1. Direct modeling of O2 flows
   1. I am not sure if this would be useful because MO2 only changed below 2.7 mg/L (on average) so this would do nothing to explain effects on hatch timing, growth, and survival at 2.7 mg/L and above. There is also a difference in that the metabolic measurements applied acute hypoxia and the other experiments applied chronic hypoxia.
   2. The shift to O2 limited metabolism would not explain the effects because it happens at a lower oxygen level.
2. Use of a damage variable
   1. Gene expression to indicate which DEB fluxes could be affected
   2. Direct biochemical information such as lactate production. Either add an anaerobic pathway explicitly into the model or treat the anaerobic products as a measure of damage.
   3. Metabolomics. “Conventional wisdom among DEB workers is that data on intermediate metabolites (e.g. ATP) has little value because of their high turnover rate compared with the processes on which DEB models focus.” But Roger has been doing work with Louise Stevenson and Phil Antzsac where Phil identified important pathways through the metabolome.
3. Bringing in CO2?
   1. No, only as a discussion point because the data are too inconsistent to try and fit anything unless we just did a simulation. But also I want to be done with this and not add anything else on.

Goals

* Look for evidence for the three damage proxies.
* Understand how damage is quantitatively modeled and applied to a parameter.
* Once damage proxies or mechanisms are identified, match them up to one or more DEB parameter they would affect.