

CO-EXPOSURE TO SUNLIGHT ENHANCES THE TOXICITY OF NATURALLY WEATHERED DEEPWATER HORIZON OIL TO EARLY LIFESTAGE RED DRUM (*SCIAENOPS OCELLATUS*) AND SPECKLED SEATROUT (*CYNOSCION NEBULOSUS*)

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**Abstract:** The 2010 *Deepwater Horizon* oil spill resulted in the accidental release of millions of barrels of crude oil into the Gulf of Mexico. Photo-induced toxicity following co-exposure to ultraviolet (UV) radiation is 1 mechanism by which polycyclic aromatic hydrocarbons (PAHs) from oil spills may exert toxicity. Red drum and speckled seatrout are both important fishery resources in the Gulf of Mexico. They spawn near-shore and produce positively buoyant embryos that hatch into larvae in approximately 24 h. The goal of the present study was to determine whether exposure to UV as natural sunlight enhances the toxicity of crude oil to early lifestage red drum and speckled seatrout. Larval fish were exposed to several dilutions of high-energy water-accommodated fractions (HEWAFs) from 2 different oils collected in the field under chain of custody during the 2010 spill and 3 gradations of natural sunlight in a factorial design. Co-exposure to natural sunlight and oil significantly reduced larval survival compared with exposure to oil alone. Although both species were sensitive at PAH concentrations reported during the *Deepwater Horizon* spill, speckled seatrout demonstrated a greater sensitivity to photo-induced toxicity than red drum. These data demonstrate that even advanced weathering of slicks does not ameliorate the potential for photo-induced toxicity of oil to these species. *Environ Toxicol Chem* 2017;36:780–785. © 2016 SETAC

**Keywords:** Polycyclic aromatic hydrocarbon   Red drum   Speckled seatrout   *Deepwater Horizon*   Photo-induced toxicity

## INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a class of organic molecules with multiple carbon rings. As a class, PAHs vary widely in toxicological and chemical properties [1]. In general, PAHs are lipophilic and can bioaccumulate [2]. They exert toxicity through several mechanisms, but photo-induced or photo-enhanced toxicity often occurs at concentrations below the threshold of other modes of toxicity [3–5]. Photo-induced toxicity is the phenomenon in which a compound exhibits increased toxicity in the presence of certain light wavelengths [5]. Effects of PAH photo-induced toxicity include increased mortality [6–11], reduced fecundity [3], delayed hatching [12] increased photo-avoidance behaviors [5,13], and feeding inhibition [14]. The photo-induced toxicity of PAHs to aquatic species is well documented in the literature [5,6,11,15–17].

On the 20 April 2010, the mobile offshore drilling unit *Deepwater Horizon* experienced a series of events that resulted in the sinking of the vessel itself and subsequent release of millions of barrels of oil from the wellhead unit until it was sealed on 15 July 2010 [18]. Crude oil contains PAHs including those with photodynamic activity [19]. During the *Exxon Valdez* and *Prestige* oil spills, for example, releases of oil resulted in potential photo-induced PAH toxicity to early lifestage finfish [11,16,20], shellfish [9,12,21], and marine zooplankton [21].

Red drum (*Sciaenops ocellatus*) are found along the southern Atlantic and Gulf of Mexico coasts [22]. It is an important commercial and recreational species. Commercial fishing of red drum peaked in 1986 at nearly 15 million pounds caught [23]. Concerns about stock depletion led to responses at the state and federal level to protect red drum populations, including US Executive Order 13449 in 2007, which limited the catch of red drum in federal waters [24]. Recreational fishing of red drum has increased since 1986 from 5 million pounds to more than 15 million pounds harvested in 2008 [23]. The mean catch between 2002 through 2011 for Gulf of Mexico red drum was 8.9 million fish [23].

Red drum spawn in the open water neritic zone beginning in August and continuing into December; however, regional variation in timing may shift this window by several weeks [22]. The number of eggs released per spawning event is dependent on female body size, varying in laboratory conditions from 200 000 eggs to in excess of 2 million eggs per female [22]. Over the course of a single spawning season, a female may spawn every 2 d to 4 d [25]. Red drum larvae migrate with the tide into estuaries and settle in the cover of submerged seagrasses [22].

Similarly, speckled seatrout (*Cynoscion nebulosus*) are distributed as far north as Virginia (USA) and are found along the shores of all Gulf of Mexico states [26]. The mean catch, combining harvest and release, between 2002 through 2011 for Gulf of Mexico speckled seatrout was 29.9 million fish [26]. Overfishing led to management responses and bans on several fishing methods to allow for stocks to recover. Speckled seatrout spawn from April through October, but, again, this window of

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time varies regionally based on local conditions [26]. Speckled seatrout spawn in a variety of habitats; they have been observed to spawn over submerged seagrass, among floating masses of detritus, and in tidal areas with little or no vegetation present [26]. Spawning typically occurs in relatively shallow waters (3–4.6 m) [26]. Although older, larger females spawn more frequently, over the course of a single season, a female may spawn every 2 d to 9 d and release >1000 eggs/g body weight [26]. Speckled seatrout larvae typically hatch in estuarine habitat very close to their spawning area [26]. As larvae, they remain associated with structure such as seagrass until they reach the juvenile lifestage [26].

