lesions in skin. This finding may reflect HHV-8's preference for skin. With the exception of body-cavity lymphomas, most HHV-8 associated cancers are primarily cutaneous.[2] This virus may be more common in the population than expected; there must be factors other than immunodeficiency which favour its presence.

Supported by a grant from the Dr Mildred Scheel Stiftung, Bonn, Germany, and the Geissler Stiftung, Munich, Germany.

*Christian A Sander, Martin Simon, Ursula Puchta, *Mark Raffeld, Peter Kind*
Department of Dermatology, LMU, Munich, Germany; *Laboratory of Pathology, Hematopathology Section, NCI, NIH, Bethesda, USA

1 Cesarman E, Chang Y, Moore PS, Said J, Knowles DM. Kaposi's sarcoma-associated herpesvirus-like DNA sequences in AIDS-related body-cavity-based lymphomas. *N Engl J Med* 1995; 332: 1186–91.
2 Rady PL, Yen A, Rollefson JL, et al. Herpesvirus-like DNA sequences in non-Kaposi's sarcoma skin lesions of transplant patients. *Lancet* 1995; 345: 1339–40.
3 Chang Y, Cesarman E, Pessin MS, et al. Identification of herpesvirus-like DNA sequences in AIDS associated Kaposi's sarcoma. *Science* 1994; 266: 1865–69.
4 Simon M, Kind P, Kaudewitz F, et al. Detection of herpesvirus-like DNA in HIV-associated and classical Kaposi's sarcoma. *Arch Dermatol Res* 1996; 288: 402–04.

Chernobyl and hypothyroidism

SIR—The USA received much less fallout from Chernobyl than did most European countries—about 1/30th of the UK exposure to ^{131}I and about 1/10 000th of the exposure of the areas around Chernobyl itself.[1,2] It was therefore surprising to read Mangano's report linking an apparent increase in neonatal hypothyroidism in areas of the USA to Chernobyl fallout.[3] The data on newborn hypothyroidism were given for 1986–87. Most of the infants born during this period will not have been exposed in utero to radioactive iodine from Chernobyl, either being born before the accident or more than 9 months afterwards. As the fetal thyroid begins to concentrate iodine only at about 3 months' gestational age, children born between May and December, 1986, will have formed the main groups exposed to the radioactive species.

Mangano suggests that no documented hypothyroid trends after Chernobyl in the former Soviet Union have yet been produced. In fact, the Sasakawa Project has published annual reports since 1992, showing the frequency of biochemically defined hypothyroidism in children in five areas around Chernobyl.[4] In 1995 the highest prevalence ($30{\cdot}2\times10^{-4}$) was found in the Gomel area of Belarus, which is the area with the highest level of radioactive iodine contamination and the highest incidence of childhood thyroid carcinoma. Incidentally, hypothyroidism has also been recorded in horses and cattle that were not evacuated from an area near Chernobyl.[1]

It is difficult to envisage a mechanism that can cause hypothyroidism from such a minute increase in exposure to ^{131}I as occurred in the USA after Chernobyl. It should be possible to analyse the data in a more meaningful way, and also to inquire whether there was any change in the screening method used during the time of the study.

Dilwyn Williams
Department of Histopathology, University of Cambridge, Addenbrooke's Hospital, Cambridge CB2 2QQ, UK

1 The International Chernobyl Project, Vienna: International Atomic Energy Agency Technical Report; 1991.
2 Malone J, Unger J, Delange F, Lagasse R, Dumont JE. Thyroid consequences of Chernobyl accident in the countries of the European Community. *J Endocrinol Invest* 1991; 14: 701–17.
3 Mangano JJ. Chernobyl and hypothyroidism. *Lancet* 1996; 347: 1482.
4 Sasakawa Memorial Health Foundation. Sasakawa Foundation, Japan: A report on the 1995 Chernobyl Sasakawa Project Workshop; 1995.

Author's reply

SIR—I agree with Williams that the apparent link between Chernobyl fallout and rising neonatal hypothyroidism in the USA is initially surprising. However, the association takes on greater meaning after consideration of the evidence that the biological effects of low levels of fission products follow a logarithmic, not linear, dose response. The linear model was derived from analyses of brief, high-dose exposures (medical use, laboratory studies, and nuclear explosions). Since a 1972 report by Petkau,[1] a growing body of evidence has pointed to the damaging effects of low-dose, chronic exposure, and shows that this radiation damage follows a logarithmic (concave-downward) curve. This greater per-dose damage is attributed to the more efficient action of free-radical oxygen molecules at low radiation doses, which attack cell membrane proteins and impair immune function. A concave-downward association has also been described[2] between breast-cancer mortality in US regions and airborne radioactivity. The same type of curve exists between ^{131}I levels and hypothyroid rate changes in US regions after Chernobyl (figure).

