NEWS & VIEWS

EARTH SCIENCES

Signature required

L. Paul Knauth

Most geologists agree that Earth's atmosphere was oxygen-free until 2.4 billion years ago. But the latest sulphur-isotope measurements from sedimentary rocks suggest otherwise.

L. PAUL KNAUTH

Newly embraced scientific paradigms usually have their share of lingering critics, quiet sceptics and bewildered agnostics. Nevertheless, few doubt the recently established consensus that Earth's atmosphere was completely oxygen-free before a great oxygenation event 2.4 billion years ago. But on page 908 of this issue[1], Ohmoto *et al.* report data on sulphur isotopes in sedimentary rocks that seem to contradict this theory.

The history of atmospheric oxygen is crucial for understanding the origin and history of life, the evolution of the atmosphere and the peculiar nature of early sediments. Geological evidence has always leaned towards an oxygen-free — anoxic — early atmosphere, but was never conclusive. Strong support for this theory finally arrived when a special kind of sulphur compound was found in sedimentary rocks. This compound has a particular ratio of isotopes that indicates ultraviolet exposure, and has only been found in rocks more than 2.4 billion years old[2]. This isotopic signature must have been created in an ancient environment that lacked an ozone layer. The absence of ozone would have allowed ultraviolet radiation to penetrate deep into the atmosphere, where it could interact with sulphur dioxide emitted by volcanoes, so forming new sulphur-containing products with distinctive isotopic ratios.

But this isotopic signature could only have survived and entered rocks if the reaction products had avoided recombination with oxygen. Such recombination occurs with even the tiniest amount of atmospheric oxygen, so the presence of this signature in old rocks is compelling testimony for an anoxic atmosphere before 2.4 billion years ago[3]. The isotopic data are robust, numerous and agree with the most common interpretations of the geological evidence, and apparently have no sensible alternative interpretations.

So far so good, but now imagine a lake 2.8 billion years ago, sitting beneath an anoxic, ultraviolet-drenched atmosphere. Sulphur dioxide emitted by distant volcanoes gets shattered in the atmosphere by ultraviolet light to form elemental sulphur and sulphuric acid, giving them a distinctive isotopic signature. These products mix into the lake water, where the sulphur settles along with small clay particles in quiet areas away from shore. As this sediment accumulates, iron reacts with the sulphur to make tiny crystals of pyrite (FeS_2), in a process well known to geochemists. Nowadays, streams that trickle into lakes contain sulphur from the Earth's crust in the form of sulphates, which are derived from the weathering of sulphur-containing minerals with oxygen. But this cannot happen in the anoxic atmosphere of our imaginary ancient world, so the pyrite begins its long burial mostly free of the contamination that could obscure its atmospheric isotope signature. Aeons later, Ohmoto and colleagues drill into the deposit and measure the concentrations of the different sulphur isotopes[1]. If our daydream is accurate, the ultraviolet-induced isotopic signature should be seen. But it isn't.

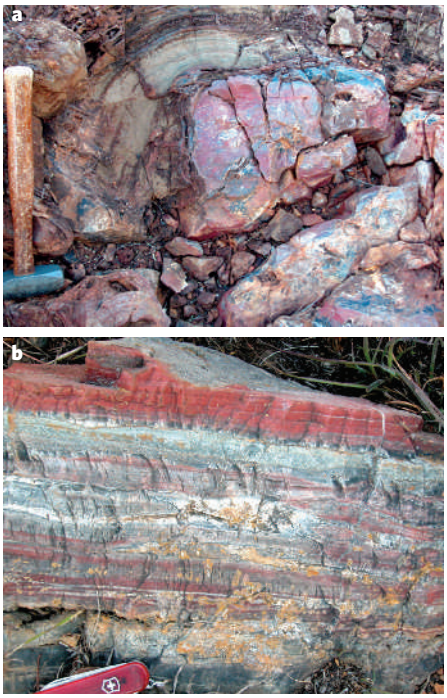

Figure 1 | Between a rock and a hard place. These oxidized sedimentary rocks formed at a time when the atmosphere is thought to have been oxygen-free. They may have arisen in special ways, such as at localized 'oxygen oases' where photosynthesizing organisms enriched waters with oxygen. But they could indicate problems, also raised by Ohmoto and colleagues' data[1], with the consensus that Earth's atmosphere was oxygen-free until 2.4 billion years ago. **a**, Strelley Pool chert at Pilbara Craton, Western Australia, is 3.43 billion years old, and is draped over older cherts holding oxidized iron. **b**, Sedimentary jasper beds in the Barberton Mountains, South Africa, are 3.227 billion years old.

