serial pH and Bilitec monitoring, with appropriate therapeutic modification to achieve predetermined end points. Patients who are randomised to laparoscopic fundoplication should have additional PPI therapy, as required, to achieve the same end points. Outcomes should be measured by standard serial endoscopic assessment and also by examination of a panel of molecular and cellular markers that is important in the pathogenesis of Barrett's adenocarcinoma.

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Improving hepatitis C services across the UK: response to a walk-in HCV testing service

The Department of Health (DH) estimates that approximately 0.4% of the UK population are chronically infected with hepatitis C virus (HCV) (that is, 200 000 people). As few as 10% of these individuals, who are at risk of end stage liver disease, are thought to be aware of their infection. Clearly action is required to identify and treat these patients with current drugs (pegylated interferons and ribavirin) that can cure over 50% of infected patients.

The UK voluntary sector have responded to the government identified need for more public information about HCV by organising a hepatitis C awareness day. We took advantage of the publicity around hepatitis C awareness day to assess the value of a walk-in HCV testing clinic.

Our clinic was held over four days (4–7 July 2003) and was widely publicised in the local press and television. Patients who wished to be tested were invited to attend a clinic in the Minor Injuries Unit at St Bartholomew's Hospital in the City of London. The clinic was manned between 8am until 11am for counselling and informed testing (hepatitis C antibody test and liver function tests). Results were available the next day and patients were informed in person 24 hours later.

Nineteen people attended and two were infected. One of these patients had been lost to follow up due to non-attendance at a local liver clinic 12 years ago.

Open access confidential hepatitis C testing clinics may play an important role in encouraging people to come forward for HCV testing and may facilitate public education about this important treatable infection. However, these clinics are labour intensive and, in our experience, unlikely to provide a cost effective solution to the identification of people with this treatable, sometimes fatal, infection.

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Conflict of interest: Dr Foster acts as a consultant to companies who sell drugs for the treatment of viral hepatitis and has received research funding from such companies. He has received fees from companies who market antiviral therapeutics.

Influence of mode of delivery on gut microbiota composition in seven year old children

Intestinal microbiota development begins immediately following birth.[1] The composition of the infant's evolving microbiota is initially defined by the mother, the source of the newborn's first microbial inoculum. Colonising bacteria rapidly adapt to breast milk and epithelial mucins as sources of nutrients.

The prevalence of caesarean section delivery in Western countries is increasing. Caesarean born babies are deprived of contact with the maternal/vaginal microbiota and the first exposure is characterised by a lack of strict anaerobes and the presence of facultative anaerobes such as *Clostridium* species.[2] Caesarean born infants have a more slowly diversifying microbiota, with differences reported from normally born infants, even after six months of age. Aberrancies in early microbiota acquisition can affect immunophysiological development with a heightened disease risk.[2 3] This study assessed microbiota composition in seven year old children and compared the respective effects of normal delivery and caesarean section.

In all, 60 seven year old children were randomly selected from Southwestern Finland, representing caesarean and vaginal deliveries.[4] The children were invited to attend a clinical examination, including skin prick testing and determination of serum total and antigen specific IgE antibodies. Perinatal data were derived from hospital medical records. Questionnaires were completed by the parents to verify a history of allergic symptoms.

Faecal samples were produced at clinical examination and frozen at −70°C for microbiota assessment. Faecal microbiota profiles were determined using the culture independent fluorescent in situ hybridisation method. Probes specific for bifidobacteria, lactobacilli/enterococci, bacteroides, clostridia, and total bacterial numbers were applied.[5] Written informed consent was obtained from parents and the study was approved by the ethics committee of the university.

Of the study population, 31 children had been delivered by caesarean section and 29 by vaginal delivery. At seven years of age, significantly higher numbers of clostridia were found in children delivered vaginally compared with caesarean born children ($p = 0.0055$) (table 1). No differences were observed in other faecal bacteria or total numbers of bacteria (table 1).

Children with asthma diagnosed by a physician ($n = 6$) had lower numbers of clostridia in their faecal specimens while healthy children ($n = 54$) had higher clostridial numbers.

