

Loophole for snowball Earth

Bruce Runnegar

The snowball Earth hypothesis posits an ice-covered planet. New climate simulations of 'snowball' conditions allow ice-free equatorial oceans that may be crucial for a theory about early animal evolution.

Snowball Earth is a script for global catastrophe that rivals giant-impact theories in the severity of its postulated environmental effects. In the 'hard' version of this hypothesis¹, low concentrations of atmospheric carbon dioxide (around 150 μ bar) and fainter sunlight (by some 6%) allowed polar ice caps to grow until reflected sunlight cooled the Earth and the oceans froze to a depth of about one kilometre. According to proponents, this happened at least twice during the Cryogenian period of the late Precambrian (850–590 million years ago) and perhaps more frequently a billion-and-a-half years before that^{2,3}. Recovery from these episodes depended on CO₂ emissions from volcanoes, and consequent greenhouse warming.

The effect on the biosphere of snowball events is thought to have been catastrophic. Carbonates immediately below and above the glacial deposits record carbon-isotope ratios characteristic of Earth's mantle^{1,4}, rather than of life processes, implying that oceanic photosynthesis was effectively eliminated during each snowball event. The result of this and anoxic conditions beneath the ice should have annihilated most kinds of eukaryotic life — that is, almost everything except bacteria. So successive snowball Earths should represent bottlenecks in the evolutionary history of eukaryotes through which comparatively few organisms passed. Conversely, the final disappearance of snowball conditions may have permitted and even stimulated the Cambrian explosion of complex multicellular life that began some 565 million years ago.

On page 425 of this issue, Hyde *et al.*⁵ challenge the 'hard' snowball Earth hypothesis. Their energy-balance and general-circulation climate models allow open water in equatorial regions to coexist with snowball Earth conditions elsewhere. According to their calculations, massive continental ice sheets up to 5 km thick flowed outwards into low latitudes. However, providing that atmospheric CO₂ was above a plausible level (roughly double the present amount), these flows did not create global oceanic ice shelves. Instead, they calved and melted to leave a narrow circum-equatorial refugium that might have included some access to coastal zones.

This 'softer' picture of a snowball Earth is similar to the original version proposed by

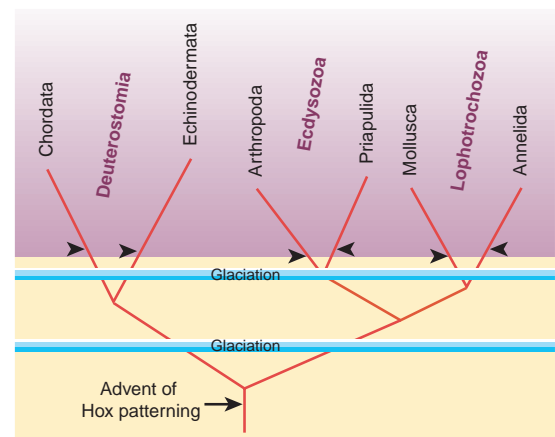
Kirschvink a decade ago⁶, and it allows for the evolution of the biosphere to proceed in a more orderly fashion than does the 'hard' alternative. But which of those views is likely to be correct?

A key question not addressed by Hyde *et al.* is whether enough CO₂ could accumulate in an atmosphere in contact with the global ocean to end a snowball Earth event. Hoffman *et al.*¹ assumed that more than a tenth of a bar of CO₂ would be required to overcome the increased reflectivity of an ice-covered Earth, so it is not a trivial matter.

Presumably, all continental silicates and shallow-water carbonates were protected from chemical weathering by very cold ice cover, so the usual controls on CO₂ build-up would not have applied⁷. If total alkalinity remained relatively constant, only a fraction of the carbon that needed to accumulate in the atmosphere could have been absorbed by the ocean. However, such an ocean would have been acidic, freezing, undersaturated in calcium carbonate, and inimical to the formation of mineral skeletons. This is one possible reason why the