So what now? Perhaps more data should be obtained from the commonly analysed ancient seabeds. Unfortunately, ocean sediments may not display an atmospheric sulphur-isotope signature if they are close to seafloor hydrothermal vents. These vents emit large amounts of sulphur that carries a crustal distribution of isotopes, swamping any contribution from the atmosphere. Lakes, however, should be free of this hydrothermal activity, and so their sediments should contain sulphur derived mainly from the atmosphere, providing a good test for early atmospheric oxygen content.

Although the expected atmospheric isotope signature is absent from Ohmoto and colleagues' data[1], these do not give quite the normal isotope signature for crustal sulphur either. Perhaps the atmospheric signature is there, but much smaller than was anticipated. If so, the oxygenation of Earth's atmosphere must have occurred less than 2.4 billion years ago, because similar values for the isotopic signature show up in younger sediments and are not considered to indicate 'sunburnt' sulphur. Also, the authors have yet to measure levels of the heaviest sulphur isotope, which provides a double-check on the signature if isotopic variations are small. Such analytical fussiness was used originally to define the global atmospheric-oxygenation paradigm, so true believers still have room to question the authors' negative results.

Could the pyrite crystals sampled by Ohmoto *et al.*[1] have washed into the lake from somewhere else? Possibly, but they have the form of crystals that grew in place. Maybe hydrothermal fluids bathed the sediments with solutions rich in crustal sulphur, so forming the pyrite? Perhaps, but tell-tale cross-cutting veins and hydrothermal minerals are absent, undermining this possibility. The atmosphere present when the lake sediments formed remains the most likely source of sulphur for the pyrite.

Earth scientists may welcome one of the

authors' possible explanations of their findings — a 'yo-yo' atmosphere, in which oxygen levels rose and fell before the great oxygenation event. Beautiful Archean jaspers, rose-coloured cherts (Fig. 1) and other sediments older than 2.4 billion years that contain oxidized iron require special explanation if they formed in anoxic waters. Ultraviolet oxidation of surface waters offers one possibility, but this doesn't fit the geochemical evidence. Yet another theory involves isolated 'oxygen oases', where early photosynthesizers briefly enriched isolated pools with oxygen. But this conflicts with the widespread distribution of oxidized deposits. Geologists have long asked why periods of oxygenation could not have occurred in fits and starts, instead of in a great, single event. Atmospheric modellers are reticent on this[4], but it would certainly help to explain the geology.

Finally, could the great oxygenation paradigm be wrong because there are other, unknown ways to make the ultraviolet-induced sulphur-isotope signature? For example, heating a mixture of sulphate and amino acids apparently produces a weaker version of the 'ultraviolet' signature, without using ultraviolet light[5]. Suggestions for alternative mechanisms usually induce howls of disbelief, but a similar prejudice was challenged after the discovery of oxygen-isotope anomalies in meteorites[6]; the oxygen-isotopic signature was subsequently created experimentally in two completely unexpected ways[7,8]. However, there is no known mechanism that excludes ultraviolet and that can still produce the large sulphur-isotope signature observed in old rocks.

Like much of the geological evidence, Ohmoto and colleagues' measurements[1] are not easily reconciled with the global oxygenation paradigm. Bewildered agnostics can rejoice. These data cannot be ignored, and the field has just become even more interesting. ■

L. Paul Knauth is in the School of Earth and Space Exploration, Arizona State University, Tempe, Arizona 85287-1404, USA.
e-mail: knauth@asu.edu

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CELL BIOLOGY

A licence for duplication

Erich A. Nigg

Genome stability in animal cells requires strict control over the numbers of the organelles called centrosomes. An attractive 'licensing' model now explains how centrosome duplication is restricted to just once per cell cycle.

