels increased following *Helicobacter pylori* eradication, this was speculatively proposed to link *H pylori* infection, obesity, and gastro-oesophageal reflux disease.<sup>20</sup>

New tools may be required to unearth novel mechanisms of pathogenesis, and microarray technology was used to study the effects of naproxen on the stomach. Data on about 8000 genes was presented, suggesting that the drug causes apoptosis and may affect DNA integrity and repair.<sup>21</sup> Finally, we were posed the provocative question of how much are you willing to pay to cure one case of non-ulcer dyspepsia?<sup>22</sup> Answers on a postcard please.

**Abbreviations:** BSG, British Society for Gastroenterology; IBD, inflammatory bowel disease; TMPT, thiopurine methyl transferase.

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