	Children with severe anaemia (n=77)	Children without severe anaemia (n=467)	Odds ratio (95% CI)
Height-for-age	–1·99 (1·40)	–1·39 (1·36)	0·72* (0·60–0·86)
Weight-for-age	–2·44 (1·13)	–1·76 (1·12)	0·58* (0·46–0·73)
Weight-for-height	–1·62 (0·79)	–1·21 (0·89)	0·58* (0·43–0·78)
Number of falciparum malaria episodes in previous 6 months	1·29 (1·04)	1·31 (0·97)	0·92† (0·71–1·18)
Falciparum malaria ⩽10 days before haemoglobin measurement (yes/no)	2/75	32/435	0·32† (0·07–1·39)

Data are mean (SD) unless otherwise stated. *For continuous anthropometric score, odds ratio refers to increase of 1, adjusted for sex and age. †Adjusted for sex, age, and weight-for-age.

Association between severe anaemia, falciparum malaria, and malnutrition in young west African children

Plasmodium falciparum per μL) over the 6-month observation period, for the prevalence of falciparum malaria within 10 days before the December survey, and for their mean HAZ/WAZ/WHZ SD scores. In logistic regression analyses (adjusted for age and sex), anaemia was not associated with the frequency of malaria episodes, nor with malaria prevalence. However, it was significantly associated with malnutrition, defined as HAZ, WAZ, and WHZ of –2 or less (table).

These data contribute to the growing evidence for the importance of malnutrition in the development of anaemia in young children living in malaria-endemic areas.[3] Given the well known grave implications of malnutrition on morbidity and mortality, programmes with the aim of improving the health of young children in developing countries need to put much more emphasis on improving the overall nutritional situation of young children.[4,5] Finally, these findings also have implications for use of anaemia as an outcome in malaria control trials.

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Tianeptine, plasma serotonin, and pulmonary hypertension

Sir—After reading the Commentary by Raed A Dweik (Sept 21, p 886),[1] we felt obliged to add some information. Free serotonin in the plasma is known to trigger bronchial and pulmonary vascular constriction.[2] Blood serotonin arises from enterochromaffin cells, and the concentration of serotonin in the lung is closely correlated with that of platelets in the blood.[3] Normally, total blood serotonin comprises 95–97% platelet serotonin and 3–5% free serotonin. The ratio of free serotonin to platelet serotonin increases as a result of platelet aggregation and interference with serotonin uptake by platelets.[4] Platelet aggregation is seen during stressful situations (secondary to an increase in plasma epinephrine concentration), whereas platelet serotonin uptake is affected by circulating acetylcholine or dopamine concentrations, increases in which are seen during parasympathetic hyperactivity and other patho-physiological phenomena.[5]

The above information is consistent with the successful treatment of patients with bronchial asthma and pulmonary hypertension seen with low doses of tianeptine—a drug inadequately labelled as an antidepressant.[2–5]

In summary, all pathophysiological or clinical investigations dealing with bronchial or pulmonary vascular constriction should not ignore the role of plasma serotonin, which, in our experience, is one of the most important aetiopathogenic factors of both disorders.

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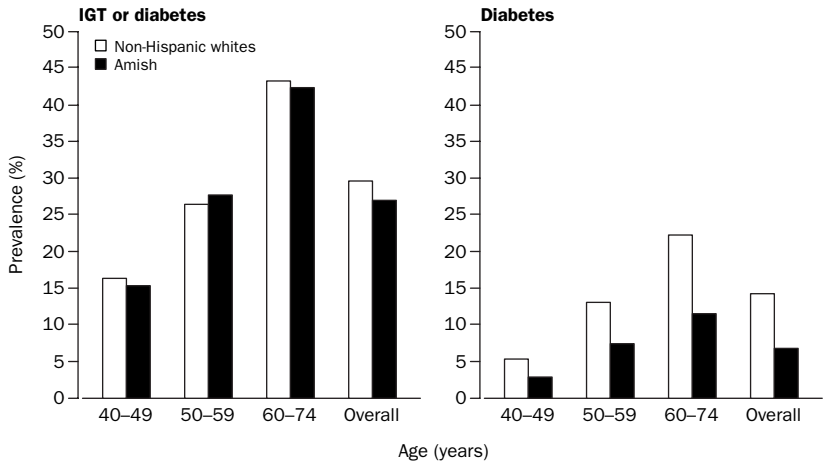
Physical activity and prevention of type 2 diabetes

Sir—Two landmark prospective studies[1,2] have shown that incidence of type 2 diabetes can be decreased by 58% in individuals with impaired glucose tolerance (IGT) through diet and exercise. However, these studies did not address whether the accompanying 4–6 kg weight loss itself was responsible for the striking decreases in incidence rates, or whether exercise had an independent effect. Nor did these studies address whether the beneficial effects of the intervention would be sustained beyond the relatively short duration of the studies.

