Four Cycles of Oxygenation in the Phanerozoic

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Sedimentary pyrite captures trace elements (TE) from the oceans and tracks variations in their seawater concentrations through time [1]. LA-ICPMS analysis of sedimentary pyrites, based on newly developed standards, has enabled the development of temporal ocean concentration curves for 22 TE [2].

Our results show that TE variations over the last 700 million years of ocean history have been strongly cyclical. We interpret these cycles to indicate that the Late Neoproterozoic to Phanerozoic oceans went through dramatic changes in mean oxygen content. Four major cycles are recognised: Late Cryogenian to Late Ordovician, Early Silurian to late Devonian, Early Carboniferous to Late Permian and Triassic to Quaternary. Oxygen maxima, indicated by Se, U and Mo proxies, occur at 540, 390, 310 and 0 Ma, supporting previous models [3, 4]. Oxygen minima, indicated by trace element drawdown, occur at 700, 455, 365 and 200 Ma. Extended periods of low oxygen in the oceans have led to extreme deficiency of some elements that are critical for life. The periods of extreme Se depletion coincide with the mass extinction events at end Ordovician, Late Devonian and the Triassic-Jurassic boundary, suggesting that Se-deficiency in the oceans may be a contributing cause of marine mass extinctions.

[1] Halpin *et al*.. (2013) *Min. Mag*., this volume. [2] Danyushevsky *et al*.. (2013) *Min. Mag*., this volume. [3] Berner (2006) *Am. J. Sci*. **309,** 603-606. [4] Dahl *et al*.., (2010) *PNAS* **107**, 17911-17915.

High precision isotope measurements unveil poor control of copper metabolism in Parkinson's disease

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Parkinsonism is a neurodegenerative disorder, and propagates from the loss of dopaminergic cells due to oxidative stress. Disordered copper metabolism may be important in the causation of Parkinsonism, as the main Cu carrying enzyme, caeruloplasmin, is key in handling oxidative stress and is involved in the synthesis pathway of dopamine [1, 2].

The human Cu metabolism of ten Parkinsonism patients was compared to ten healthy controls with the aid of a stable ^{65}Cu isotope tracer. The analyses of blood serum $^{65}Cu/^{63}Cu$ ratios yielded individual isotopic profiles spanning four days. The use of multiple-collector inductively coupled plasma mass spectrometry and the associated sample preparation techniques [3] is necessary to detect the small differences in Cu metabolism between subjects with Parkinsonism and controls.

The isotopic profiles indicate that the Cu metabolism is less controlled in patients with Parkinsonism. In addition, modelling suggests that (i) 30% of the subjects affected by Parkinsonism have abnormally large Cu stores in tissues and (ii) the absorption of Cu in the gut may control subsequent Cu metabolism. This pilot investigation supports full-scale medical studies into the Cu metabolism of those with Parkinsonism [4].

[1] Vassiliev *et al*.. (2005) *Brain Res. Rev*. **49**, 633-640. [2] Jenner (2003) *Ann. Neurol*. **53**, S26-36. [3] Larner *et al*.. (2011) *J. Anal. Atom. Spectrom*. **26**, 1627-1632. [4] Larner *et al*.. (2013) *Metallomics* **5**, 125-132.

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