Box 1 How to make an animal — indirectly

In a provocative series of articles, summarized in ref. 9, Peterson, Cameron and Davidson have proposed a new model for the origins of the disparate body plans of the various bilaterian metazoan phyla — primitively mobile animals that have an anterior–posterior axis, a central nervous system, and tissues and organs derived from a middle body layer. Molecular phylogenies segregate all bilaterian phyla into three large groups: deuterostomes, ecdysozoans and lophotrochozoans, shown in the figure here with some distinctive component phyla. The last two are sister groups and they jointly share the last common ancestor of all living bilaterians with the deuterostomes. That animal, argue Peterson *et al.*, had a free-living larval stage that bore little or no resemblance to the adult. The adult body was built from undifferentiated 'set-aside' cells that were free of growth constraints imposed on all other parts of the embryo. These cells could thus participate in a more sophisticated developmental process, involving Hox gene expression patterns, that is



characteristic of all bilaterian phyla.

The key point here is that similar bilaterian larvae are thought to have given rise to very different adult body plans. A classic case is the dissimilarity of adult molluscs and sipunculan worms and the great similarity of their larvae. But here lies the difficulty. Molluscs and sipunculans are lophotrochozoans, so their origins lie well above the common bilaterian ancestor in the metazoan tree. Somehow, deployment and patterning of pre-existing set-aside cells had to be delayed until the various lineages leading to the bilaterian phyla had been

separated for long periods of time. For example, distantly related members of the three bilaterian groups appear abruptly in the fossil record at the beginning of the Cambrian around 545 million years ago (arrows). However, molecular clocks¹⁰ suggest that the common ancestor of all three groups lived before the Cryogenian glaciations. Early-diverging bilaterians may therefore have been kept in 'larval mode' after the invention of set-aside cells and Hox cluster patterning by being forced to survive one or more snowball Earth glaciations in open-ocean environments. **B.R.**

Cambrian explosion might have been postponed (Box 1).

But there is another outcome of the work of Hyde *et al.*⁵. Let us accept, tentatively, that there were two global glaciations that occurred about 200 and 50 million years before the beginning of the Cambrian⁴. If so, each could have had one of three effects on the biosphere, in decreasing order of severity.

A snowball bottleneck. Conditions: global oceanic ice cover for around ten million years; anoxic oceans; almost complete loss of photosynthesizing biomass; extinction of all but a few lineages of eukaryotes, which survived mainly in geothermal oases on the continents. Probable consequences: a massive bottleneck in eukaryotic lineages; survivors that were predominantly terrestrial in origin.

A blue-water (pelagic) refugium, such as emerges from the paper of Hyde *et al.* Conditions: massive continental ice sheets and marginal ice shelves, but an open-water circum-tropical ocean with unglaciated volcanic islands; almost complete destruction of terrestrial biota and shallow-water, bottom-dwelling life; oxygenated oceans; prolific pelagic biomass in the nutrient-rich equatorial waters. Probable consequences: snowball episodes filtered eukaryotic lineages by allowing the survival of organisms that inhabited either the pelagic, open-ocean realm, or ocean-floor hydrothermal vents.

A uniformitarian outcome. Conditions: global refrigeration, but minimal impact on the biosphere because habitat loss was offset by positive effects such as the increased solubility of oxygen in sea water. Probable consequences: little or no evidence for snowball episodes in the evolutionary histories of post-Cryogenian marine eukaryotes.

The possibility that early metazoan (multicellular animal) lineages went through a pelagic filter may help to overcome a fundamental difficulty with one explanation for the origin of metazoan body plans^{8,9}. According to this view, described in more detail in Box 1, a unique innovation — the evolutionary origin of larval ‘set-aside’ cells — provided the canvas on which a wide range of animal body plans could ultimately be sketched. But to generate the main metazoan groups by this method it is necessary to separate lineage-splitting in time from body-plan specification. In other words, some environmental filter was required to maintain early metazoans in ‘larval mode’ after they had invented set-aside cells. This enabled them to diversify into well-separated lineages that ultimately became the independent sources of radically different body plans. A blue-water refugium could have served this purpose. If so, the Cambrian explosion might have begun with pelagic organisms discovering or rediscovering the sea floor. ■

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Cancer

New link in a web of human genes

Jean Y. J. Wang

Biologists have solved complex problems through genetics for more than a century. The simple approach of cataloguing genetic mutants often reveals interesting and important relationships between genes. By applying this principle, several groups^{1–4}, including two whose reports appear on pages 473 and 477 of this issue^{2,3}, have now established a functional link between *ATM* and *NBS1* — two genes involved in human diseases.