Insights into these questions can be gleaned from the Old Order Amish. These 30 000 or so individuals, whose



Prevalence of impaired glucose tolerance (IGT) or type 2 diabetes (left) and of type 2 diabetes (right) in non-Hispanic whites and Amish individuals

ancestors arrived on US shores in the 18th century, maintain religious and cultural beliefs that preclude regular use of modern conveniences such as electrical appliances, telephones, and cars, and have a physically active lifestyle. By comparison, the 200 million typical Americans living alongside them have, over the past 250 years, willingly adopted advances of modern technology, making life less physically demanding. Both groups have similar diets.

Surprisingly, Amish adults are just as obese as the general US white population (mean body-mass index 27·9 kg/m^2 [SD 4·6] in Amish[3] *vs* 27·0 kg/m^2 [5·1] in non-Hispanic whites[4]) and almost as likely to have an abnormal oral glucose tolerance test (ie, either IGT or diabetes; figure). However, manifest diabetes is about half as prevalent in the Amish as it is in the general population (6·7% in Amish[3] *vs* 14·3% in non-Hispanic whites;[4] prevalence ratio 0·54, 95% CI 0·23–0·84; figure). The apparently low rate of conversion from IGT to diabetes suggested by these cross-sectional data is supported by follow-up glucose tolerance tests 2–5 years later in more than 100 Amish individuals with IGT (unpublished data).

The lower prevalence of diabetes in the Amish suggests that physical activity has a protective effect against type 2 diabetes, independent of body-mass index. This observation is in agreement with the Da Qing Study.[5] Furthermore, stratification of our Amish individuals by age (figure) shows that the protective effect of the 18th century lifestyle against diabetes occurs over all age-groups from 40 to 74 years,[3] suggesting that physical activity has durable effects.

Because dieting attempts are frequently unsuccessful, these observations suggest that increasing physical activity is an appealing way to reduce the risk of diabetes. The concept of incorporating physical activity into daily life seems more feasible than dedicated exercise sessions, but will require societal changes including urban planning issues, transportation policies, and employer participation.

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Simian virus 40 infection in lymphoproliferative disorders

Sir—Lymphomas are associated with several molecular pathways.[1] Two reports suggested a role for simian virus 40 (SV40) in lymphomagenesis.[2,3] On the basis of the paradigm of other lymphoma-related viruses,[1] SV40 has several features predictive of a pathogenetic role: it is lymphotropic, exerts its transforming activity through large T antigen, and is capable of transforming B cells in vitro and inducing B-cell lymphomas in animals.[4]

We investigated the presence of SV40 in 500 individuals referred to Italian or Spanish institutions with lymphoproliferative disorders. DNA from frozen samples was tested for PCR suitability by amplification of a 748 bp *BCL6* gene fragment. SV40 DNA detection was based on three PCR reactions that targeted 5′ and 3′ sequences of large T antigen. Primers included: SVTagP1 and SVTagP2 (156 bp 5′-sequence amplimer); SVfor2 and SVrev (574 bp 5′-sequence amplimer); TA1 and TA2 (441 bp 3′-sequence amplimer).[2,3] In dilution experiments with SV40 plasmid (pZEO-SVE) DNA, all PCR (45 cycles) attained 10^{-4} sensitivity, allowing the detection of ten SV40 genomes among 200 000 human genome equivalents. Assignment of SV40 positivity required positive signals in all three PCR assays and amplimer validation by DNA sequencing. A masked pilot experiment readily identified SV40-positive mesotheliomas, but not SV40-negative tumours.

On the basis of the adopted criteria, no case of lymphoproliferative disorder scored positive for SV40 (table; see http://image.thelancet.com/02cor11020webtable.pdf for detailed histological types). Conversely, PCR assays detected human herpesvirus 8 (HHV8) infection in 11 of 11 primary effusion lymphomas (eight AIDS-related, three non-AIDS-related), and Epstein-Barr virus (EBV) infection in 64 of 500 cases. With respect to SV40, 17 of 500 (3·4%) cases yielded SV40 sequences in SVTagP1/SVTagP2 and SVfor2/SVrev reactions, but none was positive by the 3′-region TA1/TA2 reaction, nor with primers TA3 (nucleotides 2621–2645) and TA5 (3005–3026), mapping in the proximity of the TA1 and TA2 primers .

Lack of positivity cannot be ascribed to primer mismatches, because TA1/TA2 primers anneal to conserved regions, whereas most polymorphisms