now shown that recurrent germline mutations in *CDH1* are due to both independent mutations and common ancestry in families with hereditary diffuse gastric cancer.

Mutational analysis of *CDH1* was performed for 38 families with diffuse gastric cancer (accrued as part of an ongoing study at the British Columbia Cancer Agency), and the frequency and penetrance of *CDH1* mutations estimated. There were 26 families with two or more cases of gastric cancer including one person <50 years old with diffuse gastric cancer, and 12 families with diffuse gastric cancer in either one person <35 years old or several people >50 years old. Family members were classified as either carriers or noncarriers of *CDH1* mutations, and haplotype analysis was performed for seven families in this study and an additional seven previously reported families with the same mutations to determine whether recurring mutations were due to common ancestry.

The detection rate for *CDH1* mutations was 40%, with 13 *CDH1* mutations found in 15 of the families. Of these mutations, six were novel. Eight recurring mutations were identified, including two that occurred in more than one family. Haplotype analysis revealed that four mutations were associated with identical or near-identical haplotypes in more than one family, with one mutation seen in four families from communities in Newfoundland within a 100-mile radius. All carriers of this mutation shared a common haplotype, suggesting a founder effect.

Original article Kaurah P *et al.* (2007) Founder and recurrent *CDH1* mutations in families with hereditary diffuse gastric cancer. *JAMA* **297:** 2360–2372

Aspiration during endoscopy for physiological evaluation of pancreatic dysfunction

Measurement of exocrine secretion and 72-hour fecal fat output, the gold standard tests of pancreatic function, is time-consuming and difficult. O'Keefe *et al*. measured enzyme concentrations in pancreatic secretions sampled during upper gastrointestinal endoscopy as a rapid, alternative method for assessing pancreatic injury.

The study included 11 healthy controls and 22 patients diagnosed with severe ($n=7$), moderate ($n=5$) or mild ($n=10$) chronic pancreatitis according to the results of radiological imaging. Pancreatic secretion was stimulated with an enteral liquid diet and samples were collected by periampullary aspiration. A significant positive correlation was observed between trypsin concentrations in samples aspirated during endoscopy and trypsin concentrations secreted during a conventional 2-hour diet-stimulated pancreatic trypsin secretion and synthesis study. Trypsin secretion rates were significantly reduced in all pancreatitis patients and there was an imperfect correlation between rate reduction and the increasing severity of pancreatitis (i.e. 5 of the 7 patients with severe pancreatitis had trypsin secretion rates below the 90% reduction level associated with 'pancreatic insufficiency').

Measuring the trypsin concentration in samples obtained during endoscopy gave a more reliable indication of exocrine function than measuring the incorporation of isotope-labeled amino acids into secreted trypsin, with less variable results than a breath test measuring fat absorption. The authors suggest that measuring physiological stimulation of the pancreas by taking samples during endoscopy might, after further verification and refinement, become a useful tool for the diagnosis of chronic pancreatic dysfunction and the identification and prioritization of pancreatitis patients who would benefit from enzyme supplementation.

Original article O'Keefe SJD *et al.* (2007) Physiological evaluation of the severity of pancreatic exocrine dysfunction during endoscopy. *Pancreas* **35:** 30–36

Antiangiogenic treatment is effective against established chronic colitis in mice

Angiogenesis is part of the disease process in many inflammatory conditions, including Crohn's disease and ulcerative colitis. Danese *et al.*, therefore, hypothesized that antiangiogenic treatment might be effective in animal models of IBD. They investigated the effect of ATN-161 on experimental colitis in mice. ATN-161, a peptide that inhibits angiogenesis by binding proangiogenic integrin proteins, is currently in phase II trials as an anticancer treatment.

Having established that increased microvascular density parallels the development of colitis in mouse models of chronic and acute colitis, the researchers investigated the therapeutic and prophylactic effects of ATN-161. The peptide was an effective therapy