Individuals living in houses with a high radon concentration have a significantly increased frequency of chromosomal aberrations in peripheral lymphocytes, the α-dose being delivered mainly by the radon daughter nuclides ^{218}Po and ^{214}Po. Some epidemiological and geographical studies have suggested an association between α-particle exposure via radon and the occurrence of leukaemia, brain tumours, and kidney cancer, especially in children.[5]

The presence of ^{210}Po in vehicle exhausts may be unique in terms of what is known of its carcinogenic potential at environmental exposure levels. In our view it should be added to the list of potential carcinogens from motor vehicles to which the population is exposed.

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1 House of Commons Transport Committee. 6th report: transport-related air pollution in London. London: HM Stationery Office, Oct 19, 1994.
2 Sir John Houghton (Chairman). Transport and the environment. Royal Commission on Environmental Pollution, 18th report, Oct, 1994.
3 Henshaw DL, Allen JE, Keitch PA, Randle PH. The spatial distribution of naturally occurring ^{210}Po and ^{226}Ra in children's teeth. *Int J Rad Biol* 1994; **66:** 815–26.
4 Kadhim MA, Lorimore SA, Hepburn MD, Goodhead DT, Buckie VJ, Wright EG. α-particle-induced chromosomal instability in human bone marrow cells. *Lancet* 1994; **344:** 987–88.
5 Henshaw DL, Eatough JP, Richardson RB. Radon: a causative factor in the induction of myeloid leukaemia and other cancers in adults and children? *Lancet* 1990; **335:** 1008–12.

WHO's current policies and plans for reform

SIR—In your Jan 28 editorial you make an ill-informed attack on the World Health Organization. By coincidence, your editorial appeared as the Executive Board of WHO concluded one of its most constructive sessions in recent years, during which it endorsed a wide range of important reforms and helped to set its targets and priorities for the future. No one from *The Lancet* attended any of the board's eleven working days.

Far from adopting "a fortress mentality, fighting bitterly to retain the status quo and meanwhile allowing goodwill and opportunities for revitalisation to slip away", WHO is dedicating itself to radical change, to greater openness and to closer relations with its partners in health. You seem out of touch with WHO's current policies and reforms, and make sweeping and inaccurate assertions. For example, you say "the mental and societal dimensions of health are virtually ignored"—but WHO's Division of Mental Health made world headlines with a major 14-country study of mental illness published in December, 1994, in the *Journal of the American Medical Association.* Another example is, "Appropriate respect for national sovereignty has been overtaken by blind obeisance to narrow national wishes—eg, the desire to avoid embarrassment, as in admitting that an epidemic is underway". WHO intervened directly in the plague outbreak in India last September. The Director-General went to India, met the Prime Minister and Health Minister, and set up in India an international team that thoroughly investigated the outbreak and produced a full report.

You say that WHO "remains essentially unaccountable", but it is directly accountable to each and all of its 189 member states (whom you also describe as its "official shareholders") through the Executive Board and the World Health Assembly. The Board, in its discussions of the WHO budget during the past two weeks, has shown that it has no doubts of our accountability.

You omit recognition of some undisputed facts: that WHO policies and activities result in the savings of millions of lives a year, especially those of children in the developing world (WHO had a key role in rapidly bringing under control the deadly cholera epidemic among Rwandan refugees in Zaire last year); that WHO is regarded world-wide as the leading authority on health; and that the standards it sets and maintains are the yardsticks by which countries of the world measure their own health status.

WHO welcomes constructive criticism and responds positively to it. Our new communications policy will support such a dialogue. But it has become fashionable lately to attack WHO, especially in the public health community, without much apparent thought to the harm that this does not merely to WHO, but also to all those we seek to serve.

