

housing projects on grounds other than health. This should certainly be possible for cold, damp homes, which deteriorate rapidly through corrosion, timber decay, and electrical problems and are expensive to maintain.

The problem of cold damp houses should be tackled because of its effects on health and its economic effects on the housing stock. Standards should be set so that an acceptable safe indoor temperature, say 20°C, can be achieved at no more than 10% of the household income. Any excess needed should be provided in social payments.

Although most houses can be made warm and dry if enough is spent on heating, this is not the most economical solution. Where possible, structural defects that promote cold and condensation should be repaired so that properties can be brought up to the standard. Measures such as fungicidal washes and paints should be used only for temporary relief.¹⁷ Public sector houses should be improved with specially allocated funds, and grants should be available to encourage private owners to upgrade their properties. No family should be condemned to live in fuel poverty.

1 Curwen M, Devis T. Winter mortality, temperature and influenza: has the relationship changed in recent years? *Population Trends* 1988;54:17-20.

- 2 Mant DC, Gray JAM. *Building regulation and health*. Garston: Department of the Environment Building Research Establishment, 1986.
- 3 Parker M. *Homes for today and tomorrow*. London: HMSO, 1961. (Ministry of Housing and Local Government.)
- 4 Department of the Environment. *English house condition survey 1986*. London: HMSO, 1988.
- 5 Markus TA. *Cold, condensation, climate and poverty in Glasgow*. Warwick: Legal Research Institute, University of Warwick, 1987. (Unhealthy housing: prevention and remedies.)
- 6 Boardman B. *Defining affordable warmth*. Warwick: Legal Research Institute, University of Warwick, 1987. (Unhealthy housing: prevention and remedies.)
- 7 Institution of Environmental Health Officers. *Background notes on condensation*. London: IEHO, 1983.
- 8 Burr ML, St Leger AS, Yarnell JWG. Wheezing, dampness, and coal fires. *Community Med* 1981;3:205-9.
- 9 Burr ML, Miskelly FG, Butland BK, Merrett TG, Vaughan-Williams E. Environmental factors and symptoms in infants at high risk of allergy. *J Epidemiol Community Health* 1989;43:125-32.
- 10 Burr ML, Mullins J, Merrett TG, Stott NCH. Indoor moulds and asthma. *J R Soc Health* 1988;108:99-101.
- 11 Strachan DP, Elton RA. Relationship between respiratory morbidity in children and the home environment. *Family Practice* 1986;3:137-42.
- 12 Strachan DP. Damp housing and childhood asthma; validation of reporting of symptoms. *Br Med J* 1988;297:1223-6.
- 13 Strachan DP, Sanders CH. Damp housing and childhood asthma; respiratory effects of indoor air temperature and relative humidity. *J Epidemiol Community Health* 1989;43:7-14.
- 14 Martin CJ, Platt SD, Hunt SM. Housing conditions and ill health. *Br Med J* 1987;294:1125-7.
- 15 Platt SD, Martin CJ, Hunt SM, Lewis CW. Damp housing, mould growth, and symptomatic health state. *Br Med J* 1989;298:1673-8.
- 16 World Health Organisation. *Alma-Ata 1978; primary health care*. Geneva: WHO, 1978. (Health for all series. No 1.)
- 17 Bravery AF, Grant C, Sanders CH. *Controlling mould growth in housing*. Warwick: Legal Research Institute, University of Warwick, 1987. (Unhealthy housing: prevention and remedies.)

Letter from Brasilia

Growing problem of malaria

Philip D Marsden

At 2 30 am the telephone rang; it was a resident at a hospital in a satellite town of Brasilia worried about a case of malaria. Such calls have always been a feature of my life. Not just in endemic areas such as equatorial Africa, New Guinea, and Brazil but also in the big cities: London, New York, Sydney, etc. For today malaria travels fast, and it is a real emergency so I always get up and go. First, however, I wanted information. The resident thought he had seen malarial rings in a thin film stained with giemsa. I asked him to make a thick film and have the microscope focused on what he saw when I arrived. Then vital questions regarding the patient's mental state and his urine output. The brain and kidney are target organs in severe malaria. The patient was confused, but he had passed urine that unfortunately had not been saved and the volume had not been measured. His wife said that he was an engineer working on a hydroelectric project in the upper Amazon and had been taken ill on the plane with fever, rigors, and sweating.

At 3 30 am I was in front of the microscope. The resident was right for there was a fine blue ring with a purple nucleus in the centre of the erythrocyte. I saw few parasites in the thin film, but the thick film (a 20+ blood concentration) showed a profusion of fine rings and cytoplasmic streaks with one or two nuclei. No schizonts, no gametocytes; diagnosis *Plasmodium falciparum*, the most dangerous of the four parasites causing human malarias and the commonest here in Brasilia. I rated the density in the thick film as 15/1—that is, 15 parasites in one oil immersion field. To the resident's sorrow, for he was tired, I turned again to the thin film to look along the margin of the smear. Heavily parasitised erythrocytes such as schizonts and gametocytes get pushed to the edge of the smear with white cells, and I just wanted to check that there wasn't a

double infection with *P vivax* or *P malariae*. There wasn't.

