Great Lakes Lake Trout Early Mortality Syndrome (EMS): Contaminants, Thiamin Status, and Their Possible Interaction

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Salmonid populations in the Great Lakes experienced a decline in the early twentieth century, presumably due to over-fishing combined with the introduction of exotic parasites such as the sea lamprey. Despite intensive rehabilitation and stocking programs, today significant natural reproduction exists only in Lake Superior. Dioxin-like contaminants (i.e., PHHs) are known to cause adverse effects in early life stage lake trout, and results indicate that even the low levels currently present in Lake Michigan can result in sublethal physical lesions or behavioral alterations such as diminished C-start response. 2,3,7,8-TCDD caused significant adverse effects of both C-start behavior and feeding in rainbow trout and lake trout young. In addition to the presence of contaminants, a nutritional thiamin deficiency has been shown to cause high mortality, termed Early Mortality Syndrome (EMS), in Great Lakes swim-up fry. In the current study, fry eventually succumbing to EMS exhibited reduced embryo C-start behavior. It appears that neither the presence of PHHs nor EMS mortality can fully account for the total lack of lake trout recruitment in the lower Great Lakes. However, it is possible that an interaction between the two stressors can result in greater than expected effects on fry health and survival.