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Salinity-induced stratification and the onset of hypoxia during the Holocene Thermal Maximum and the Medieval Climate Anomaly

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During the past ~8000 years the Baltic Sea has experienced three distinct intervals of hypoxia, of which the last one is still ongoing. These intervals are characterized by enhanced sedimentary organic matter burial and enrichment of redox-sensitive metals, such as molybdenum and iron. The first two of these intervals occurred during the Holocene Thermal Maximum (HTM) and the Medieval Climate Anomaly (MCA), two phases with high temperatures and changed precipitation patterns. Studies focussing on the Holocene sedimentary record of the Baltic Sea aim at clarifying the causes of the initiation, evolution and termination of these hypoxic intervals, as well as their consequences. This information could help to potentially aid in finding solutions for the mitigation of present-day hypoxia in the Baltic Sea. The factors contributing to hypoxia development during the HTM and MCA are still debated.

Here we present data from a core retrieved during Integrated Ocean Drilling Program (IODP) Expedition 347 in the Landsort Deep basin, the deepest basin of the Baltic Sea at 463m water depth. Sediments were analysed at a high resolution using inorganic geochemical and (mainly marine) palynological proxies. Dinoflagellate cyst (dinocyst) assemblages and total elemental compositions provide clues on the role of salinity in enhancing stratification, ultimately causing hypoxia. During the onset of the HTM changes in salinity, as indicated by the palynology, closely follow changes in sedimentary organic carbon burial and trace metal concentrations. This suggests that stratification was an important cause of hypoxia during the HTM. In contrast, the palynology suggests that reduced stratification did not contribute to re-oxygenation during the termination of the HTM. We did not observe major changes in the palynology throughout the hypoxic interval of the MCA. Our results thus suggest that changes in salinity did not cause the onset and termination of hypoxia during the MCA.