



There has remained some uncertainty about whether oats are toxic to people with coeliac disease. In Sweden (*Gut* 2004;**53**:649–54) 116 children with coeliac disease were randomised to a standard gluten free diet without oats or a gluten free diet with added oats. Median daily intake of oats in the added-oats group was 15 g. All children remained symptom free at 12 months and there were no differences between the groups in coeliac serology markers or jejunal biopsy findings. The addition of oats to a gluten free diet did not prevent healing in these children.

Researchers in India now refer to “nutritional transition” with obesity increasingly causing concern especially in urban areas. In a cohort study in Delhi (*New England Journal of Medicine* 2004;**350**:865–75) impaired glucose tolerance or diabetes affected 8% of 26 to 27-year olds and 18% of 31 to 32-year olds. People who developed impaired glucose tolerance or diabetes had had a higher body mass index (BMI) as young adults but had been typically thin up to the age of 2 years. Their relative BMI (BMI SD score) had increased throughout the rest of childhood though only 3% had been overweight and none obese at the age of 12 years. The age at lowest BMI (“adiposity rebound”) was slightly but significantly less (6.3 vs 6.7 years) in those who developed impaired glucose tolerance or diabetes.

Can viruses be good for you? Usually infection with one virus makes infection with another virus worse. For instance, coinfection with cytomegalovirus or with herpesvirus 1 or 2 may speed up the progression of HIV disease. GB virus C (GBV-C), however, is not known to be pathogenic to humans and there is evidence that it may be beneficial for people with HIV infection. In an American cohort study (*New England Journal of Medicine* 2004;**350**:981–90; see also perspective article, *ibid*: 963–5) 271 men were assessed 12–18 months after HIV seroconversion and 138 were reassessed 5–6 years after HIV seroconversion. GBV-C viraemia at 12–18 months did not affect survival but men without GBV-C viraemia at 5–6 years had a 2.8-fold increase in mortality compared with men with GBV-C viraemia at that time. The men who did worst were those who lost GBV-C viraemia between 12–18 months and 5–6 years after HIV seroconversion. All this ought to have something important to teach us about HIV infection but what it is remains to be discovered.

In Sheffield (*Lancet* 2004;**363**:846–51) 111 apparently healthy babies born at term had MRI head scans performed on a dedicated neonatal unit scanner within 48 hours of birth. Nine had subdural haemorrhage: three of 49 after normal vortex delivery, five of 18 after forceps delivery following failed ventouse, and one after a traumatic ventouse delivery. None of 25 babies born by caesarean section had a subdural haemorrhage. Apart from mode of delivery, subdural haemorrhage was not related to obstetric factors. The subdural haemorrhages of all nine babies had resolved completely on repeat scanning after 4 weeks and the infants remained well at the age of 2 years. The findings may have medicolegal implications.

Data from the UK central cardiac audit database (*British Medical Journal* 2004;**328**:611–5; see also editorial, *ibid*: 594–5) show that there were 3666 surgical procedures and 1825 therapeutic catheterisation procedures performed on children with congenital heart disease in England, Scotland, and Northern Ireland between 1 April 2000 and 31 March 2001. Overall, 20-day survival after surgery was 95% and 1-year survival 92%. After therapeutic catheterisation survival was 99% and 98% at 30 days and 1 year. Rates of survival after six benchmark operations and three benchmark catheterisation procedures did not differ significantly between centres but confidence intervals are wide and the editorialist comments that the analysis is aimed at proof of difference rather than at providing an alert for safety purposes.

Early high-dose dexamethasone should not be used to prevent the development of chronic lung disease in infants with severe respiratory distress syndrome of prematurity. In Taiwan (*New England Journal of Medicine* 2004;**350**:1304–13; see also editorial, *ibid*: 1349–51) 159 survivors of a neonatal randomised, placebo-controlled trial were assessed at age 8 years. The children who had been given dexamethasone were shorter and had smaller head circumferences and poorer motor skills, co-ordination, and visual-motor integration compared with the placebo group. They also had lower full, verbal, and performance IQ scores and a higher prevalence of clinically significant disability (28 of 72 vs 16 of 74). Three Cochrane meta-analyses have also cast doubt on the safety of steroids to prevent or treat chronic lung disease whether given early or late in the neonatal period.

First it was HIV from primates, then influenza from poultry and SARS from civets, now it's malaria from monkeys. *Plasmodium knowlesi* infects macaque monkeys and there have been only two case reports of human infection. Now (*Lancet* 2004;**363**:1017–24) it has been reported as a cause of malaria in Sarawak, Malaysian Borneo. More than half (120) of 208 people with acute malaria were shown by PCR assay to have *P knowlesi* infection. Most of them had been diagnosed on microscopy as having *P malariae* infection. Nine children aged 10–15 years were among the 106 patients with single species *P knowlesi* infections.

Bartter's syndrome types I and II are characterised by maternal polyhydramnios and severe early neonatal volume depletion and they are caused by mutations in the sodium-potassium-chloride cotransporter NKCC2 and potassium channel ROMK genes respectively. Bartter's syndrome type III is classic Bartter's syndrome with hypokalaemia in early infancy or adolescence and is due to a mutation in the chloride channel CLC-Kb gene. Type IV is antenatal Bartter's syndrome with sensorineural deafness (BSND). Alterations in the protein, barttin caused by BSND gene mutation interfere with the insertion of the chloride channels CLC-Kb and CLC-Ka into the plasma membranes of cells in the kidney and inner ear. Now researchers in Germany (*New England Journal of Medicine* 2004;**350**:1314–9; see also perspective article, *ibid*: 1281–3) have described a child, born to first-cousin parents, who had the clinical features of BSND but a normal BSND gene. This child had mutations in the genes for both CLC-Kb (*CLCNKB*) and CLC-Ka (*CLCNKA*), the so far unique Bartter's syndrome type V. (Gitelman's variant of Bartter's syndrome is caused by a mutation in the gene for the sodium-chloride cotransporter (NCCT).

Skiing deaths are rare, around 2 or 3 per million skiers, but children and adolescents are thought to be at greater risk than adults. In Colorado between 1980 and 2001 (*Injury Prevention* 2004;**10**:99–102) there were 149 downhill skiing deaths, 21 of people aged 17 years or under. The youngest was 7 years old. One other child who died was under 10, eight were aged 10–13, and eleven aged 14–17. Ten were girls. Two thirds of child deaths were from head injuries and more than three quarters resulted from collision, most often with a tree. Prevention strategies should be aimed at reducing the likelihood of collision, especially with trees, and possibly the wearing of helmets.