Tunguska comes down to Earth

H. J. Melosh

FEW natural events this century have excited as much popular and scientific interest as the gigantic explosion that rocked the Siberian taiga near the Tunguska river on the morning of 30 June 1908. Second only to the Loch Ness monster and Bigfoot in the popular science press, the Tunguska explosion has moved otherwise reputable scientists to propose explanations ranging from antimatter meteorites to mini black holes to near-critical fissionable masses. One theory popular in 1930s Russia attributed it to the explosion of a nuclear-powered (!) spacecraft. And the UFO explanation seems prevalent in some quarters even today. However, serious scientific study has been converging on a more prosaic explanation wrapped up by Chyba and colleagues on page 40 of this issue[1].

At its simplest, this explanation holds that the Tunguska explosion was caused by the fall of a large meteorite that broke up and deposited its energy in atmospheric blast waves before reaching the ground. The essentials of this picture were established by L. A. Kulik, who made the first on-site investigations of the explosion in the years 1927–39. Kulik attributed some boggy depressions near the explosion site to meteorite impacts, a proposal that was later discredited. No impact crater or large meteorite fragments were ever found at Tunguska.

Kulik's work was refined by E. L. Krinov[2], who proposed that the explosion was created by a comet. The idea of a cometary impactor was strongly supported by more recent work[3], although the density of the required comet is very small, 0.01–0.001 g cm^{-3}. Compared to the density of roughly 0.6 – 1.0 g cm^{-3} reported for comet Halley[4,5], this would make the Tunguska object decidedly unusual. However, Chris Chyba, Paul Thomas and Kevin Zahnle now argue that a full consideration of the dynamics of a meteorite traversing the atmosphere shows that the Tunguska explosion is fully compatible with the entry of a roughly 30-m diameter meteorite of the common stony class.

Previous workers considered a low-density comet necessary because of the peculiar pattern of energy deposition: the arrangement of the flattened trees, microbarograph and seismic records all suggest a nearly point-like release of 10–20 megatons equivalent of TNT in the atmosphere about 10 km above the surface. Although the 'butterfly' pattern of the tree-fall does suggest some energy released along a sloping line, it is clear that most of the energy was deposited in a small region near the terminus of the giant meteor's flight. The only way for a meteorite (or comet) both to deposit the observed energy and to stop in the atmosphere at the observed height is if it possesses a very low average density. This is a very general result from the conservation of energy and momentum, and seems very difficult to get around.

Chyba and colleagues' innovation is to include the effect of aerodynamic forces on the body. They show that these can fracture it and spread out the fragments, greatly increasing its overall atmospheric drag and so increasing the rate at which energy was deposited into the atmosphere. This process proceeds nearly catastrophically, as increasing drag increases the deceleration, which in turn spreads the fragments faster. In addition, the atmospheric density rises exponentially with decreasing altitude. These effects mean that bodies of the appropriate size will ultimately deposit most of their energy in a small region near the end of the trajectory (objects much smaller than 30 m in diameter either burn up or reach terminal velocity in the upper atmosphere, whereas the largest objects penetrate to the surface and form hypervelocity impact craters). In Chyba and colleagues' picture, the low average densities are achieved by fragmentation and dispersion of an object that was originally of normal density.

The idea that meteorites are fragmented by aerodynamic forces is not new. Apart from the bursts and flare-ups plainly visible in many bright fireballs (see figure), strewn fields of meteorites such as the 1947 Sikhote-Aline fall or the Henbury craters in Australia attest to atmospheric breakup of incoming meteorites. Quinn Passey and I examined this process in some detail 10 years ago[6,7], as did Grigorian and others in the former Soviet Union[8–11]. However, it was not until Zahnle studied the origin of the 'dark shadows' on Venus[12,13] that a fragmentation model was coupled to the equations governing meteor flight in the atmosphere. Chyba, Thomas and Zahnle apply this model to the Earth, computing the energy deposition from fragmenting meteorites and comparing the results to the Tunguska explosion. They examine the entry of meteorites starting with 15 megatons of kinetic energy and with strengths associated with the known meteorite classes — the strong irons, ordinary stony meteorites and fragile carbonaceous chondrites. Several estimates for comet strengths are also used. The iron meteorites hardly fragment at all and so strike the ground at high speed, as other

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Near miss: on 10 August 1972 this brilliant fireball made a 1,500-km track as a small asteroid dipped into the Earth's atmosphere over the western United States, passing just 58 km above Montana, at 15 km s^{-1}. Estimates of the object's diameter vary widely, from 4 to 80 m. If the larger estimates are right, and if it had approached at a steeper angle, it might have caused an atmospheric explosion comparable to Tunguska's. The photo above was taken at Grand Teton National Park in Wyoming.

irons in this size range have been observed to (Meteor Crater, Arizona, was made by an iron object with approximately this energy). The comets and carbonaceous chondrites deposit their energy too high. Only the ordinary stony meteorites create an explosion resembling that at Tunguska.