Early colonisation guides subsequent microbiota development which may later impact on health, to the extent of predisposing some infants towards specific diseases.[3] Bifidobacteria are considered useful for health promotion. Reported effects are related to the individual ''balance'' of the gut microbiota and prevention of aberrancies within the gastrointestinal tract. Clostridia are generally considered harmful toxin producing species causing diarrhoea and food poisoning.[1]

Our results show that bifidobacterial levels in the faeces of cohort children were comparable at seven years of age, independent of the mode of delivery at birth, while numbers of clostridia were significantly higher in normally born children seven years after birth.

Differences in neonatal gut microbiota, in particular the balance between *Bifidobacterium* species and *Clostridium* species, have been reported to precede heightened production of antigen specific IgE antibodies, a hallmark of the atopic responder type.[1] Such differences may be related to external environmental

Table 1 Numbers of faecal bacteria (log 10 number of bacteria/g faeces) and total serum IgE concentration, and number of children with asthma or atopic dermatitis among seven year old children with a history of normal birth or caesarean section

Parameter (concn of specific microbe or total IgE)	Normally delivered	Caesarean born	p Value
Clostridia	9.29 (9.06–9.51)	8.83 (8.6–9.06)	0.0055
Bifidobacteria	10.32 (10.13–10.5)	10.29 (9.99–10.59)	0.87
Total bacteria	11.56 (11.46–11.7)	11.59 (11.5–11.68)	0.61
Lactobacilli/enterococci	9.07 (8.85–9.3)	9.05 (8.86–9.2)	0.85
Bacteroides	9.95 (9.67–10.24)	9.84 (9.52–10.17)	0.63
Total IgE	79 (16–255)	65 (25–160)	0.85

Values are median (interquartile range).

factors (for example, mode of delivery and early feeding practices). The results of this study, showing that clostridial numbers in normally born children seven years after delivery are significantly higher than in caesarean born children, demonstrate that abnormal development of the intestinal microbiota reported following caesarean section delivery may continue even beyond infancy. These findings call for further assessment of microbiota composition throughout childhood when dietary interventions may still offer a rational means of health improvement. It is of importance to characterise the optimal clostridial numbers and species composition at different ages following normal and caesarean delivery.

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Crohn's ileitis after liver transplantation from a living related donor with Crohn's disease

We read with interest the case described by Sonwalkar *et al* of a patient who developed fulminant Crohn's colitis after allogeneic stem cell transplantation (ASCT) (*Gut* 2003;**52**:1518–21). Although the donor had no known Crohn's disease (CD) and did not carry the IBD3 or IBD5 haplotypes associated with CD, HLA class III mismatches at IBD3 and a CD associated polymorphism of the 5′UTR of NOD2/CARD15 were present in the donor and in the reconstituted immune cell population of the recipient post ASCT. The authors hypothesised that adoptive transfer of CD susceptibility may have occurred between ACST donor and recipient.

Herein, we report a case of a patient who developed CD after receiving a living related liver transplant from a donor with known CD. A 24 year old female received a liver transplant from a living related donor for decompensated cirrhosis secondary to vertically transmitted chronic hepatitis C infection. The family history was significant for a maternal aunt diagnosed with CD, who served as the liver donor, and a maternal uncle and grandfather with colon cancer. Following liver transplantation, the patient was maintained on an immunosuppressive regimen consisting of tacrolimus 3 mg twice daily, sirolimus 5 mg daily, as well as TMP-SMZ prophylaxis. Her initial post-transplant course was uneventful but she later developed recurrent hepatitis C infection, treated with pegylated interferon and ribavirin. She presented with symptoms consistent with intermittent small bowel obstruction 11 months post-transplant. She was also receiving prednisone 15 mg daily at that time. A computed tomography scan of the abdomen and pelvis (see fig 1A on the *Gut* website: www.gutjnl.com) and an upper gastrointestinal with small bowel follow through study (see fig 1B on the *Gut* website: www.gutjnl.com) demonstrated marked fold thickening of the distal ileum. An enteroscopy demonstrated patchy ulcerations in the jejunum and Roux-en-Y limb of the small bowel. Biopsies showed focal ulceration and mild active inflammation without evidence of granuloma or viral inclusions. Wireless capsule endoscopy demonstrated multiple erosive and ulcerative changes in the distal small intestine (see fig 1C, 1D on the *Gut* website: www.gutjnl.com).

