**Highlights**

* The bioenergetic mechanisms responsible for previously observed hypoxia effects on young Atlantic silversides were investigated using a simplified Dynamic Energy Budget model.
* We connected physiological information with energetic processes to identify assimilation, conversion efficiency of assimilates to growth, maintenance rate, and mortality as potential mechanisms.
* Changing conversion efficiency, embryonic mortality, and larval mortality with hypoxia gave the best fit to data on early life hatching, growth, and survival.
* We used widely applicable biological theory to gain a better understanding of hypoxia effects on a model species, finding a mechanism that would impact the efficiency of energy flow across generations and ultimately through the food web.

**Abstract**

Ocean deoxygenation is intensifying worldwide due to warming and eutrophication, particularly in estuaries and coastal waters. Although the Atlantic silverside (*Menidia menidia*) has proven robust to the fluctuating environmental conditions in its estuarine environment, chronic hypoxia impairs hatching, growth, and survival in the early life stages. We used a simplified version of a Dynamic Energy Budget model (DEBkiss) to test the hypothesis that experimentally observed changes in animal performance can be explained by one or more of the rate processes in the model. We sought to identify the DEBkiss parameters that, when adjusted with a correction factor based on inhibition of Synthesizing Units, provided the best fit to hypoxia effects in the three state variables of total length, egg buffer mass, and survival over time. Because hypoxia reduces survival in embryos and newly hatched larvae, we added a survival state variable controlled by pre- and post-hatching mortality parameters. Applying the hypoxia effects to reduce the conversion efficiency of assimilates to structure accounted for some of the hypoxia-related changes in all three state variables. However, the best fit was achieved by simultaneously reducing the conversion efficiency and increasing both mortality parameters. In contrast, changing the parameter for maintenance rate with hypoxia provided little to no improvement of fit to the data. Reduced conversion efficiency under hypoxia would suggest that less of the energy invested by parents and consumed through predation is converted into biomass in *M. menidia* offspring, with implications for size at age that could threaten recruitment and alter the flow of energy through the food web.

**Conclusions**

With this simple and widely applicable DEBkiss model we were able to attribute hypoxia-related variability in total length, egg buffer mass, and survival over time to changes in DEB processes. Similar approaches have applied correction factors to DEB parameters to model other species’ responses to hypoxia (Lavaud et al., 2019; Aguirre-Velarde et al., 2019) and other stressors such as seawater acidification (Jager et al., 2016; Pousse et al., 2022) and pollutants (Muller et al., 2010; Desforges et al., 2017). The success of this approach with a wide variety of stressors makes it an ideal supplement to multistressor experiments, which are limited by logistical constraints. Modeling stressor effects with DEBkiss parameters can yield additional information about energetic mechanisms of responses and, with careful attention to the assumptions being made, may be useful in extrapolating stressor effects to additional magnitudes or combinations of stressors that would have been impractical to test experimentally, or to species with certain shared physiology or life history traits (Goussen et al., 2020; Boult and Evans, 2021). As lifelong, constant oxygen conditions are unrealistic in nature, the patterns modeled in this study should not be interpreted as a standalone prediction of what will happen to wild *M. menidia* populations as coastal hypoxia intensifies. Instead, this approach demonstrates the value of identifying DEB parameters responsible for whole-organism effects of hypoxia to understand underlying energetic processes that are often time, labor, and cost-intensive to measure empirically, particularly in the early life stages, when biomass available for sampling is small and developmental changes are rapid. Through doing so we were able to support the utility of modeling inhibition and damage to synthesizing units and highlight the conversion of assimilates to structure as a primary mechanism by which hypoxia impacts *M. menidia*. Measuring suborganismal processes to identify physiological modes of action can refine this model so that it can better model this species’ response to realistic hypoxia scenarios and, ultimately, how reductions in conversion efficiency could affect energy flow through food webs.