

Environmental Science & Technology

During the embryonic/larval stage several developmental effects. The effective sublethal/larval exposure was 1/2000 that for the juveniles and the larval body burden would be expected to increase over time through growth dilution and hepatic metabolism, potentially eliminating an effect of a residual body burden of PAHs that persisted to the juvenile stage. The observed larval effects of peroxyl and polyaromatic hydrocarbons, which were similar to those previously described for a number of different hydrocarbons to various extents of crude oil (1984, 1985), had further evidence in a likely early developmental stage for the larval effects on swimming performance.

The available data to date indicate that our exposure concentrations, which ranged from 1.2-30 µg L<sup>-1</sup> DWH, likely represent environmentally realistic exposure scenarios.

limitation in oxygen uptake or delivery that decreases  $\dot{V}O_2$  (e.g., reduced cardiac output, anemia, or increased metabolic demand) or an effect on the ability of the heart to contract and thus increase  $\dot{V}O_2$  (e.g., peroxyl, aromatic, metabolism of unsaturated, or increased oxygenator rates).<sup>10</sup> Second, glycolytic fueled white muscle may be recruited to sustain high swimming speeds approaching and including  $U_{crit}$ . Thus, an impairment to this recruitment may also limit  $U_{crit}$ . Finally, there is evidence that behavior plays a role in setting swimming speed when fish cannot generate or sustain a given speed, a particular concern when using more variable, when forward movement is greatly limited.<sup>10</sup> Although most of these potential mechanisms could conceivably play a role in reducing the  $U_{crit}$  of oil-exposed fish in our experiments, we chose to focus on development as the best mechanism of disturbance

mahi. Although direct measurements of cardiac performance were not made, the lack of an effect on aerobic scope strongly indicates that cardiac output was maintained, thereby suggesting a mechanism(s) for the reduced  $U_{crit}$  other than, or in addition to, one that limits aerobic scope for juvenile mahi-mahi exposed to DWH crude oil.

effect on the juvenile fish that in our study resulted in a 20% impairment relative to control at this time. Nevertheless, it is important to note that the effective concentrations reported herein may underestimate toxicity in the natural environment where other stresses with the potential to increase cardiac swimming rate (hypoxia, temperature, and O<sub>2</sub> demand) are commonly present. Furthermore, such effects on metabolic rate also may be other important physiological effects, such as lipid and protein, which also would be the left path along the line.<sup>10</sup>

There are several physiological behavioral mechanisms that could potentially contribute to a reduced  $U_{crit}$ . First,  $U_{crit}$  may decrease as a result of diminished aerobic scope deriving from a

compensating, limited aerobic scope (1985) or from the same cause affecting larval metabolism, may indicate that the most severely impaired fish with cardiac defects were selected out during the rearing process. Similar fish to the wild, however, would not have been subjected to the same high intensity pressures that no compensation but rather pressure from other species and environmental stresses as discussed above. In the end, impaired aerobic swimming performance by larval fish exposed to crude oil may result both from an as yet unmeasured physiological effect, as indicated by the present study, and a reduced aerobic scope owing to developmental cardiac defects or induced by other factors. Nevertheless, considering that in all likelihood the most highly ill fish were