rapidly after visilizumab administration, but had fully recovered by 30 days in 26 of 30 patients evaluated. Adverse effects associated with cytokine release were common (88% overall) but mostly mild or moderate; no serious infectious complications occurred.

Visilizumab might, therefore, benefit patients with steroid-refractory ulcerative colitis and deserves further evaluation.

Original article Plevy S *et al.* (2007) A phase I study of visilizumab, a humanized anti-CD3 monoclonal antibody, in severe steroid-refractory ulcerative colitis. *Gastroenterology* **133:** 1414–1422

NF-κB activation promotes chronic inflammation and tumor progression in gastric cancer

NF-κB expression correlates with tumor progression in several different cancers, but its role in gastric carcinoma is not clear. Now, a recent report from Wu *et al.* confirms that *Helicobacter pylori* induced NF-κB activation is related to chronic inflammation and carcinogenesis in gastric cancer.

Analysis of gastric tissue samples found that nuclear NF-κB expression was more common in cancer samples than in non-malignant samples ($P = 0.0001$). *H. pylori* infection is a well-known risk factor for gastric carcinoma and, in this study, 81% of the infected cancer specimens were also positive for NF-κB. To confirm this observation, the authors infected human gastric carcinoma cells with *H. pylori in vitro* and, as expected, observed an increase in NF-κB activation. This was associated with increased expression of the inflammatory cytokines IL-1 and IL-8, and the pro-metastatic enzyme matrix metalloprotease (MMP)-9. Returning to the tissue samples, reverse transcription polymerase chain reaction analysis revealed an elevated expression of these factors in gastric cancer compared with normal tissue—offering a link between NF-κB activation, chronic inflammation and tumor progression.

To investigate the potential for clinical intervention, the authors added a specific NF-κB inhibitor, caffeic acid phenethyl ester (CAPE), to infected gastric carcinoma cells. CAPE treated cells were less viable, produced less IL-1 and IL-8 and demonstrated reduced invasive capacity in a Matrigel assay. CAPE, therefore, has promising therapeutic potential and warrants further investigation for the treatment of gastric cancer.

Original article Wu C-S *et al.* (2007) Predictive role of nuclear factor-κB activity in gastric cancer: a promising adjuvant approach with caffeic acid phenethyl ester. *J Clin Gastroenterol* **41:** 894–900

An important step towards the introduction of a vaccine for ETEC

Infection with enterotoxic *Escherichia coli* (ETEC) is a major cause of acute diarrhea and death from diarrheal disease in children in the developing world and a common cause of traveler's diarrhea, but there is no safe and effective vaccine. In the first licensed UK release of genetically modified oral vaccines, Daley *et al.* evaluated the immunogenicity and safety of three genetically modified strains of ETEC that express colonization factor antigen (CFA)/I or CFA/II—two of the most common antigens involved in ETEC infection.

The study included 98 healthy volunteers (aged 18–49 years; 40 men). Dose escalation studies were used to establish the highest tolerated dose of each vaccine; these doses were then used in placebo-comparison studies. The antibody-secreting cell response was measured by enzyme-linked immunospot, and antibodies in lymphocyte culture supernatants and serum antibodies (IgG or IgA) in peripheral blood were measured by enzyme-linked immunosorbent assay; whole gut lavage fluid (WGLF) was obtained to measure the mucosal immune response.

The vaccines induced significant mucosal IgA responses to CFA/I or CFA/II; for the CFA/I-expressing vaccine a dose–response relationship for mucosal IgA was detected in WGLF. The vaccines did not induce intestinal inflammation, as demonstrated by the lack of IL-6 or IL-8 secretion in WGLF in vaccine recipients, and they were also well tolerated.

The vaccines were immunogenic without impairing natural immune responses and have the potential to be included in a polyvalent oral vaccine for ETEC in the future.

Original article Daley A *et al.* (2007) Genetically modified enterotoxigenic *Escherichia coli* vaccines induce mucosal immune responses without inflammation. *Gut* **56:** 1550–1556