Apart from low-level radioactive iodine exposure, there may be other reasons for rising hypothyroidism, and for why thyroid cancer in Belarus and the USA[3] increased only 5 years after the Chernobyl incident. For example, it is known that excess thyroid cancer from therapeutic radioiodine or head and neck irradiation occurs 20 to 30 years after exposure. The Chernobyl fallout contained dozens of isotopes besides iodine, including strontium-90. ^{90}Sr concentrates in bone, and its daughter product yttrium-90 infiltrates organs such as the pituitary, the source of thyroid stimulating hormone. Because ^{90}Sr has a long half-life (28 years), its action may extend the period of increased newborn hypothyroidism beyond 1986, and shorten the latency period for development of thyroid cancer.

More efficient actions of radionuclides at chronic low doses, and the combined effects of various isotopes in the Chernobyl fallout may provide greater insight into the initially surprising patterns of thyroid cancer and hypothyroidism after 1986. These factors may also account for earlier increases in fetal, infant, and neonatal mortality in the USA and UK from nuclear bomb test fallout,[4] and increased childhood cancer mortality in the UK from 1953 to 1979 due to natural and man-made background radiation.[5] They also may help to explain why thyroid disorders, and not

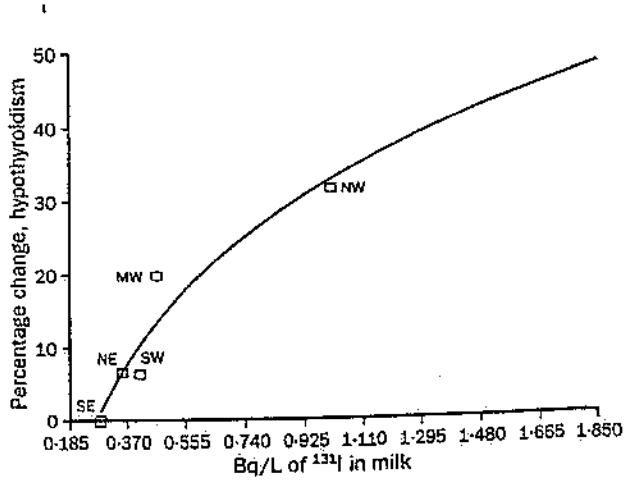

Figure: Change in hypothyroidism 1984–85 *versus* 1986–87 compared with ^{131}I in milk May/June 1986 in 32 US states

other cancers or leukaemia, were the first documented adverse health effects of the Chernobyl disaster.

Joseph J Mangano
Radiation and Public Health Project, Brooklyn, New York, NY 11215, USA

1 Petkau A. Effects of $^{22}Na^{+}$ on a phospholipid membrane. *Health Physics* 1972; 22: 239.
2 Sternglass EJ, Gould JM. Breast cancer: evidence for a relation to fission products in the diet. *Int J Health Services* 1993; 23: 783–804.
3 Mangano JJ. A post-Chernobyl rise in thyroid cancer in Connecticut, USA. *Eur J Cancer Prevention* 1996; 5: 75–81.
4 Whyte RK. First day neonatal mortality since 1935: re-examination of the Cross hypothesis. *BMJ* 1992; 304: 343–46.
5 Knox EG, Stewart AM, Gilman EA, Kneale GW. Background radiation and childhood cancers. *J Radiol Prot* 1988; 8: 9–18.

Exercise, wellbeing, and endogenous molecules of mood

SIR—Steptoe and Butler (June 29, p 1789)[1] show that vigorous physical activity is associated with positive emotional wellbeing in adolescents. The antidepressive effect of vigorous exercises can be related to changes in the endogenous opioid system during physical activity. Plasma β-endorphin concentrations are increased during and after vigorous exercises.[2,3] It is also known that naloxone, an opioid antagonist, blocks analgesia produced by long-distance running.[3] Intravenous administration of β-endorphin improves mood and reduces anxiety.[4] Opioids were widely used as antidepressants before the introduction of electroconvulsive therapy,[4] and endogenous opioids have been implicated in the mechanism of the antidepressive effect of vigorous physical exercises.[5] Probably, this mechanism plays an important part in the effects Steptoe and Butler recorded. In fact, it means that people who exercise vigorously may feel "high". Possibly, this is one of the fundamental mechanisms for mankind's survival: an endogenous euphoric reward system that reinforces physical training.

