

The Permo-Triassic crisis – A new cause for a well-documented effect

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Radiometric dating of geologic events recorded in geologic materials (e.g. minerals, organics) is our universal reference frame for earth history. The long-arriving and long-teetering Permo-Triassic biotic catastrophe culminated at ~252 Ma, but the crime wave leading to extinction of nearly all life forms remains unsolved. What was the cause?

We propose increasing *felsic* magmatism and the accompanying release of unprecedented volumes of toxic metal-rich volatiles and sulphur as a cause. Critically, our evidence is pinned in time by detailed Re-Os dating of molybdenite generations from several large and well-studied felsic magmatic provinces hosting Mo-Sn-W mineralization. A hallmark of many of these provinces is low to drastically low Re in the molybdenites. The flip-flop in oxidation state of these ore systems, the dearth of pyrite, the paucity of sulphur, and clear geologic evidence that sulphide-stable events in felsic magma chambers were universally arrested, teetering, and only locally revitalized, provide tantalizing clues.

In the Oslo Rift, we have undertaken reconnaissance re-logging of 1980s exploration drill holes for Mo deposits. The oldest dated magmatism (300 Ma) is derived from alkaline basalts at the southernmost exposed end of the rift [1]. Our Re-Os geochronology for granitoid-hosted mineralization shows systematic unzipping of the rift from south to north (~280-250 Ma), and a remarkable record of increasing oxidation with time captured in vein parageneses. We suggest the real entrée to the Permo-Triassic transition was in the making for tens of millions of years, and driven by increasing heating and oxidation of the crust. The oxidation state, vein sequencing, and lithologic cover required to create large economic Mo deposits were never achieved; rather, the atmosphere and biosphere became the repository for unprecedented volumes of toxic metals. The effect may have been cumulative, and with each successive breach, recovery was compromised. As an example, the Re/Os ratios in organic material from uppermost Permian shales on the Norwegian shelf and East Greenland are extraordinarily high [2]. Profound changes in mantle chemistry that gained momentum over perhaps 50 Ma may have been behind the crime wave.

[1] Corfu & Dahlgren (2008) *EPSL* **265**, 256–269.

[2] Georgiev *et al.* *GCA*, this volume.

Characterization of outer membrane proteins involved in iron reduction and biofilm formation in *Geobacter sulfurreducens*

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Geobacter sulfurreducens is a ubiquitous iron reducing species found in subsurface environments. In these environments, iron exists predominantly in the oxidized form as iron oxides. The mechanism of electron transfer to metal oxides has been proposed to occur through: i) direct contact with the oxide, ii) production of electron shuttles and iii) production of metal chelators which solubilizes the metal allowing interaction with the cell. Researchers have provided evidence to support all three possible mechanisms. However, there is a lack of evidence for indirect iron oxide reduction with *Geobacter* and as a result, metal oxide reduction by direct contact has been proposed for this microbe. Biofilm formation allows direct contact with insoluble terminal electron acceptors. Our research will focus on differential protein expression in multi-layer *Geobacter* biofilms.

Based on the known roles of outer membrane c-type cytochrome (Omc) proteins OmcB, OmcE and OmcS in iron reduction and of pili proteins PilA and PilT in biofilm formation, we will examine their expression and localization in *Geobacter* biofilms. Preliminary results show that OmcB may be differentially expressed in the biofilm. In addition to understanding the expression of these cytochrome and pili proteins in *Geobacter* biofilms, we are also interested in determining whether any of the outer membrane cytochrome proteins are present in protein complexes. Preliminary results show that there are at least seven heme proteins present in complexes as determined by Blue Native-PAGE and chemiluminescent heme staining.