Abstract

Following the Deepwater Horizon oil spill in April 2010, much research has been conducted on the cardiotoxic effects of oil on fish. Sensible life history stages, such as the embryonic period, are especially vulnerable to the cardiotoxic effects of oil. Embryonic fish may be more vulnerable because their cardiovascular system is still rapidly developing, and they may lack the ability to compensate for oil exposure. This study examines the cardiotoxic effects of oil on developing cardiovascular systems of fish. However, much of this research has focused on rapidly developing anuran species, with little emphasis on estuarine species with longer embryological periods. Moreover, previous studies have used heart rate as the primary endpoint to measure cardiac performance in embryos and larvae as an endpoint that is in some cases overlooked in embryos in cardiac performance. This study aims to fill these knowledge gaps and provide a more holistic approach for assessing the effects of oil on cardiac function by exposing sheepshead minnow (Cyprinodon variegatus) embryos to two oil doses (150 and 300 µg/L tPAH) throughout embryonic development and measuring cardiac responses through the identification of cardiotoxic phenotypes (pericardial edema) as well as analysis of cardiac output at day 4 post fertilization (dpf). Results of this study show significant increases in pericardial edema at both oil doses relative to controls as well as significantly reduced cardiac output driven by reductions in ventricular stroke volume. This study is one of the first to assess cardiac output in embryos exposed to oil and methods described here allow for more physiologically relevant measures of cardiac performance in early life stages through established and non-invasive measures.

Introduction

- The Deepwater Horizon oil spill coincided with the spawning season of many marine fisheries
- Weathered oil has high concentrations of 3 and 4 ring PAHs
- Known cardiotoxins, especially phenanthrenes

Hypothesis

Embryos exposed to oil will exhibit cardiotoxic phenotypes and have a reduced cardiac output resulting from altered heart rates and stroke volumes.

Methods

- Sheepshead minnow embryos exposed to oil throughout embryonic period
- Exposure concentrations were 150 and 300 µg/L tPAH
- Exposure solutions were refreshed daily to maintain stable PAH-exposure

Results

- Significant increases in pericardial edema

Oil induced cardiac effects in embryonic sheepshead minnows, <em>Cyprinodon variegatus</em>

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Conclusions

- Embryos exposed to oil induce cardiotoxic phenotypes in embryonic sheepshead minnows
- Cardiac output may be a better measure of cardiac performance than heart rate
- Pericardial edema and incomplete/failed heart looping
- Significant reductions in cardiac output
- Cardiac output may be a better measure of cardiac performance than heart rate
- No differences in heart rates associated with oil exposure
- Reduced ventricular stroke volume results in significantly reduced cardiac output when embryos are exposed to oil
- No observed mortality suggests LC50 value for embryonic sheepshead minnows is above 300 µg/L tPAH
- Further studies tracing sublethal effects of impaired cardiac function in post-hatch larval, juvenile, and adult individuals are needed