Mushroom cloud the result of a meteor?

SIR — On 9 April 1984, a number of airline pilots witnessed a mushroom cloud above the northern Pacific Ocean, about 400 kilometres off the east coast of Japan. The cloud was first witnessed by the pilots of Japan Airlines flight JAL 036 at 14:06 GMT (23:06 local time) while flying route A-90 eastbound near the checkpoint PAWES. The location of the first sighting was 38.5° N, 146.0° E. The pilot reported that a large spherical cloud rose from the cloud deck below his aircraft and expanded rapidly to its full height of 60,000–70,000 feet and its full diameter of 200 miles in something between 1 min 50 s and 2 min 30 s. There was a heavy deck of clouds at the 14,000 feet level, but visibility was fair because of moonlight. About 12 s later, near 42° N, 151° E, they felt light turbulence. JAL 036 dispatched a May-day call at 14:21 GMT, put the crew on oxygen, and descended. JAL 036 was diverted to Elmendorf Air Force Base in Anchorage, Alaska, where radioactivity tests were conducted, but no trace was found. There was no communications breakup, nor any problems with the aircraft instruments.

Radioactivity tests conducted on other aircraft reporting the cloud also found no trace. Subsequent interviews with those pilots showed that the cloud exerted no fireballs or flash of lights. However, slight luminosity of the cloud was reported by several pilots. The pilot of KLM 868 reported that at first the cloud was opaque, then as it got larger it became possible to see stars through it. The phenomenon was visible for about 55 min.

The event was investigated by the US Department of Defense, Japanese Defence Agency, Federal Aviation Administration (FAA) and many others. All investigations concluded that it was not a nuclear explosion and it was possibly a natural phenomenon, but they failed to give a reasonable explanation.

We have investigated the event by querying FAA pilot interviews, newspaper reports, weather satellite photographs, and geophysical data from various institutions. Geophysical data include seismic, hydrophone, atmospheric electricity, telluric current, geomagnetic, and microbarograph data. However, none of the data we have studied contains a detectable signal from this event. Measurements of SO_2 in the ozone layer made by the National Aeronautics and Space Administration were also negative, suggesting that it was not a volcanic event.

We also obtained comment and opinions from experts in various related fields about this event. Some of the possible explanations we have pursued are:

(1) A gaint cumulus cloud.
(2) A vapour plume from the Kaitoku Seamount, 1,500 km south-west of the event.
(3) Burning of rocket fuel causing the ionization of cloud particles and the subsequent formation of a plume.
(4) A meteor causing the ionization of cloud particles and subsequent formation of a plume.

Of these possibilities, we have concluded that only the meteor plume has sufficient energy to explain the event. Meteors can enter the Earth's atmosphere at almost any angle. The speed of a meteor ranges from 15 to 110 km s^{-1}, and its height ranges from 150 km to ground level. Meteors slow down considerably upon entering the Earth's atmosphere. Normally, when a meteor does reach the ground as a meteorite, it travels the final portion of its trajectory as a dark body; it is rarely luminous below heights of 10 to 20 km.

A meteor's energy is very high. For example, the muzzle velocity of a 30-calibre bullet is about 840 m s^{-1}. A meteoroid the weight of a bullet (10 g), entering the lower atmosphere at a speed of 30 km s^{-1} has as much kinetic energy as a bullet leaving a muzzle. At this speed, even cloud particles striking at the surface of the meteor can produce a shattering effect. Collisions with atmospheric particles ionize atoms and produce visible luminosity.

Our hypothesis is that a meteor encountered the cloud deck and almost simultaneously shattered into pieces. Most of the kinetic energy was converted to heat which evaporated cloud particles, and the hot gas formed a plume. Because of the high speed of the meteor, the shattering took place over a distance spanning a few tens of kilometres, thus forming a large plume. The large distance over which the shattering occurred explains why only a slight disturbance was felt by the pilot and why microbarographs in Japan failed to detect the signal. It explains why there was neither a fireball nor a flash of light associated with the event; and it also explains the slight luminosity of the mushroom cloud.

If our explanation is correct, the pilot's experience on the night of 9 April may be the first close encounter of an aircraft with a meteor. We would welcome readers' comments on this event.

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A mechanism for prion replication

SIR — The putative causative agents of some infectious diseases which attack the brain, such as Creutzfeldt-Jakob disease of man and scrapie of sheep, have been called[1] 'prions'. Prions consist of protein, and apparently they contain no nucleic acid[1], although this contention has been challenged[2]. As reviewed recently in News and Views[3], two alternative mechanisms have been proposed for the replication of prions. (1) Activation of transcription of a host gene coding for prion protein. (2) 'Reverse translation' or protein-directed protein synthesis. There is no direct evidence for either of these mechanisms and therefore the replication of prions remains an enigma, although possible mechanisms for self-replication of proteins have been proposed[4].

We wish to propose an alternative possible mechanism for the replication of prions, which could operate if prions are indeed devoid of nucleic acid.

Prions consist of a single major protein component called[5] PrP, of relative molecular mass 27,000-30,000. We propose that PrP is a very stable slow-acting proteolytic enzyme, which may produce brain pathology precisely because of its protease activity. We further propose that the mechanism by which PrP 'replicates' is proteolytic attack of a precursor protein (which we call pre-PrP) present in many cells, and certainly in the brain, having an as yet unknown normal function. This precursor is folded in a configuration that has no proteolytic activity but, being a 'substrate' of PrP, it can be proteolytically cleaved with production of a fragment (or domain) which is PrP itself. The newly released PrP is then able to digest other pre-PrP molecules, thus bringing about multiplication of an apparent 'infective agent'. This proteolytic multiplication is reminiscent of other well-known cases in which a proteolytic enzyme (such as pepsin) acts on a precursor (such as pepsinogen) splitting off a portion to yield more of the same active protease.

The model we propose is consistent with the 'autocatalytic' time course of the diseases in question, in which there is a long incubation period followed by rapid disease progression. Moreover, the prior existence of pre-PrP in the body would account for lack of immune response (characteristically observed in these diseases), and ought to be detected also in normal cells, either directly or through its messenger.

We hope experiments will be devised to test this hypothesis, which may have relevance to other so-called 'slow infections' and degenerative diseases of the brain.

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