The centrosome is one of the most intriguing organelles found in animal cells. It organizes the construction and maintenance of the microtubule cytoskeleton, a molecular 'scaffold' that confers shape and polarity on the cell[1]. Most strikingly, the centrosome duplicates during the cell-division cycle[2], and the duplicated centrosomes then help to form the spindle apparatus that segregates the duplicated chromosomes into the daughter cells. Because aberrant centrosome numbers can cause chromosome mis-segregation, such abnormalities have been proposed to contribute to the development of cancer[3,4] — cancer cells often have abnormal chromosome numbers. The way in which centrosome numbers are controlled has been shrouded in mystery, but in this issue Tsou and Stearns (page 947)[5] provide evidence supporting a simple control mechanism.

Every centrosome comprises two centrioles — barrel-shaped structures made of nine microtubule triplets — embedded in a complex protein matrix[1]. So, from a mechanistic perspective, the problem of centrosome duplication essentially comes down to the question of how centrioles are duplicated. At the morphological level, the centrosome/centriole duplication cycle is well understood[2]. One notable aspect of the centrosome cycle is that parent and progeny centrioles show a tight right-angled (orthogonal) association (Fig. 1). This 'engagement'[5] is established during duplication and persists through the subsequent phases of cell-cycle progression until 'disengagement' late in M phase and/or early G1 phase of the cycle separates the progeny centriole from its parent. So, during G1 phase, the two (future parent) centrioles are clearly separated, albeit loosely tethered to one another, and centriole disengagement has long been thought to be a prerequisite for duplication. However, the full significance of this peculiar process in the cell cycle is just beginning to emerge.

Three years ago, Wong and Stearns[6] provided evidence that there is a block intrinsic to centrosomes that prevents the centrosome from duplicating again during late S and G2 phases — thus, it can duplicate only once per cell-division cycle. This implies that passage through M and/or G1 phases might be required to create permissive conditions — or 'issue a licence' — for a new round of centriole duplication in the next S phase.

Tsou and Stearns[5] now use an *in vitro* assay based on frog (*Xenopus*) egg extracts and purified centrosomes to monitor simultaneously centriole disengagement and the growth of new centrioles under different experimental conditions. Their results strongly suggest that centriole disengagement requires the activity of separase, a protein-digesting enzyme previously shown to have a leading role in triggering the separation of duplicated chromosomes[7]. Furthermore, their data show that centriole disengagement is a prerequisite for the subsequent growth of new centrioles. This work provides an appealing model for how centriole duplication is normally limited to occurring only once in every cell cycle. In essence, centriole engagement (established during centriole duplication in S phase) prevents further centriole duplication until the activation of separase during M phase triggers centriole disengagement. Disengagement licenses the centrioles to undergo a new round of duplication (Fig. 1). This mechanism for limiting centrosome duplication to once per cycle is reminiscent of the controls that prevent multiple rounds of DNA replication.

Even landmark studies do not answer all the questions — on the contrary, they generally raise interesting new ones — and the work by Tsou and Stearns[5] is no exception. Although the data strongly implicate separase in centriole disengagement, the evidence is mostly indirect. Definitive proof will have to await a direct and positive demonstration of the action of this enzyme. If separase does induce centriole disengagement, then how does it act? The simplest mechanism would be that it cuts one or more of the centriole-associated proteins that glue centrioles together (Fig. 1). Alternatively, it could act indirectly, for example by regulating the activity of another enzyme (a kinase or phosphatase, perhaps) that in turn triggers centriole disengagement.

These findings may have implications for the mechanisms that underlie the increased centrosome numbers typical of human cancer cells[4]. Deregulation or premature activation of separase might cause multi-polar spindle formation or create conditions allowing multiple rounds of centriole duplication in a single cell cycle. Alternatively, centriole numbers might be increased through loss of other controls yet to be discovered. Although the licensing model neatly explains how centriole duplication is limited to once per cell cycle, it does not explain how cells ensure that only one centriole is built next to each parental centriole. In the case of DNA replication, it is clear how each strand of the double helix forms a template for the synthesis of exactly one complementary strand, but how the growth of progeny centrioles is controlled is unknown. What prevents the simultaneous growth of two or more progeny in each round of duplication, for instance? And how can a cylinder form a template to grow a new centriole at a right angle to itself?

Centriole duplication is known to require