Leonid Sher
Hillside Hospital, Long Island Jewish Medical Center, Glen Oaks, NY 11004, USA

1 Steptoe A, Butler N. Sports participation and emotional wellbeing in adolescents. *Lancet* 1996; 347: 1789–92.
2 Schwarz L, Kinderman W. Beta-endorphin, adrenocorticotropic hormone, cortisol and catecholamines during aerobic and anaerobic exercise. *Eur J Appl Physiol* 1990; 61: 165–71.
3 Davis GC. Endorphins and pain. *Psychiatr Clin North Am* 1983; 6: 473–87.
4 Freeska E, Davis KL. The opioid model in psychiatric research. In: Nemeroff CB, ed. Neuropeptides and psychiatric disorders. Washington, DC: American Psychiatric Press, 1991: 169–91.
5 Allen M. Activity-generated endorphins: a review of their role in sports science. *Can J Apple Sport Sci* 1983; 8: 115–33.

SIR—The article by Steptoe and Butler[1] extends to adolescents what is known already about adults—namely, the importance of regular activity in promoting physical and psychological health. We have reported that many British children and adolescents have sedentary lifestyles[2] and, because inactive youngsters are unlikely to become active adults, their participation in physical activity needs to be encouraged.

We are bound to draw attention to the limitations of the physical education programme in schools in England and Wales: less time is devoted to curricular physical education than in any other comparable country.[3] The curriculum is dominated by competitive team games despite unequivocal evidence to show that, with the possible exception of football for boys, they are not transferring to out-of-school participation.[4] A competitive ethos seems more acceptable to boys than to girls, but, notably, not acceptable to low-exercising boys; many girls reject competition, even in the high exercisers.[5] Individual sports and activities are far more popular with girls than competitive team games.[4] The dislike of many girls for team games may well contribute to their low levels of physical activity. In our view young people need to be exposed to a balanced programme of competitive, cooperative, individual, partner, and team activities, to lay the foundation for present and future physical activity.

**Neil Armstrong, Brian Kirby*
Institute of Clinical Sciences, University of Exeter, Exeter EX2 5DW, UK

1 Steptoe A, Butler N. Sports participation and emotional wellbeing in adolescents. *Lancet* 1996; 347: 1789–92.
2 Armstrong N, Balding J, Gentle P, Kirby B. Patterns of physical activity among 11 to 16 year old British children. *BMJ* 1990; 301: 203–05.
3 Armstrong N, McManus A. Children's fitness and physical activity—a challenge for physical education. *Br J Phys Educ* 1994; 25: 20–26.
4 Sports Council. Young people and sport: national survey selected findings. London: Sports Council. Young people and sport in England, 1994. London: Sports Council, 1996.
5 Gentle P, Caves R, Armstrong N, Balding J, Kirby B. High and low exercisers among 14 and 15 year old British children. *J Publ Hlth Med* 1994; 16: 186–94.

Jewish women, breast cancer, and commercial risks

SIR—I was astonished by *The Lancet's* publication of two papers on breast cancer in Jewish women, and wondered why the editors decided they were appropriate for such a highly reputable and influential journal. In the paper by Offit and colleagues (June 15, p 1643)[1] the authors stated that "all participants were given the option not to know their test results". However, I submit that the women's employers, people from insurance companies, or bankers might take a different—and inquisitive—view. They would be tempted to think that Jewish women would hide their test results. In the paper by Egan and colleagues (June 15, p 1645)[2] a strong association between Jewish religion alone and breast cancer was dismissed; however, a five times greater relative risk in the Jewish (compared with non-Jewish) subgroup with positive family history was publicly revealed. If I were a life insurance company official, I would ask Myriad Genetics Inc—one of whose researchers was a co-author with Offit and colleagues[1]—to screen blood samples from Jewish women. Insurance premiums could be raised, or future contracts rejected against Ashkenazi Jewish women. What is *The Lancet's* policy on the publication of papers that will not only have serious implications for the gene carriers themselves, but also for their relatives? What can be done to prevent the commercial exploitation of genetic information on the risk of lethal diseases?

Kazuo Komamura
Division of Cardiology, National Cardiovascular Centre, Suita, Osaka 565, Japan

1 Offit K, Kilewski T, McGuire P, et al. Germline *BRCA1* 185delAG mutation in Jewish women with breast cancer. *Lancet* 1996; 347: 1643–45.
2 Egan KM, Newcomb PA, Longnecker MP, et al. Jewish religion and risk of breast cancer. *Lancet* 1996; 347: 1645–46.