

workload involves the care of individuals with various stages of dementia, though the potential symptomatic benefits of cholinesterase inhibitors are denied my patients through inadequate resourcing of memory clinics and the artificially restrictive constraints of prescribing the drugs; National Institute of Clinical Excellence (NICE) guidelines use a mini-mental state examination as the principle criterion for deciding appropriateness of a therapy, for which the practical benefits are with behavioural improvements and reductions in anxiety.² As a result, a 75 year old with mild dementia is more likely to receive a cocktail including aspirin, an angiotensin converting enzyme inhibitor, a statin, and a β blocker as preventive therapy for their possible angina, than medication with potential symptomatic benefits that could have a great effect on their independence and functional state.

Unfortunately, we will probably continue with the simplistic application of evidence-based medicine through guidelines and misguided rationing. We are now in a position in which we have to justify the withholding of potentially protective cardiac medications, where general consensus and guidelines have that everyone receives them, often despite a fairly small reduction in risk. Guidelines seem to be getting priority over the individual patient, and the problem is compounded through audit and targets. We should focus our limited resources better, though to do so will require a major change to our approach to modern medicine. We are already losing our clinical freedom, and look back fondly to the days when we could use our experiential knowledge and prescribe the treatments felt to be most beneficial to the patient.

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- 2 National Institute for Clinical Excellence. Donepezil, rivastigmine and galantamine for the treatment of Alzheimer's disease: technology appraisal guidance No 19, Jan 2001. <http://www.nice.org.uk/article.asp?a=14487> (accessed April 13, 2003).

SARS—a clue to its origins?

Sir—We detected large quantities of viable microorganisms in samples of stratospheric air at an altitude of 41 km.^{1,2} We collected the samples in specially designed sterile cryosamplers carried aboard a balloon launched from the

Indian Space Research Organisation/Tata Institute Balloon Facility in Hyderabad, India, on Jan 21, 2001. Although the recovered biomaterial contained many microorganisms, as assessed with standard microbiological tests, we were able to culture only two types; both similar to known terrestrial species.² Our findings lend support to the view that microbial material falling from space is, in a Darwinian sense, highly evolved, with an evolutionary history closely related to life that exists on Earth.

We estimate that a tonne of bacterial material falls to Earth from space daily, which translates into some 10^{19} bacteria, or 20 000 bacteria per square metre of the Earth's surface. Most of this material simply adds to the unculturable or uncultured microbial flora present on Earth.

The injection from space of evolved microorganisms that have well-attested terrestrial affinities raises the possibility that pathogenic bacteria and viruses might also be introduced. The annals of medical history detail many examples of plagues and pestilences that can be attributed to space incident microbes in this way. New epidemic diseases have a record of abrupt entrances from time to time, and equally abrupt retreats. The patterns of spread of these diseases, as charted by historians, are often difficult to explain simply on the basis of endemic infective agents. Historical epidemics such as the plague of Athens and the plague of Justinian come to mind.

In more recent times the influenza pandemic of 1917–19 bears all the hallmarks of a space incident component: “The influenza pandemic of 1918 occurred in three waves. The first appeared in the winter and spring of 1917–1918 . . . The lethal second wave . . . involved almost the entire world over a very short time . . . Its epidemiologic behaviour was most unusual. Although person-to-person spread occurred in local areas, the disease appeared on the same day in widely separated parts of the world on the one hand, but, on the other, took days to weeks to spread relatively short distances.”³

Also well documented is that, in the winter of 1918, the disease appeared suddenly in the frozen wastes of Alaska, in villages that had been isolated for several months. Mathematical modelling of epidemics such as the one described invariably involves the ad hoc introduction of many unproven hypotheses—for example, that of the superspreader. In situations where proven infectivity is limited only to close contacts, a superspreader is someone who can, on occasion, simultaneously infect a large number of susceptible individuals, thus causing the sporadic emergence of new clusters of disease. The recognition of a

possible vertical input of external origin is conspicuously missing in such explanations.^{4,5}

With respect to the SARS outbreak, a *prima facie* case for a possible space incidence can already be made. First, the virus is unexpectedly novel, and appeared without warning in mainland China. A small amount of the culprit virus introduced into the stratosphere could make a first tentative fall out East of the great mountain range of the Himalayas, where the stratosphere is thinnest, followed by sporadic deposits in neighbouring areas. If the virus is only minimally infective, as it seems to be, the subsequent course of its global progress will depend on stratospheric transport and mixing, leading to a fall out continuing seasonally over a few years. Although all reasonable attempts to contain the infective spread of SARS should be continued, we should remain vigilant for the appearance of new foci (unconnected with infective contacts or with China) almost anywhere on the planet. New cases might continue to appear until the stratospheric supply of the causative agent becomes exhausted.

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- 1 Harris MJ, Wickramasinghe NC, Lloyd D, et al. The detection of living cells in stratospheric samples. *Proc. SPIE Conference 2002*; **4495**: 192–98.
- 2 Wainwright M, Wickramasinghe NC, Narlikar JV, Rajaratnam P. Microorganisms cultured from stratospheric air samples obtained at 41 km. *FEMS Microbiol Lett* 2003; **218**: 161–65.
- 3 Weinstein L. Influenza: 1918, a revisit? *N Engl J Med* 1976; **6**: 1058–60.
- 4 Hoyle F, Wickramasinghe NC. *Diseases from Space*. London: JM Dent, 1979.
- 5 Wickramasinghe C. *Cosmic dragons: life and death on our planet*. London: Souvenir Press, 2001.

DEPARTMENT OF ERROR

Donnelly CA, Ghani AC, Leung GM, et al. *Epidemiological determinants of spread of causal agent of severe acute respiratory syndrome in Hong Kong*. *Lancet* 2003; **361**: 1761–66—In this Article (May 24), in the sixth sentence in the fifth paragraph of the Results section (p 1763), 48·5 days should be: “4·85 days”, and 10·71² days should be “10·71 days” (p 1764). In the first sentence of the sixth paragraph of Results (p 1764), 572·9² days should be “572·9 days” and 62·1² days should be “62·1 days”.

Ruan YJ, Wei CL, Ling AE, et al. *Comparative full-length genome sequence analysis of 14 SARS coronavirus isolates and common mutations associated with putative origins of infection*. *Lancet* 2003; **361**: 1779–85—In figure 3 of this Mechanisms paper (May 24), the sequence for the Hong Kong CUHKW1 isolate should be, from top to bottom: “TCTGCCCGCAACCCA”.