Progress Report for Chapter 4

November 2022

Goals

* Model how hypoxia affects energy budget to understand potential mechanisms behind the data better.
  + Predict hatch time (time at egg buffer=0) and hatch length under different oxygen levels to match the data, by applying an oxygen-based stress function to one or more parameters.
* Learn how life history is affected by hypoxia
  + Simulate reproduction and survival for population implications/effects in different scenarios.

Metabolism-Oxygen Relationship

* Mean Pcrit values in control conditions (ambient CO2):
  + Embryo: 2.044(±0.209) mg/L, n=15
  + 2dph larvae: 1.653(±0.154) mg/L, n=19
  + 5dph larvae: 1.561(±0.183) mg/L, n=16
  + High CO2 can decrease Pcrit of larvae, making them less sensitive to hypoxia.
* Hatching success:
* Hatch time:
* Hatch length:
* Larval length:
* Larval survival:
* I guess these would ideally be in partial pressure units
* Oxyconformity in many embryos, maybe embryos have a closer to linear relationship between oxygen and stress. Can/should we make the curve different for before and after hatching?
* Larvae are more likely than embryos to have transient sharp increase in MO2 below Pcrit, right before MO2 reaches zero.
  + This increase in oxygen consumption has been attributed to lactate and other products of anaerobiosis having a stimulatory effect (Pörtner and Grieshaber, 1993). Embryos can’t move much compared to larvae.
  + The embryo stress function could be more gradual and over a longer range of O2, and start to increase at a higher O2 than larvae. The function for larvae would start increasing at lower O2 and sharply increase about halfway to zero.
  + Could use an asymmetrical logistic function to encompass both OR could use two different stress functions, each for a different parameter.
    - Being below Pcrit would affect yVA or sJAm assuming not able to use aerobic metabolism for all conversions, they would be less efficient.
    - Being at and below the spike would affect sJM because the lactate buildup is increasing activity and potentially causing damage that requires repair.
* Acute vs chronic hypoxia – which do I want to focus on, or both?

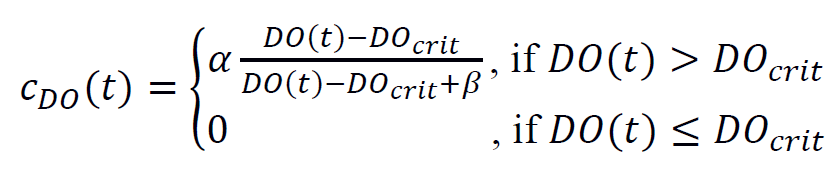
DEBkiss model with survival

* Most influential parameters (improved fit most based on AIC change) are fB (scaled food level), yVA (yield of structure on assimilates), kappa, Lwp (length at puberty), sJAm (max assimilation rate), f, and sJM (maintenance rate).
* Adding a second and third parameter improved fit, and best combo of three (excluding the ones that don’t make sense to include) was sJAm, sJM, and kappa.
  + But the AIC was very close to that of sJAm, yVA, and kappa.
* Is it normal to have two inflection points in survival and egg buffer? It looks like the egg buffer isn’t getting used up fast enough with these parameters maybe. Maintenance is too low? Or assimilation?

Questions for Roger

* Do you invite people whose published data you use for a model to be coauthors, or simply cite them?
* Should I use all survival data or an average for each age?
* Is the modeling I just did validation of the parameters? What is the difference between fitting and validating?