Substantial progress has thus been made in reducing the Tunguska explosion from the realm of the near-miraculous to a natural, although rare, occurrence. Instead of an implausibly low-density comet, the Tunguska projectile was more likely a representative of the most common class of meteorites. This picture leaves a few questions unanswered, however. One of the more notable phenomena associated with the Tunguska explosion were the widely reported 'light nights' observed over Eurasia for the first few nights after the explosion. Proponents of the cometary impact theory could point to the possibility of light reflected from parts of the comet tail that missed the Earth, but stony meteorites do not possess tails. Instead, Chyba and colleagues revive an earlier suggestion that the light nights were due to sunlight reflected from high noctilucent water-ice clouds. They estimate that the rise of the Tunguska 'fireball' of air heated by the explosion injected a large quantity of water into the upper atmosphere, which could have been transported over Europe by high-altitude winds.

There is still work to be done on the physics of Tunguska-like explosions. Chyba and colleagues' model of meteoroid fragmentation and dispersal is relatively crude. However, the physics of meteoroid fragmentation in the Earth's atmosphere is similar to that needed to study impact cratering on Venus, so there is reason to hope that further work on this problem will be forthcoming from both planetary and meteoritical scientists. □

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CELL BIOLOGY

Sticking to the point

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INFLAMMATION involves the cooperation of many different types of cells and the interaction of myriad different proteins. A possible role for one of these — the cytokine macrophage inflammatory factor (MIP-1β) — is provided by Tanaka *et al.* in a paper on page 79 of this issue[1]. They find that MIP-1β triggers one stage in the sequence of events that leads to lymphocytes adhering to the walls of capillaries before migrating out of the blood stream to sites of inflammation in tissues.

A consensus model emerged last year in which at least two different classes of adhesion molecules are required for leukocyte extravasation[2]. The first step involves members of the selectin family which slow the flow of leukocytes through postcapillary venules, promoting their transient adhesiveness to the endothelium. The second involves integrins, which convert the transient interaction to a strong adhesion. The 'adhesion triggers', which were thought to influence the activity of integrins, remained incompletely defined. MIP-1β is one of a number of cytokines involved in the initial stage of leukocyte migration, and Tanaka *et al.* now show that MIP-1β could be such a trigger. It acts on the $CD8^+$ subset of T lymphocytes — cytotoxic T cells — which respond by increasing the adhesiveness of their integrin VLA-4 for one of its ligands — VCAM-1. The problem of delivering this stimulus efficiently to cells that are still within the blood stream is apparently solved by tethering the cytokine to endothelial cell-surface proteoglycans — immunostaining of tissue sections has revealed the cytokine in that location. Thus, lymphocytes that slow sufficiently to interact with the immobilized cytokine will be encouraged to activate integrins, which in turn mediate firm adhesion by recognition of ligands on the vessel wall.

Extravasation of cells from the blood stream is essential for normal lymphocyte migration and inflammation, and similar mechanisms operate in the metastasis of some tumours. The importance in this process of members of both the integrin and selectin families of cell adhesion molecules (CAMs) has been known for some time. The two families of CAMs work in sequence[3]. Transient interactions of rapidly moving leukocytes with endothelium are mediated by one of three animal lectins (L-selectin, E-selectin or P-selectin) and their carbohydrate ligands. This favours multiple collisions between the two cell types and the typical 'rolling' behaviour of neutrophils, which precedes their flattening against the vessel wall. This latter step is clearly mediated by integrins.

The functions of some CAMs are regulated by changes in their levels of expression on the cell surface. Integrins, however, are constitutively expressed and their functions are regulated in other ways. 'Activation' states of cells expressing integrins and the extracellular divalent-cation concentration dramatically affect the specificity and avidity of integrins for their ligands. This active metabolic process, termed inside/out signalling, involves phosphorylation/dephosphorylation steps as well as interaction of cytoplasmic integrin tails with the cytoskeleton or other molecules[4].

The integrin VLA-4 (α_4, β_1) studied by Tanaka *et al.* is found on lymphocytes, monocytes, eosinophils and blood-cell precursors, and their experiments indicate how VLA-4 might be selectively activated on a particular T lymphocyte subpopulation.

There is growing awareness of the functional interdependence of proteoglycans, cytokines and CAMs[5,6]. Certain growth factors and cytokines, including members of the so-called 'intercrine' family to which MIP-1β has been assigned, have consensus sequences for binding to glycosaminoglycans, such as heparan sulphate on cell-surface proteoglycans. Cytokines thus immobilized can be sequestered, protected from degradation and presented to neighbouring cells. Heparan sulphate is also a critical component of the receptor for at least one cytokine fibroblast growth factor — and can be recognized as a ligand by at least one CAM (N-CAM). Tanaka *et al.* emphasize that some intercrines are localized on endothelial cells in postcapillary venules. These 'pro-inflammatory' cytokines are made in abundance by stimulated macrophages and other cells[7] and it is now clear that MIP-1β can be 'pro-adhesive' for at least one T-cell subset. A mechanism is provided for tethering it to the luminal surface of blood vessels, thus preventing its being washed away by the blood stream.

Tanaka *et al.* used heparan sulphated cell-surface antigen CD44 to coat plastic dishes and to immobilize MIP-1β for presentation to T lymphocytes. It is not clear that CD44, rather than some other transmembrane proteoglycan, bears the cytokine on endothelial cells. It was, however, an interesting choice of molecule for a number of reasons. CD44 is clearly involved in a variety of processes, and is widely distributed on endothelial cells and blood cells. It has recently been found to have many protein isoforms,