

initial  $^{134}\text{Cs}$  to  $^{137}\text{Cs}$  ratio was 0.5, then the  $^{134}\text{Cs}$  whole body activity rises to a maximum of 178 Bq after 100 days and reduces to half that in the next 180 days, whereas the predicted value at the midpoint of the present measurement period is 152 Bq.

This simple model, based on milk consumption alone, predicts whole body activities of  $^{134}\text{Cs}$  and  $^{137}\text{Cs}$  at the time of our present measurements with reasonable accuracy. Given seasonal changes in food production and consumption, the longer term validity of the model cannot be guaranteed, but it is instructive to calculate the whole body radiation doses based on the activity-time predictions of the model.

The predicted average additional effective dose equivalent for 1986 due to the contamination from Chernobyl is 4.7  $\mu\text{Sv}$  for  $^{134}\text{Cs}$  and 6.9  $\mu\text{Sv}$  for  $^{137}\text{Cs}$  (+  $^{137\text{m}}\text{Ba}$ ) whereas the predicted average committed effective dose equivalent is 7.2  $\mu\text{Sv}$  for  $^{134}\text{Cs}$  and 11.6  $\mu\text{Sv}$  for  $^{137}\text{Cs}$  (+  $^{137\text{m}}\text{Ba}$ ). These predicted doses are relatively small compared to those resulting from naturally occurring  $^{40}\text{K}$ , (174  $\mu\text{Sv}$  per annum for males and 116  $\mu\text{Sv}$  per annum for females) and the combined committed dose for both Cs radioisotopes amounts to only 1% of the total average annual effective dose equivalent due to natural background radiation<sup>4</sup>.

I thank Mr D. Smith of the NRPB for his help.

W.S. WATSON

Department of Clinical Physics  
and BioEngineering,  
Southern General Hospital,  
Govan Road,  
Glasgow G51 4TF, UK

1. Williams, E.D. *et al.* *Health Phys.* **40**, 1-4 (1981).
2. Ministry of Agriculture, Fisheries and Food Household Food Consumption 1984: A. Rep. of the National Food Survey Committee (HMSO, London, 1986).
3. International Commission on Radiological Protection *Limits for Intakes of Radionuclides by Workers* ICRP Publ. 30 (Pergamon, Oxford, 1979).
4. Fry, F.A. *et al.* *NRPB R121* (HMSO, London, 1981).

## Origin of HTLV-I virus in Japan

SIR—If the human T lymphotropic virus I (HTLV-I) was brought to Japan from Africa by the Portuguese in the sixteenth century as in the proposal of Gallo *et al.*<sup>1,2</sup>, which is the subject of controversy<sup>3-6</sup>, the virus should be prevalent in India, Ceylon and Malacca, where the Portuguese constructed their bases long before arriving in Japan. Yet, recently, we tested 90 sera in Bombay for HTLV-I antibody and found none of them to be positive<sup>7</sup>.

Portuguese missionaries began their activities in Japan in 1559 and the Portuguese dominated trading in Japan until the seventeenth century. Japanese history has no record of African descendants in Kyusyu. I therefore agree with the hypothesis of Ishida and Hinuma<sup>6,8</sup> that

HTLV-I was originally carried by prehistoric Japanese (Asiatic, Caucasoid, Ainu and Ryukyuan). Interestingly, the Ainu are ethnically related to the Eskimos, who have a high prevalence of HTLV-I, according to Gallo and his colleagues<sup>9</sup>.

So, are all HTLV-I positive populations in Japan directly related to Ainu or Ryukyuan? I suggest that HTLV-I was once enclosed in Okinawa and was first transmitted to Kyusyu through the trade between Okinawa and southern province of Kyusyu (Kagoshima). Further expansion of HTLV-I to other coastal areas of Japan might have been carried out by fishermen along the ocean current *Kuroshio*. Until 100 years ago, the movement of ordinary people beyond province borders was strictly limited by *Shogun* but, even then, fishermen could travel rather freely by ship. The situation is well demonstrated in Kochi prefecture<sup>10</sup> where a higher rate of HTLV-I positivity is found in the east and west fishermen's bases than in the central area. Thus, it is clear that evidence for the hypothesis of an African origin of HTLV-I in Japan is still lacking.