Notes on other studies

* How did Lavaud et al. (2019) model hypoxia?
  + Their fourth additional assumption: Food ingestion was impacted by DO level. This is based on Thomas et al. (2019), saying that energy uptake decreases under hypoxia and this drives growth and reproduction.
  + Used a correction factor applied to the ingestion rate (similar to stress function).
  + This was justified with previous results showing that Atlantic cod had reduced growth under hypoxia (Chabot and Dutil, 1999).
  + Correction factor: 
  + It is a Michaelis-Menten saturation function where DO(t) is the oxygen at time t, and α and β are constants for which the correction factor is 1 at 100% oxygen. Below DOcrit ingestion is zero.
  + They estimated the parameters by fitting the correction function on an experimental dataset of ingestion rates at various levels of DO (Chabot and Dutil, 1999). **What data can I use to fit a correction function?** 
    - They used nls function in R to do this.
    - Could I try this with two different equations and use metabolism to fit the stress curve?
    - The type of data also depends on which parameter(s) the correction factor would be applied to (instead of ingestion).
  + They ran simulations over time using environmental data for Gulf of St. Lawrence for two cod populations.
    - I could do DO scenario(s) of realistic fluctuations and compare to a few constant DO levels.
    - DO doesn’t get very low in a lot of monitoring data (Flax doesn’t go below 2 mg/L) but that doesn’t mean there isn’t patchy hypoxia in very shallow water.
    - Reference simulation didn’t have DO effect because they assumed the stomach contents already reflected the DO experienced in the wild.
    - Scenario 2 added temperature increase to test assumption of how temperature alone would affect energy uptake.
    - Scenario 3 removed effect of DO on energy uptake by multiplying f by 1/cDO and measuring resulting gain in growth in both populations (using temps from reference scenario).
    - Used RSME to measure goodness of simulation model fit to data (S1). Calculated differences in length and mass due to temperature by comparing S1 and S2. Calculated hypoxia effects using mean differences between predictions for S1 and S3.
  + They also used simulations to see how sensitive life history traits are to duration, frequency, and intensity of hypoxia (compared to the reference scenario).
    - 50, 100, and 200 days of summer hypoxia duration (winter left at reference level).
    - 30, 50, 70, and 100% DO saturation intensities
    - They compared asymptotic length reached after 15 years of simulation, age at 40cm (recruitment), reproductive investment (cumulative egg production in 15 years), age at maturity (time to EpH)
* How did Aguirre-Velarde model hypoxia effects?
* How did Pousse et al. (2022) model CO2 effects?
  + In Intro they said OA needs to be added to DEB models as a forcing variable.
  + They cite others who implemented OA effects by calibrating parameters to a static CO2 level, via maintenance costs, assimilation efficiency, ingestion rate, and cost for structural growth. But static is different than using it as a forcing variable, which allows for temporal variation in realistic projections of effects (Signorini et al., 2013). **Does oxygen work this way in DEBkiss?**
  + General method:
    - Calibrate model parameters to physiological and growth data measured under lab conditions (this is what I’m doing now I think).
    - Scaling pCO2 effects as a third forcing variable.
    - Validate the model under contemporary conditions using field growth data.
      * They used 35 years of survey data to compare shell lengths in field to the ones predicted by the DEB model when forced with the forcing variables from the same time period.
      * Would I be doing this by testing the hypoxia model’s predictions against lab data at dif oxygen levels?
    - Project future growth and reproduction in MAB and GB using RCP scenarios.
  + Previous results showed decreased feeding rate and increased metabolic loss (respiration and excretion) under OA.
  + They used a feeding depression factor based on % changes in feeding at two elevated pCO2 levels relative to ambient. This one scales changes in feeding linearly with pCO2. Between the boundaries of no effect and no feeding at all, they use this equation for feeding correction factor: 
  + Metabolism increases with pCO2 but above a threshold the metabolic pathway can switch to a more energy-efficient strategy of balancing intracellular pH (cite their own surf clam work and a blue mussel paper: Thomsen and Melzner, 2020).
  + Metabolism costs associated with pCO2 (pMC) calculation: if pCO2 is lower than the minimum threshold for metabolism costs then it is zero. If it is higher than the minimum threshold but below the maximum threshold then the amount of CO2 above the threshold is multiplied by a coefficient then by the Arrhenius temperature factor and the structural volume raised to 2/3. If it is above the maximum CO2 threshold then the max threshold minus min threshold is the amount of CO2 multiplied by all this.
* How did Jager explain the implementation of the stress function in the DEBkiss book?