An outbreak of diarrhoea due to *Escherichia coli* 0157:H7 in Pennsylvania in September 2000 was traced to a dairy farm open to the public where children observed and petted the animals (*New England Journal of Medicine* 2002;**347**:555–60). Of 51 confirmed or suspected cases 47 were under 11 years old, of whom eight developed the haemolytic-uraemic syndrome. Twenty-eight of 216 cattle on the farm were colonised with an organism that appeared to be identical to the one isolated from patients. Handwashing protected against infection. Several recent reports have suggested that farm animals are a more important source of this infection than has been realised.

A case report from Copenhagen (*New England Journal of Medicine* 2002;**347**:576–80; see also editorial, ibid: 609–12) has shown that mitochondrial DNA may be inherited from the father. Usually mitochondria from sperm are effectively eliminated in the early embryo by selective destruction, inactivation, or dilution by oocyte mitochondria. The Danish report describes a 28-year old man with a mitochondrial myopathy. His muscle tissue had a novel mitochondrial DNA deletion in the *ND2* gene but there was no such deletion in blood lymphocytes, hair roots, or cultured fibroblasts. Sequence analysis showed that the mitochondrial DNA in the patient's muscle was the same as his father's mitochondrial DNA although in other tissues he had his mother's mitochondrial DNA. His father did not have the mitochondrial DNA deletion so the gene deletion must have occurred in paternal mitochondrial DNA either in the paternal germ line or in the early embryo.

Do you routinely examine the ears of children with a chronic cough? In France (*Lancet* 2002;**360**:618) a boy was very extensively investigated over several years for a dry cough which began at the age of 8 years, one year after surgery for an attic cholesteatoma. When he was 14 and still had the cough he was examined in an ear, nose, and throat department and found to have a stenotic right external auditory meatus with accumulated wax and an ulcer of the meatal skin. Stimulation of the anterior wall of the ear canal with a cotton bud induced coughing. Removing the wax stopped the cough for several weeks and this cycle was repeated several times. After almost a year of this the surgeons removed the ulcer and enlarged the bony stenosis. Three years later he had had no more spontaneous coughing but rubbing the skin of his ear canal still made him cough. The ear-cough reflex was described by Arnold 130 years ago.

Benign familial neonatal seizures begin on the second or third day of life and stop after a few weeks though epilepsy develops later in about 11% of cases. Mutations in potassium-channel genes may explain many cases. Benign familial infantile seizures begin between 4 and 8 months of age and usually stop before 1 year. No gene mutations have been associated with this syndrome. Now (*Lancet* 2002;**360**:851–2) an intermediate syndrome has been described, benign familial neonatal-infantile seizures, with seizures beginning at around 2 months and stopping at around 4 months on average. Thirteen individuals were affected in several generations of two families, one of Irish origin in Australia and one Ashkenazi Jewish family in Canada. Mutations of the sodium-channel subunit gene *SCN2A* were found in affected individuals in both families. All three of these benign familial seizure disorders are inherited in an autosomal dominant fashion.

One surgeon at Moorfields Eye Hospital in London (*British Journal of Ophthalmology* 2002;**86**:1282–6) performed surgery for ptosis on 340 children in the 8 years 1990–97. In 280 cases the ptosis was unilateral and the most common cause was congenital levator myopathy (240 patients, 71%). Thirty-seven children (11%) had neurogenic ptosis, 26 of whom showed the Marcus Gunn jaw-winking phenomenon. Ten children had congenital syndromes including Noonan's syndrome, ocular fibrosis, fetal alcohol, Down's, Turner's, and XXX syndromes. Surgery was cosmetic in 57% and for visual indications in 43%. Several surgical techniques were used but in 77% of patients the outcome was judged to be good and in 13% poor. Outcome was not affected by the degree of ptosis.

Increased lifetime cancer risk may be one of the consequences of excess weight in early life. A long term (mean 41 years) follow up study of students at the University of Glasgow (*Journal of Epidemiology and Community Health* 2002;**56**:780–4) has shown increased body mass index while at university to be associated with increased risk, especially of cancers not related to smoking. High BMI as a student was particularly associated with later prostate and breast cancers.

Frequent (most days or daily) use of paracetamol in late pregnancy may increase the risk of wheezing in young children. A birth cohort study in southwest England (*Thorax* 2002;**57**:958–63) showed that it more than doubled the risk of wheezing at age 30–42 months. It could, though, account for only 1% of wheezing in children of that age.

A review (*Journal of Medical Screening* 2002;**9**:135–41) based on a report by the Antenatal Screening Sub-committee of the National Screening Committee concludes that the introduction in the UK of prenatal or neonatal screening for congenital toxoplasmosis would not be justified by the currently available evidence. At present probably about 1 in 10 000 children is congenitally infected but only five in a million have severe neurological disease in infancy and some 20 or 30 per million develop brain or eye lesions by the age of 3 years. Adequate studies on the effect of treatment either before or after birth are lacking. From a community point of view acquired toxoplasmosis is a bigger problem than congenital. Improved hygiene with respect to meat and meat products and, in some countries, improved water supply might be the most productive approach to control of both congenital and acquired disease.

A national case-control study in Sweden (*American Journal of Clinical Nutrition* 2002;**75**:914–21) has shown that, in children under 2 years old, breastfeeding at the time of introduction of gluten into the diet reduces the risk of coeliac disease by 40%. In children who continued to breast feed after that time the risk reduction was 64%. The risk was related to the amount of gluten in the diet. A commentator in *Gut* (*Gut* 2002;**51**:767–8) hypothesises that protection against intestinal infection is the most likely explanation for the protective effect of breastfeeding. Gut infection increases expression of the enzyme tissue transglutaminase (TG2) that promotes deamidation of gluten peptides and these deamidated peptides induce the T cell reaction that leads to coeliac disease.

In India late weaning is associated with stunting of growth (*International Journal of Epidemiology* 2002;**31**:855–63). Data for 1992–93 from six regions of the country showed that children weaned at 6 months were 57% more likely to be stunted (>2SD below median height for age) at age 2–4 years compared with children weaned before 6 months. Children weaned later than 6 months had an 88% increase in risk. Overall, over half of Indian children under 4 years old are stunted. In this study 77% of children living in poor conditions, not fully immunised in the first year, and weaned later than 6 months were stunted.