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Red wine consumption and oxidation of low-density lipoproteins

SIR—There is growing interest in the hypothesis that red wine phenolics can protect low-density lipoproteins (LDL) against oxidative modification, thereby reducing the risk of cardiovascular morbidity. Wine flavonoids inhibit Cu^{2+}-mediated oxidation of LDL when added in vitro,[1] but the effect of flavonoids eaten with foods is unclear. Although Kondo and colleagues (Oct 22, p 1152) report that red wine (Château Lagrange, 1989) consumption for two weeks was associated with an increased resistance of LDL against oxidative modification, enthusiasm for their hypothesis should be tempered. We have assessed whether the consumption of alcohol-poor red wine affects susceptibility of LDL to Cu^{2+}-mediated oxidative modification.

24 healthy non-smoking normolipidaemic volunteers consumed white wine for two weeks (baseline period). They were then randomly assigned to consumption of 550 mL (about 4–5 glasses) daily of white (Loire, 1993) or red wine (Italian Chianti Classico, 1991) for 4 weeks (test period), with stratification for age, sex, and plasma cholesterol concentration. Before distribution, the alcohol content of both wines was reduced to 3% by evaporation at 35°C without affecting the flavonoid concentrations of the wines. Subjects followed a flavonoid-poor diet and abstained from tea and red wine. Subjects were not taking vitamin or mineral supplements. Fasting blood samples were obtained on two days at the end of the baseline period and of the test period, to determine the susceptibility of LDL to oxidation (variation coefficients <3%). Plasma samples were stored at −80°C under nitrogen after the addition of saccharose to stabilise the lipoproteins. Lag times and oxidation rates,

	Red wine (n=13)	White wine (n=11)
M/F ratio	3/10	2/9
Age (yr)	38·2 (9·8)	36·4 (11·8)
Plasma cholesterol (mmol/L)	5·25 (0·71)	5·37 (0·82)
Lag time (min)		
Baseline period	61·8 (7·7)	64·5 (10·4)
Test period	62·7 (11·8)	63·3 (10·8)
Oxidation rate (nmoL dienes/mg LDL protein per min)		
Baseline period	13·9 (2·1)	14·1 (3·1)
Test period	14·6 (2·3)	13·3 (2·4)

M=male, F=female.

Table: **Subjects' details and in-vitro oxidation characteristics of LDL**

indicating the resistance of LDL against oxidative modification, were closely similar in both wine groups (table).

Thus, in contrast with Kondo and colleagues' findings, in our hands daily consumption of flavonoid-rich red wine did not influence the oxidisability of LDL. Variables that might account for the differences are the use of different brands of red wine, the ethanol content of red wine as an unwanted confounding variable, and the analytical precision of the assay method used. Our findings do not accord with the proposed beneficial effect of red wine consumption on LDL oxidation.

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1 Frankel E, Kanner J, German J, Parks P, Kinsella JE. Inhibition of oxidation of human low-density lipoprotein by phenolic substances in red wine. *Lancet* 1993; **341:** 454–57.

Carcinogenicity of coal-tar shampoo

SIR—van Schooten and colleagues report (Nov 26, p 1505) the absorption of polycyclic aromatic hydrocarbons and the excretion of 1-hydroxypyrene (1-OH-P) after application of a 12·5% coal-tar solution. Coal-tar solution is derived from crude coal tar which contains polycyclic aromatic hydrocarbons. The level of urinary excretion of the polycyclic aromatic hydrocarbons metabolite 1-OH-P has been used as a measure of occupational exposure to these hydrocarbons and a reflection of the degree of dermal absorption of them from coal-tar-containing products.

van Schooten and co-workers examined the urinary excretion of 1-OH-P in 11 volunteers in the Netherlands for two days after a single hairwash consisting of two applications for 20 s each with a coal-tar shampoo. A peak urinary 1-OH-P of about 13 nmol was shown at about 24 h after application during the two-day period of this study. According to their findings, this level of urinary 1-OH-P is equivalent to that recorded in occupationally exposed coke-oven workers. Details of coal tar exposure, medical condition, occupation, and smoking status of the subjects tested were not noted. The results were for a limited number of subjects and a small control group.