Then we went to see the patient, who was restlessly tossing in bed with a hot, sweaty skin and staring unfocused eyes. He did not seem to be clinically anaemic, but jaundice could not be assessed in the artificial light. It was impossible to palpate his spleen for he could not cooperate. The bed sheet was wet with urine, which seemed to be normal in colour, a good sign. He had been in the ward for five hours and had not vomited so we gave him six tablets of mefloquine ground up through a gastric tube. I left the usual instructions about investigations in the morning, warned that blood parasites might disappear only on the second day, and got to bed at dawn. I was not contacted again.

Malariology like leprology is sufficiently complicated to be a discipline in its own right, and in a brief communication such as this I can touch on only some of the points relevant to Brasilia. There is a great movement of people in and out of the city, mainly by air from the Amazon regions. In Brazil 95% of malaria is contracted in the states of this region: Para, Amazonas, Roraima, Rondonia, Acre, Mato Grosso, Maranhao, etc. Often companies do not advise their employees of the risk of malaria or arrange chemoprophylaxis. But then this is a global problem. I think of a French baby of 18 months who died in the Gambia or an air hostess who slept in Lagos, Nigeria, for four hours and died in London two weeks later. Tourist companies are the worst culprits because it is bad for business if you make much of malaria in Malawi, yellow fever in Youndi, or kala-azar in Cannes. Much stronger international legislation is needed, and I hope that the European Community will take a lead on this matter. Outside the army things are equally bad in the United States.

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Atomisers used in insecticidal work

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The recent invasion of the Amazon area by non-immune people is creating serious health problems. Ten years ago the situation regarding malaria was much more satisfactory. I travelled extensively in the Amazon around that time, and the ministry campaign against malaria (SUCAM) had it fairly well controlled. Brazil was fortunate in that the major anophelene vector, *Anopheles darlingi*, had not developed genetic resistance to dichlorodiphenyltrichlorophane (DDT). The fleet of small boats in Manaus would go up the multitude of rivers spraying the houses: people live mainly on the banks of a river as it is the only route of cheap transport. Naturally eradication was not

possible but the level of control was high. At that time *P falciparum* resistant to chloroquine had been reported but was still uncommon; now it is the rule. Also *A darlingi* has developed so called behavioural resistance; it is no longer resting on the sprayed walls but bites its hosts around dwellings. Therefore the malaria problem grows. Also a large part of this population influx is speculators mining mainly for gold or diamonds or cattle rearing. The miners are the worst because they are virtually impossible to control, flying into remote areas, often secretly, because of the lure of what is beneath the ground. Nobody knows how many mining concerns there are in this region but it must be hundreds. The ministry struggles with the problem of controlling malaria among them as well as the transmission of AIDS. It is a herculean task.

Development of antimalarial drugs has been related to wars and the availability of quinine. After the second world war six groups of antimalarials were available but *P falciparum* has a remarkable capacity to develop resistance. The Vietnam war highlighted the problem for the American army, which set up an admirable drug screening programme at the Walter Reed Army Institute of Medical Research in Washington. To date this programme has screened 300 000 compounds for antimalarial activity. Mefloquine is the result of this programme, but because it is another quinoline (like quinine) resistance has already been reported, though it is still unknown in Brazil. Such powerful, effective schizonticides must be rigorously controlled otherwise indiscriminate use will result in resistance as occurred with chloroquine. Today the situation for treating severe falciparum malaria in Brazil is better than it has been for some years, as apart from mefloquine there are other new schizonticides to which resistance is still unknown.

Scientifically Speaking

Growing catalogue of fraud

Bernard Dixon

"Why does this book fail? Primarily because the authors took reports of scientific fraud and strung them together, claiming that their analysis would reveal something profound about science. It doesn't. From fraud, one only learns about fraud."

Thus the distinguished molecular biologist Norton Zinder of Rockefeller University, New York, writing in *Science* 83 (January/February 1983, p 94). His target was William Broad and Nicholas Wade's *Betrayers of the Truth*, published by Simon and Schuster some months earlier. In private, as I know from talking to Zinder around that time, he was rather more intemperate in his criticisms of a book which had for the first time painted a synoptic view of cheating and data fabrication in science. Despite the existence of such classics as the Piltdown forgery (an undoubted contrivance) and Gregor Mendel's too perfect breeding data (tidied up by an over zealous assistant?), most scientists fiercely resisted the notion that dishonesty was an endemic part of the scientific enterprise. Peer review, at the heart of research evaluation, rendered the very idea absurd.

We now know otherwise. Month by month throughout the 1980s the catalogue of fraud has grown. Retractions, corrections, and warnings in scientific journals have become if not commonplace then

certainly no longer rare and conspicuous exceptions to the passing parade of "normal science." Even within the scientific community there has been a gradual, grudging acceptance that numbers of biologists and physicists, chemists and psychologists *do* sometimes depart from the conventionally rigorous standards of their profession—and that the temptation to do so has been aggravated by the financial and other pressures of modern, highly professionalised and politicised science.

Political interference

Unfortunately, however, we are now reaping ill rewards from scientists' own haughty rejection of suggestions of dishonesty at the beginning of the decade. Indeed, politicians in the United States have latched on to the cheating phenomenon with such zeal that conventional scientific intercourse is itself now in serious danger of being distorted and corrupted by political interference.

Consider the following (true) story. In April 1986 two collaborating laboratories published a paper in *Cell*, describing the way in which a foreign gene inserted to make transgenic mice affected antibody production by existing genes. The principal authors

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