HIROKUNI TAGUCHI

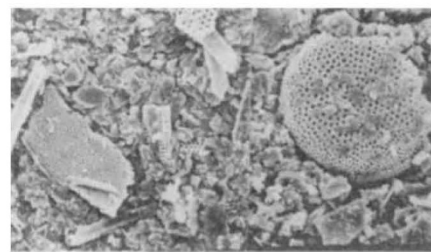
Department of Internal Medicine,  
Kochi Medical School,  
Kochi 781-51, Japan

1. Gallo, R.C., Sliski, A.H. & Wong-Staal, F. *Lancet* **ii**, 962-963 (1984).
2. Wong-Staal, F. & Gallo, R.C. *Nature* **317**, 395-403 (1985).
3. Rosenior, J. *Nature* **318**, 728 (1986).
4. Gallo, R.C. & Sliski, A.H. *Nature* **320**, 219 (1986).
5. Kantha, S.S. *Nature* **321**, 733 (1986).
6. Ishida, T. & Hinuma, Y. *Nature* **327**, 504 (1986).
7. S.H. Advani *et al.* *Ind. J. med. Res.* (in the press).
8. Ishida, T. *et al.* *J. Infect.* **11**, 153-157 (1985).
9. Robert-Guroff, M. *Int. J. Cancer* **36**, 651-655 (1985).
10. Taguchi, H. *et al.* *Lancet* **ii**, 1029 (1983).

## Diatom mystery

SIR—The mystery object in Wolstenholme's chromosome preparation<sup>1</sup> is a fragment of the silica skeleton of an alga known as a diatom. We had significant problems with contamination from this tiny organism's remains for several years. Eventually we accidentally discovered that the source of the contamination was diatomaceous particles from rubber bulbs placed on Pasteur pipettes, used for the harvesting phase of chromosome preparations<sup>2</sup>. Diatomaceous earth is apparently used by some manufacturers in the production of rubber goods.

Diatom particles clung to many cells, cutting their membranes and releasing the contents (in the same manner that diatomaceous earth is used as an organic insecticide). This diminished the numbers of well-spread chromosome metaphases. The particularly vulnerable cells in this procedure were those in metaphase (and therefore without a nuclear membrane) which were under internal osmotic pressure due to prior hypotonic treatment. The greater the dose of diatomaceous frag-



Diatom fragments on inner surface of rubber bulb. Left,  $\times 880$ ; right,  $\times 1,190$ .

ments, the more cells in metaphase that were affected, and fewer spreads were analysable.

THOMAS S. MCCONNELL  
GERALD T. ALLGOOD  
PATRICIA O. BACA  
GLENDA E. MILLER  
ROBERT E. WATERMAN

Departments of Pathology and Anatomy,  
The University of New Mexico  
School of Medicine,  
Albuquerque, New Mexico 87131, USA

1. Wolstenholme, J.W. *et al.* *Nature* **323**, 300 (1986).
2. McConnell, T.S. *et al.* *Karyogram* **11**, 59-60 (1985).

SIR—When I showed the crossword puzzle-like but only 10-micrometre "mystery object amid the chromosomes" reported by Wolstenholme *et al.*<sup>1</sup> to my mythical friend Dr Orpheus of the Institute of Neuropoetics, he responded with the following:

The late Al Lehninger, of textbook<sup>2</sup> fame,  
In 1966 was deemed insipid  
By players of nucleic acid's game  
For speculating<sup>3</sup> that a membrane's lipid  
Might code environmental information  
Across its two-dimensional array,  
A thought that called forth righteous  
indignation  
From priests of one-dimensional DNA,

But just as viruses, which travel light,  
Have usefully evolved the economic  
Reading frame-shifts, to the left or right,  
And then both left and right, or palindromic,  
So that, as if possessed of frugal wits  
That make the most of being just a line  
Of four-state points that can't bear many bits,  
The Palimpsest-like viruses do fine,

So, likewise to save space, eukaryotic  
Cells, to which the neurones dictate chatter,  
Must complement their chromosomes' demotic  
Morse-like coding of genetic matter  
By interlocking news Across and Down  
Their protein-punctured lipid membranes,  
As shown by Wolstenholme *et al.*<sup>1</sup> . . .

Don't frown,  
Oh 1-D dogmatists! (Did God give them  
brains?)

TED MELNECHUK

Helicon Foundation,  
4622 Sante Fe Street, San Diego,  
California 92109, USA

1. Wolstenholme, J. *et al.* *Nature* **323**, 300 (1986).
2. Lehninger, A.L. *Biochemistry*, 2nd edn (Worth, New York, 1975).
3. Lehninger, A.L. in *Neurosciences Research Symposium Summaries* Vol. 1 (eds Schmitt, F.O. & Melnechuk, T.) 55 (MIT Press, Cambridge, 1966).