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Toward a quantitative reconstruction of hypoxia from varve records in the large perialpin Lake Bourget over the last 150 years

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Hypoxia -defined as dissolved oxygen $\leq$2 mg/l- is a severe detrimental factor for aquatic environments. In lakes, despit the importance for management, it is generally still hard to estimate hypoxia because of the lack of appropriate proxies or restricted number of sample cores. In this study, by using (40) sediment core data from chosen depths, we propose to go a step further toward a quantitative reconstruction of hypoxia integrating the extension of hypoxic water layer, both through space (volume) and time (yearly value). For that we went a step further in using an existing proxy: varve preservation. It is generally well-adapted for hypoxia detection, but not yet developed for small scale time and space variations through a complete large lake basin. Varves preservation is the consequence of the death of most of benthic macro-organisms that normally mix-up first millimetres of sediments, due to oxygen depletion. In Lake Bourget recent laminated sediments correspond to biochemical varves. We assume that their preservation results from a threshold in dissolved oxygen concentrations induced by seasonal hypoxia. Chironomids, organic matter and Mn/Fe ratio (XRF) were used as complementary proxies of hypoxia to validate our assumptions concerning varves.

Our results show that volume of hypoxia can be annually estimated according to varve records through lake. Volumes of hypoxia varied through time in the Lake Bourget. Sediments recorded first the onset of severe hypoxia in the deepest part of the basin (-140m) in AD 1935±1, corresponding to 11.103m3 of hypoxic waters. Then hypoxic surface progressively extended on the slope until reaching a maximum at -90m in AD 1960, leading to 306.103m3 of hypoxic waters. After a retreat dated to AD 1980, hypoxia seemed to re-extend until today. Those fluctuations over the "oscillating zone" of hypoxia (-90 to -133m) were compared with potential forcing factors.

The onset of hypolimnetic hypoxia is commonly attributed to eutrophication depending on the inflow of nutrient-rich water (sewage water and/or diffuse contamination). However we argue that two other forcing factors could have strengthened the eutrophication effect on hypoxia. Floods are known to be a source of reoxygenation via underflows that could explain the oxygen retreat in 1980 despite the maximum of eutrophication. It can also explain the increase of hypoxia in 1983 after dam settlement which regulated water inflows. Rise of temperatures since 1970 has increased time-length of stratification by positive feedback on primary productivity that could explain the last increase of hypoxia in spite of human efforts for lake-water quality restoration. Whereas flood activity and global warming seem to be both strongly implicated in the recent fluctuations in hypolimnetic hypoxia it is not yet clear how much each of those parameters act on it.