Is television bad for your health? A birth cohort study in New Zealand (*Lancet* 2004;**364**:257–62), see also Comment, ibid: 226–7) suggests so. Nine hundred and eighty people followed up from birth were reassessed at age 26 years. The average duration of weeknight television watching between the ages of 5 and 15 years was positively correlated with current body mass index, poor fitness, cigarette smoking, and serum cholesterol, after adjustment for potential confounding factors. It was estimated that watching television for 2 hours or more a day during childhood accounted for 17% of overweight, 15% of raised serum cholesterol, 17% of smoking, and 15% of poor fitness at age 26.

Currently about 80% of children with acute lymphoblastic leukaemia (ALL) can be cured with chemotherapy but the causes of treatment failure are ill understood. Gene expression profiling may identify treatment resistant cases and point the way to even greater rates of success. Researchers in the Netherlands, the USA, and Germany (*New England Journal of Medicine* 2004;**351**:533–42, see also editorial, ibid: 601–3) used 14 500 probe sets to identify differentially expressed genes in leukaemia cells from 173 children with ALL. In vitro sensitivity to four drugs was first determined and gene expression profiling identified sets of genes that were expressed differently in cells sensitive or resistant to the drugs (prednisolone (33 genes), vincristine (40 genes), asparaginase (35 genes), and daunorubicin (20 genes)). The gene expression profiles were significantly related to treatment outcome. Research into the functions of these genes may open the way to new and better treatments.

Zinc stimulates immune responses. Adding it to the treatment of pneumonia for young children in poor countries may accelerate recovery. In Bangladesh (*Lancet* 2004;**363**:1683–8) 270 children aged 2–23 months were given antibiotics for pneumonia and randomised to zinc acetate (20 mg elemental zinc daily) or placebo. The signs of pneumonia resolved significantly quicker in the zinc group and the mean length of stay in hospital was reduced from 6 days to 5 days.

If reduced exposure to bacteria and bacterial products in early life promotes the development of atopy what are the biological mechanisms involved? Researchers in Montreal (*Lancet* 2004;**363**:1689–97) have examined the effects of allergen and bacterial lipopolysaccharide on cultured nasal mucosa from 15 atopic children (mean age 3.2 years) and 10 atopic adults (mean age 40.4 years). They found that in children's, but not in adults', mucosa the lipopolysaccharide suppressed the allergic (T-helper type 2, Th2) response and promoted non-allergic (Th-1) cytokine production. The change in response to allergen was accompanied by increased expression of lipopolysaccharide receptor (toll-like receptor 4, TLR4) on mucosal T cells suggesting that TLR4 may play an important part.

Policies about chemoprophylaxis (usually with either rifampicin or ciprofloxacin) for the contacts of patients with meningococcal disease are based on inadequate evidence. A systematic review of four observational studies and one small trial (*British Medical Journal* 2004;**328**:1339–42) has suggested that treating household contacts might reduce the risk of secondary cases arising within 30 days by about 89%. There are no satisfactory data about chemoprophylaxis in day care settings or about the effectiveness of prophylaxis given to patients before they leave hospital but it is estimated that in the absence of chemoprophylaxis at least 3% of patients are carriers at hospital discharge.

In Denmark between 1943 and 1987 a total of 6627 people were born with cleft palate, cleft lip, or both and 6394 were identified for collection of follow up data (*British Medical Journal* 2004;**328**:1405–6). One thousand and sixty-three people (17%) were excluded from the study because they had associated anomalies. Among the remaining 5331 the standardised mortality ratio (SMR) to the age of 55 was 1.4 for males and 1.8 for females. The SMRs were 1.4 and 1.9 in infancy, 1.4 and 1.8 at ages 1–17, and 1.5 and 1.7 at ages 18–55. Prematurity, pneumonia, postoperative complications, asphyxia, aspiration, and sepsis accounted for most infant deaths but later mortality was increased for all major causes although deaths from cancer were not significantly increased. There was a 60% increase in suicide in both sexes and six females died of epilepsy (1.2 epilepsy deaths would have been expected). Mortality rates were increased for people with cleft palate with or without cleft lip but not for people with cleft lip alone. What can be done to prevent excess later mortality among people born with cleft palate is yet to be determined but much of the excess infant mortality should presumably be preventable.

Renal replacement therapy (dialysis and transplantation) for children with end-stage renal disease saves lives but rates of survival are still much less than for children in the general population. In Australia and New Zealand (*New England Journal of Medicine* 2004;**350**:2654–62, see also perspective article, ibid: 2637–9) mortality rates fell rapidly between 1963–72 and 1983–92 but there appears to have been little or no improvement since the early 1980s. Overall, survival of patients aged under 20 years at the start of therapy between 1963 and 2002 was 96%, 78%, 71%, and 66% at 5, 10, 15 and 20 years. Survival of infants was 73%, 67%, and 67% at 5, 10, and 15 years. Over the four decades of the study the rate ratio for death within 10 years, compared with population data, fell from 116 to 32 for children aged 0–4 years at the start of treatment, from 236 to 94 (30 in the third decade of the study) for children aged 5–9, from 111 to 35 for children aged 10–14, and from 52 to 30 (19 in the third decade) for children and adolescents aged 15–19. Improvement will depend on increasing the proportion receiving a transplant and on tackling the long-term problems of cardiovascular and malignant diseases.

Can muscle bulk and strength be altered by manipulation of a muscle growth factor? In Germany (*New England Journal of Medicine* 2004;**350**:2682–8, see also perspective article, ibid: 2642–4) a child with increased muscle bulk and strength was shown to have a probable loss-of-function mutation in the myostatin gene and myostatin was undetectable in his serum. He was homozygous for the mutation and his mother, who had been a professional athlete, was heterozygous. The child had had noticeably increased muscle bulk at birth and stimulus-induced myoclonus for the first 2 months. At the age of 4.5 years his muscular hypertrophy persisted and he was unusually strong but otherwise normal. Myostatin (growth and differentiation factor 8, GDF-8) is a negative regulator of muscle mass. In animals overexpression of the myostatin gene causes muscle wasting and underexpression is associated with muscle hypertrophy. The exciting possibility is that myostatin blockade could prove to be beneficial for patients with muscle wasting. The downside is that it is said that some athletes and their minders already have their eyes open to the possibilities.