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Does ocean acidification affect the bioenergetics and susceptibility to pathogenic disease in juvenile Pacific herring (Clupea pallasii)?

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Does ocean acidification affect the bioenergetics and susceptibility to viral disease in Pacific herring?

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Introduction

In fish, sublethal environmental stressors can compromise aerobic performance and immune function. The Salish Sea ecosystem is particularly vulnerable to ocean acidification, but whether near-future pCO_2 levels reduce growth and increase susceptibility to pathogenic disease in marine fish remains understudied. Pacific herring (*Clupea pallasii*) is a keystone forage fish that is highly vulnerable to epizootic outbreaks of viral hemorrhagic septicemia virus (VHSV). To this end, we monitored the growth of juvenile herring reared under predicted future pCO_2 levels, measured juvenile critical swim speed, and tested their resilience to an induced VHSV infection.

Methods

Wild Pacific herring we reared at the USGS Marrowstone Marine Field Station from hatch under three pCO_2 conditions: low (500 µatm); intermediate (1500 µatm); and high (3000 µatm). Fish were subsampled for size measurements every 3-5 days and juveniles were were tested for critical swim speed (Ucrit) 90-105 days-post hatch (dph) using a Blazka-type swim flume. After 98 dph, groups of 60 fish (three groups per pCO_2 treatment) were treated with VHSV (mean waterborne exposure levels were tittered at 807 PFU mL^{-1}) for 2 h before the tanks were flushed. Mortalities were collected for 28 days post-infection (dpi) and tissues from all mortalities and survivors were measured for viral titer in kidney / spleen pools by plaque assay onto polyethylene glycol-pretreated EPC cells.

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Virus Challenge

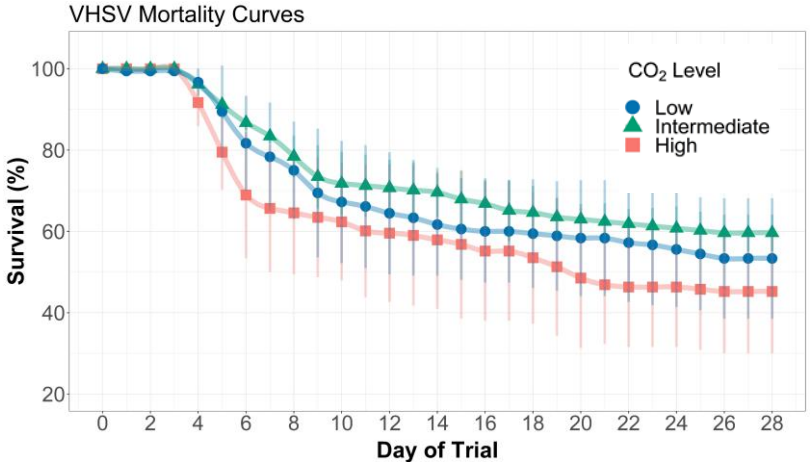

Fig. 1: Juvenile herring survival post-VHSV exposure. Colored shapes show daily mean survival percentages per CO_2 treatment. Vertical lines denote ± 1 s.d.

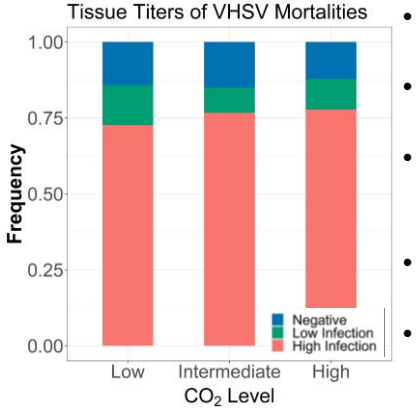

Fig. 2: The frequency of viral titers binned into three categories: fish negative for VHSV at time of death, showing low infections (<400 PFU·g^{-1}) , and high infections (>400 PFU·g^{-1})

- Herring from all pCO_2 treatment were susceptible to VHSV.
- Initial mortality rates 4-8 dpi slightly faster under high pCO_2.
- Second mortality wave (18-21 dpi) under high pCO_2 hinted at a longer infectious period.
- However, no significant difference in survival at day 28.
- No difference in viral titers taken from tissues of VHSV mortalities.

Growth & Critical Swim Speed

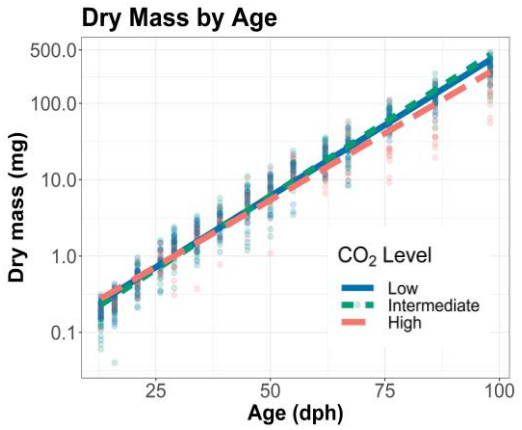

Fig. 3: Dry mass (mg) of fish sampled from 1 to 98 dph. Colored circles show individual fish. Colored hashed lines represent linear regression fits for log(dry mass) by age per pCO_2 treatment.

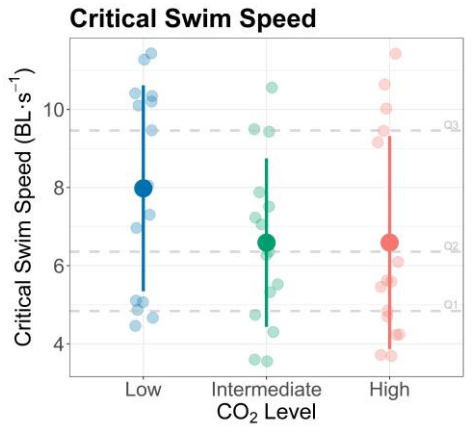

Fig. 4: Critical swim speed (scaled to body lengths per second) of juveniles measured between 90-105 dph. Large circles show treatment means and vertical lines denote ± 1 s.d. Small circles show individual fish. Horizontal dashed lines identify quartiles calculated on combined CO_2 data.

- Long-term growth rate (dry mass) under high pCO_2 showed a small (and not significant) reduction (-6.2%) compared to control fish.
- Juveniles from both elevated pCO_2 treatments showed an average reduction in Ucrit (scaled to body lengths per second) but high variability precluded a statistically significant result.

Conclusions

- Juvenile Pacific herring were relatively tolerant to pCO_2 levels 3× and 6× current spring/summer surface ocean conditions in the Salish Sea.
- Early life growth was largely insensitive to pCO_2 level.
- A general trend of slower critical swim speed under elevated pCO_2, with most poor performing individuals from high pCO_2.
- High pCO_2 induced an increase in initial VHSV mortalities, but final survival was not significantly different between treatments.