The potential carcinogenic properties of therapeutic coal-tar-containing products and the risks associated with their use have previously been evaluated. The relevance of van Schooten's results should be assessed in relation to data for the potential carcinogenicity of coal-tar shampoos. In the USA coal tar is monographed by the Food and Drug Administration (FDA) as category I GRAS/E (generally regarded as safe and effective) for over-the-counter use in the treatment of dandruff, seborrhoea, and psoriasis.[1] A risk analysis of the potential carcinogenicity of a coal-tar-containing solution shampoo would be based on several clinical variables that are specific to this mode of exposure and which differ from those of occupationally-exposed workers. These factors include the amount of shampoo used, the duration and extent of application, the number of applications per use, the frequency of application, the residual amount left on the scalp, and the coal tar absorption rate and amount.

Hansen and co-workers' results[2] suggest that raised urinary 1-OH-P concentrations might not persist after continuous exposure to polycyclic aromatic hydrocarbons. In two patients treated once a day with coal-tar pitch covering more than 50% of the skin, the concentration of urinary 1-OH-P was increased about 100-fold. However, after three weeks of continuous daily coal-tar treatments, urinary excretion of 1-OH-P returned to baseline values. The long-term effects on urinary excretion of a coal-tar shampoo such as used by van Schooten and colleagues would be of interest.

Data that specifically link coal tar polycyclic aromatic hydrocarbons components such as benzo(a)pyrene to an increased risk for cancer in exposed populations are inadequate. Although lung cancer has resulted from polycyclic aromatic hydrocarbons exposure such as cigarette smoke, roofing tar, and coke-oven emissions, it cannot be concluded that benzo(a)pyrene is the causative agent.[3] In particular, there are no reported studies specifically linking use of coal-tar-solution-containing shampoos to an increased risk of cancer. Current evidence suggests that the carcinogenic risk of coal tar, if it exists, is small. The US FDA, with respect to labelling, generally warns only of the risks that are scientifically documented, clinically significant, and important for the safe and effective use of products by average consumers.

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1 Dandruff, seborrheic dermatitis and psoriasis drug products for over-the-counter human use; tentative final monograph; Federal Register, 51 (146) p 27 346–60. Compilation of OTC drug regulations. Nonprescription Drug Manufacturers Assocation, Tab 20 p 286.
2 Hansen AM, Poulsen OM, Menne T. Longitudinal study of excretion of metabolites of polycyclic aromatic hydrocarbons in urine from two psoriatic patients. *Acta Derm Venereol* 1993; **73:** 188–90.
3 United States Environmental Protection Agency (EPA), Dermal exposure assessment: principles and applications. EPA/600/8–91/011B. Interim report. Washington DC: Office of Health and Environmental Assessment, 1992.

Bird attacks on milk bottles and campylobacter infection

SIR—Species of campylobacter are the most commonly reported causes of gastrointestinal disease in the UK.[1] The outstanding feature of campylobacter epidemiology is the rise in isolations reported by laboratories over a 3–4 week period in the spring. In Wales the spring peak in 1994 was in week 21 (ending May 27), with a 3-fold increase in reports over the preceding 3 weeks (figure). Previous studies in the UK have identified drinking milk from bottles whose tops had been pecked by magpies and jackdaws as sources of infection.[2–4] But colleagues still have difficulty in accepting this explanation. To find out if birds pecking milk bottle tops is still a significant public health problem in Wales we monitored human campylobacter infections from April to June, 1994, in collaboration with environmental health departments. 21 departments agreed to ask questions about milk bottle attacks as part of routine interviews with cases. Between April 1 and June 30, 657 cases were reported to these departments by laboratories; 551 (84%) of patients were interviewed and 93 (17%) reported drinking milk from bottles attacked by birds in the week before onset (table). In

Number	April	May	June
Of campylobacter infections reported	112	226	319
Interviewed	87	182	282
Reporting milk bottle attacks	1	54 (30%)*	74 (26%)
Reporting drinking milk from such bottles	1	42 (23%)	50 (18%)

*Of those interviewed.

Table: **Results of enhanced surveillance of campylobacter infection in Wales (1994)**