Perhaps our fetal echogenic bowel scoring system might shed some light.

Of our 145 cases of fetal echogenic bowel, 40 were classified as FEB 1 (echogenicity less than adjacent iliac crest) and the remainder (105) classified as FEB 2 or 3 (echogenicity equal to or greater than adjacent iliac crest). Six of 40 FEB 1 patients had a recent (within 2 weeks) history of vaginal bleeding and five of 14 pregnancies undergoing amniocentesis showed amniotic fluid evidence of haemorrhage. In only one of these amniocentesis studies has cytomegalovirus (CMV) been identified. Our FEB 2 and 3 categories were remarkably different: there were no patients with a recent history of vaginal bleeding, and in only one of 39 amniocentesis specimens could evidence of haemorrhage be confirmed. There have been no CMV-positive cultures in this group of fluids.

The histological analysis of the bowel in the fetus or neonate with CF showed lumenal epithelial cells strikingly distended with PAS (periodic-acid Schiff) positive material along their apical (lumenal) border. This inspissated material might well provide a multitude of sonographically appreciated interfaces and resultant increased echogenicity (analogous to the infantile polycystic kidney). Its lumenal release/secretion in the late second trimester would result in the decrease in the FEB score noted in our paper at that time.

We suspect that the increased bowel echogenicity associated with intra-amniotic bleeding is a different entity (with a lower FEB score) from FEB 2 and 3 bowel echogenicity. Inspissation and mesenteric ischaemia may both be linked to the brightest of fetal bowel, and we agree that ". . . the cause of echogenic bowel in most fetuses . . . remains unknown".

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Mutant factor V (Arg506Gln) in healthy centenarians

SIR—The factor V mutation (Arg506Gln) causing resistance to activated protein C (APC) is an established risk factor for idiopathic and recurrent venous thrombosis.[1] It is common in populations of European descent, with carrier rates varying between 2 and 10%.[2] To explain such a high carrier rate, it has been suggested that the mutation confers evolutionary advantages. It may have provided our ancestors with enhanced coagulation mechanisms, useful for fighters; facilitate the implantation of the embryo; or the abnormal allele may be in close linkage with one or more "protective" alleles. Healthy centenarians are a model of successful ageing and positive selection.[3] We evaluated whether or not the factor V mutation was present in a cohort of Italian centenarians and compared the frequency of the abnormal allele with that found in a group of non-centenarians.

Centenarians (n=124; 31 males, 93 females; median age 102 years, range 100–109) were ambulatory, self-sufficient, and lived in their homes in metropolitan communities of northern Italy (Milan, Modena, Genova, and corresponding provinces). They were selected according to the Senieur protocol.[4] Reasons for exclusions were infection, inflammation, cancer, dementia, diabetes, and renal and liver disease. Non-centenarians (n=378; 183 males and 195 females, median age 43, range 19–76) were also from northern Italy and were chosen to mirror the age distribution of the Italian adult population as established by the most recent national epidemiological survey. Genomic DNA was prepared from blood samples by standard procedures. Mutant factor V was detected by amplification of the factor V gene by PCR and digestion of the fragment with *Mnl* I.[5]

There were five heterozygotes (four women, one man) among 124 centenarians (allele frequency 2%; 95% CI 0·3–3·7) and ten among 378 non-centenarians (allele frequency 1·3%; 95% CI 0·5–2·1). Allele frequencies did not differ in the two groups (p=0·30; odds ratio 0·65). Since there was a greater proportion of women among the centenarians, 93 female centenarians were compared with 195 female non-centenarians. Allele frequencies (2·2% *vs* 1·5%) did not change appreciably. During their lives all the five centenarians carrying the factor V mutation had been exposed without antithrombotic prophylaxis to circumstantial risk factors for thrombosis, such as trauma, pregnancy, and major surgery (table), but developed no thrombotic episodes.

Case	Sex	Risk factors
1	F	Major surgery at age 59; fractured right humerus at age 73
2	F	Two pregnancies
3	F	Three pregnancies; orthpaedic surgery for fractured right femur at age 71
4	F	Four pregnancies; major abdominal surgery at age 76
5	M	Orthpaedic surgery for fractured left femur at age 73

Table: **Lifelong exposure of five centenarians with the factor V mutation to circumstantial risk factors for thrombosis**

Our findings indicate that the factor V mutation is compatible with extreme longevity and successful ageing. Perhaps these exceptional individuals possess still unknown protective factors against thrombosis that counteract the thrombotic risk carried by the factor V mutation.

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Mercury contamination in the home

SIR—Bonhomme and colleagues (Jan 13, p 115)[1] provide further documentation that cleaning activities can spread contamination of elemental mercury. The contaminant is often spread when persons responsible for custodial care of the facility use common cleaning equipment such as vacuum cleaners, brooms, and mops; this is especially true after spills of elemental mercury. I have spoken with several individuals responsible for clean-up after elemental mercury spills who found that initial use of common vacuum cleaners spread mercury vapour throughout the building. In addition, the vacuum cleaner contributed to tracking mercury from the spill site, thus expanding the area of localised mercury contamination in the carpet. However, the larger issue in the spread of contamination is the discharge of air containing