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Towards a palaeosalinity proxy: hydrogen isotopic fractionation between source water and lipids produced via different biosynthetic pathways in haptophyte algae

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Palaeosalinity is one of the most important oceanographic parameters that cannot currently be quantified with reasonable accuracy from sedimentary records. Hydrogen isotopic fractionation between water and alkenones is dependent, amongst other factors, upon the salinity in which alkenone-producing haptophyte algae grow and is represented by the fractionation factor, α, increasing with salinity.[1] As such, the hydrogen isotopic composition of alkenones is emerging as a palaeosalinity proxy. Understanding the mechanism behind the sensitivity of fractionation to salinity is important for the correct application of the proxy, however this mechanism is currently unknown.

Here we present hydrogen isotopic compositions of lipids produced via different biosynthetic pathways from batch cultures of *Emiliania huxleyi* CCMP 1516 and *Isochrysis galbana* CCMP 1323 grown over a range of salinities and discuss the possible sources of the sensitivity of hydrogen isotope fractionation to salinity. α for C_{37} alkenones (produced via an unknown biosynthetic pathway but assumed to be acetogenic; e.g.[2]) and that for $C_{14:0}$, $C_{16:0}$, and $C_{18:1}$ fatty acids (acetogenic) from exponential growth phase *I. galbana* show a similar sensitivity to salinity, increasing at 0.0013-0.0019 per salinity unit (S^{-1}). Meanwhile, in exponential growth phase *E. huxleyi*, α for C_{37} alkenones and α for brassicasterol (mevalonate pathway) increase at 0.0015-0.0022 S^{-1}, but α for phytol (methylerythritol pathway) shows no significant relationship with salinity. These results suggest that fractionation is sensitive to salinity for lipids formed both in the chloroplast and cytosol. They also suggest that the sensitivity may either originate in glyceralde-3-phosphate or pyruvate but is then lost through hydrogen exchange with cell water during sugar rearrangements in the methylerythritol pathway or sensitivity originates with the production and consumption of acetate.

References

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