The use of n-alkanes $\delta^{13}C$ to evaluate the trigger(s) of the Cretaceous Oceanic Anoxic Event 1a

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Oceanic anoxic events (OAEs) are time envelopes in the Cretaceous when ocean conditions favoured the episodic deposition of organic-rich sediments on a global scale. The first Cretaceous OAE (1a, ~120 million years ago) is marked by an enigmatic negative carbon isotope (d13C) spike of up to 3‰ in marine carbonates and of 4 to 5‰ in the organic matter (OM) at its base. This carbon-cycle perturbation is believed to reflect a fast and massive release of 13C-depleted carbon into the ocean and the atmosphere. The trigger(s) of OAEs remain however poorly known because exchanges between ocean and atmosphere pools are faster than the resolution of the C-isotope records available so far and because bulk d13C combines effects of a whole ecosystem. Within the entire range of lipid biomarkers preserved in the sedimentary OM, those showing fairly well constrained precursors provide the highest potential for accurate paleoenvironmental reconstruction using compound specific d13C. In this study, a high resolution C-isotope record for the carbonates, the bulk OM and specific alkanes has been performed at the base of OAE1a of the Cismon core and the Pusiano section (Southern Alps, Italy). Linear alkanes, derived from their unsaturated relatives, are ubiquitous biosynthetic compounds. Their origin in the samples of Cismon and Pusiano, particularly for the extra-long chain n-alkanes (n-C33, n-C35, n-C37), is discussed based on distribution characteristics, compound specific d13C and nannofossil data. Additional lipid biomarkers d13C are also presented. These high resolution bulk and compound specific d13C records show that an increase in C-isotope fractionation, most probably triggered by elevated CO2 emissions from the Ontong-Java Plateau Large Igneous Province, mostly caused the OAE1a negative d13C spike. A release of light gas-hydrate derived carbon into the ocean and the atmosphere likely played only a minor role at the onset of OAE1a.