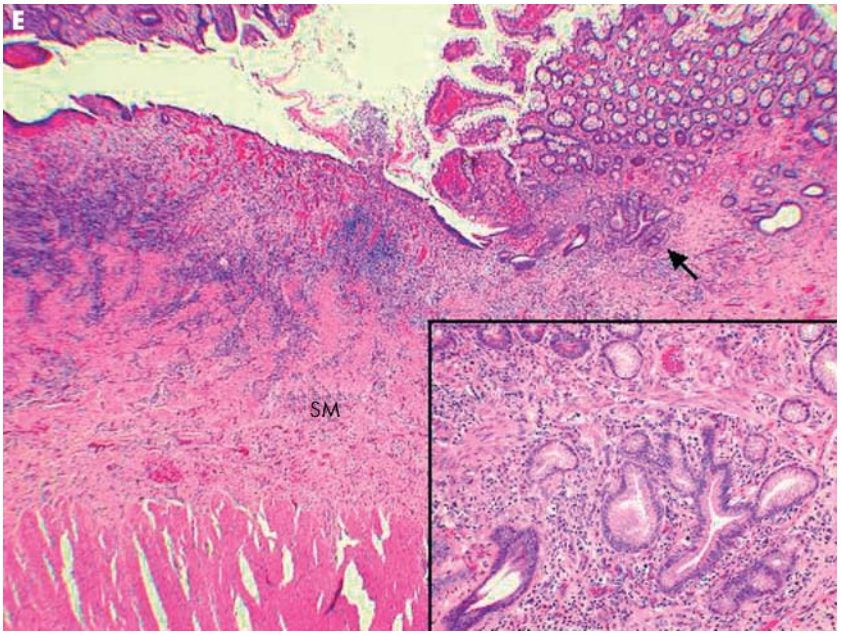

Figure 1 Histopathological examination of a resected ileal specimen demonstrated focal villous blunting, expansion of the lamina propria with acute and chronic inflammatory cells, reactive crypt changes, and occasional crypt abscesses and focal gastric metaplasia (arrow and insert). SM, submucosa.

Because of persistent symptoms and concern for possible lymphoproliferative disorder, the patient underwent an open laparoscopy which revealed nodularity of the terminal ileum. Intraoperative colonoscopy demonstrated nodularity and three ulcers in the distal ileum. Histopathological examination of the resected ileal specimen demonstrated focal villous blunting, expansion of the lamina propria with acute and chronic inflammatory cells, reactive crypt changes, and occasional crypt abscesses and focal gastric metaplasia (see fig 1E, arrow and insert below). Mucosal ulcerations were underlined by inflamed granulation tissue containing occasional histiocytes and multinucleated giant cells. The submucosa also showed intense fibrosis and hyperplasia of the nerve bundles (not shown).

Few cases of de novo IBD developing after liver transplantation for chronic liver disease other than primary sclerosing cholangitis have been described.[1–4] We present a case of CD developing in the recipient of a liver transplant from a living related donor with a known history of CD. The recipient tested negative for any of the three common CD associated NOD2/CARD15 variants (R702W, G908R, 1007fsinsC) but unfortunately we were unable to screen the liver donor for these polymorphisms. Our case, similar to that described by Sonwalkar *et al*, raises the intriguing possibility that CD susceptibility may have been transferred to the recipient with liver transplantation as well. Collins *et al* have reported complete and stable replacement of recipient haematopoiesis and B lymphopoiesis with donor derived cells approximately six weeks following orthotopic liver transplantation for haemochromatosis.[5] T lineage reconstitution also occurred and derived almost exclusively from expansion of mature memory/effector T cells from the transplanted liver. One possibility is that the expanded immune cells have become tolerant to the graft but not to the intestinal luminal antigens leading to the development of CD.[4] Whether liver donor selection should exclude those with a known diagnosis of CD is unclear and is still premature to answer.

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