ATM is mutated in the disease ataxia

telangiectasia (AT), the symptoms of which include degeneration of a particular brain region (the cerebellum), immune dysfunction, sterility, sensitivity to radiation, and increased risk of cancer. *NBS1* is mutated in a rare disease called Nijmegen breakage syndrome (NBS). Patients with NBS have symptoms similar to those seen in AT. Although AT and NBS can be distinguished by clinical criteria, cells of AT and NBS patients show striking similarities, including the increased breakage of chromosomes.

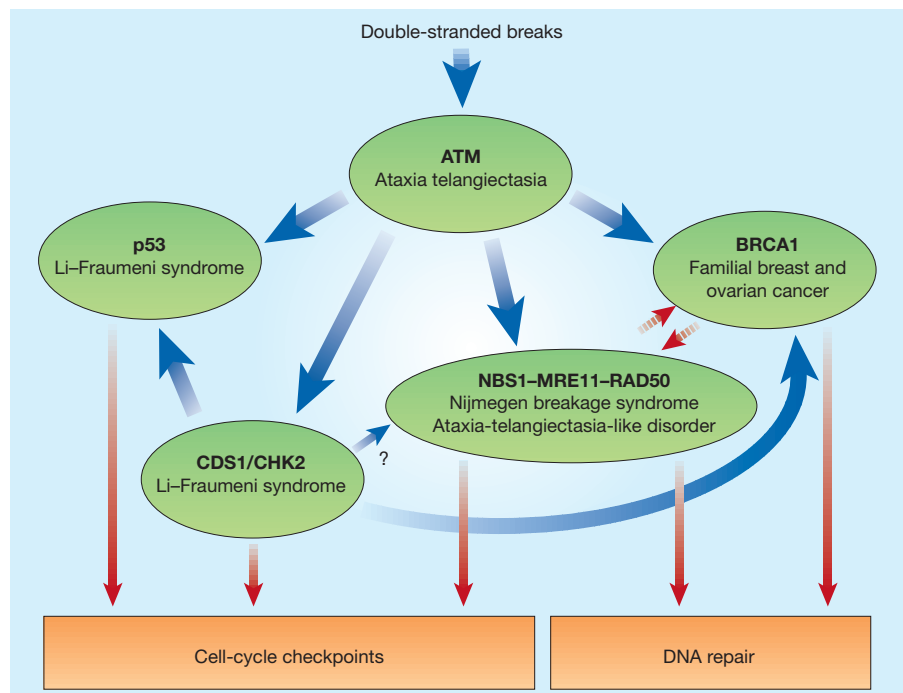


Figure 1 A network of proteins, implicated in human cancer, that regulate cellular responses to DNA damage. Double-stranded breaks (DSBs) in chromosomal DNA can activate a network of phosphorylation events to regulate DNA repair and progression through the cell-division cycle. Phosphorylation events are indicated by blue arrows. The protein kinase ATM is activated by DSBs to phosphorylate p53 (refs 1, 5, 6), CDS1/CHK2 (ref. 7), NBS1 (refs 1–4) and BRCA1 (ref. 9). (Mutations in the genes encoding these proteins are characteristic of the diseases indicated.) Further phosphorylation of p53 (refs 13–15) and BRCA1 (ref. 16) by CDS1/CHK2 might reinforce the actions of ATM. It will be interesting to know whether CDS1/CHK2 might also phosphorylate NBS1. The NBS1–MRE11–RAD50 complex also interacts with BRCA1, perhaps to coordinate DNA repair (dashed red arrows). Phosphorylation of p53, CDS1 and NBS1 activates cell-cycle checkpoints, which prevent the duplication and propagation of damaged DNA. Phosphorylation of NBS1 and BRCA1 may regulate the repair of DSBs. Together, the genes in this network protect the integrity of the genome, and mutations in the network predispose people to cancer.