In both red drum and speckled seatrout, the eggs are buoyant, and larvae hatch near the surface where they are likely to encounter ultraviolet (UV) light. The goal of the present study was to determine the sensitivity of red drum and speckled seatrout larvae to photo-induced toxicity following exposure to *Deepwater Horizon* spill oil. To accomplish this goal, larvae of both species were exposed to a range of dilutions of 2 different field-collected oils and 3 gradations of UV. Data from the present study may be used as a component of the *Deepwater Horizon* Natural Resource Damage Assessment.

## MATERIALS AND METHODS

### Test organisms

Organisms were obtained from in-house cultures at the Texas Parks and Wildlife Sea Center (Lake Jackson, TX, USA). Two species were tested, red drum and speckled seatrout. Prior to use in bioassays, embryos were held until hatch (~18–24 h post fertilization [hpf]). All organisms used in tests were less than 48 hpf at the commencement of testing.

### Test solutions

Two oil samples collected in the field under chain of custody were used in these experiments. Slick A is a surface slick oil collected on 29 July 2010 from the hold of barge number CTC02404, which was receiving surface slick oil from various skimmer vessels responding to the spill. Slick B is a more weathered surface slick oil collected on 19 July 2010 by the skimmer vessel *USCGC Juniper*. Both of these oil samples are routinely used in testing as part of the *Deepwater Horizon* Natural Resource Damage Assessment. Filtered seawater from the Sea Center Hatchery (collected from a gulf channel) was used in all control solutions and in preparation of treatment dilutions.

Stocks of high-energy water-accommodated fraction (HEWAF) of Slick A or Slick B were prepared by mixing 4 L of filtered seawater with a mass of slick oil (1 g/L) in a Waring CB15 blender, on low power, for 30 s. The mixture was transferred to a separatory funnel and allowed to settle for 1 h before sampling for PAH analysis and utilization in test dilutions. Exposure solutions were prepared by dilution in filtered seawater to a nominal percentage of HEWAF. Water quality parameters were taken with dilution preparation and found to be within the following ranges: 6.1 mg/L to 6.8 mg/L dissolved oxygen; 30.7 ppt to 32.3 ppt salinity; 49.8 mS to 53.0 mS conductivity; and 7.8 pH to 8.2 pH. Samples of stocks and dilutions were taken with every preparation and shipped (at 4 °C) to ALS Environmental (Kelso, WA, USA) for analysis. Fifty PAH analytes being used for Natural Resource Damage Assessment analyses were quantified using gas chromatography–mass spectroscopy in single-ion monitoring mode (GC/MS–SIM), based on US

Environmental Protection Agency method 8270D [27]. The sum concentrations of these 50 PAH analytes are hereafter referred to as tPAH<sub>50</sub>.

### Toxicity tests

Larvae were exposed to 1 of a range of PAH concentrations combined with 1 of a range of UV intensities in a fully factorial design for approximately 20 h. Exposures were conducted in 250-mL borosilicate glass crystallizing dishes containing 10 larvae per dish with no renewal of exposure solutions. The exposure period included an approximately 8-h equilibration period (under indoor low-intensity lighting), followed by a 5-h to 6-h solar exposure, followed by a 5-h no-UV period before mortality was assessed. Each bioassay had 5 PAH treatments with 15 replicate dishes per PAH treatment, which were divided into 5 dishes per PAH treatment within each UV treatment. Tests were conducted with 3 UV ( $\lambda = 380$  nm) treatments (nominally 10%, 50%, and 100% ambient natural sunlight).

Replicate dishes were suspended in an outdoor, flow-through water bath to maintain a target temperature of 28 °C. Dishes were floated in polystyrene foam insulation board with holes cut to hold replicate dishes in contact with the temperature bath water across the underside, and the majority of the sidewall. Sunlight was used as the source of UV radiation. Screening materials were suspended over selected dishes to achieve treatment gradations of UV. A specially formulated plastic sheet transparent to UV was used for a full-intensity (100% ambient) UV treatment (Professional Plastics). A metal mesh screen was added over the top of the full-intensity plastic as an additional, neutral-density filter to achieve an approximately 50% ambient UV treatment. A different formulation of plastic sheet allowing transmission of <10% of ambient UV (Professional Plastics) was used as a control. The UV was measured continuously during the exposures using a Biospherical Instruments BIC2104R radiometer.

### Phototoxic units

All tests were performed outdoors using ambient UV. Tests not performed in parallel received different UV doses as a result. To account for differences in UV exposure, a phototoxic unit was calculated using methods similar to those of Oris and Giesy [5] and previously described in Alloy et al. [12]. Briefly, 14 known phototoxic PAHs present in the water-accommodated fraction (WAF) preparations were used in the calculations: anthracene, benzo[*a*]anthracene, benzo[*e*]pyrene, benzo[*ghi*]perylene, chrysene, fluoranthene, fluorene (as well as C1 and C2), phenanthrene (as well as C1, C2, and C3), and pyrene. The aqueous concentration of each PAH was calculated as a molar value and multiplied by its relative photodynamic activity compared with anthracene [5]. The sum of each of these values was used to calculate the sum concentration of anthracene equivalents. The UV doses are reported as the integration of the test duration at a resolution of 1 s, expressed as mW/s/cm<sup>2</sup>. The anthracene equivalent concentration is multiplied by the integration of the UV irradiance at  $\lambda = 380$  nm to produce the phototoxic dose.

$$\text{Phototoxic dose} = \text{Anthracene equivalent} \times \text{mW/s}$$

where mW/s refers to the integration of irradiance only at  $\lambda = 380$  nm. Phototoxic units are expressed as  $\mu\text{M/L} \times \text{mW} \times \text{s/cm}^2$ .

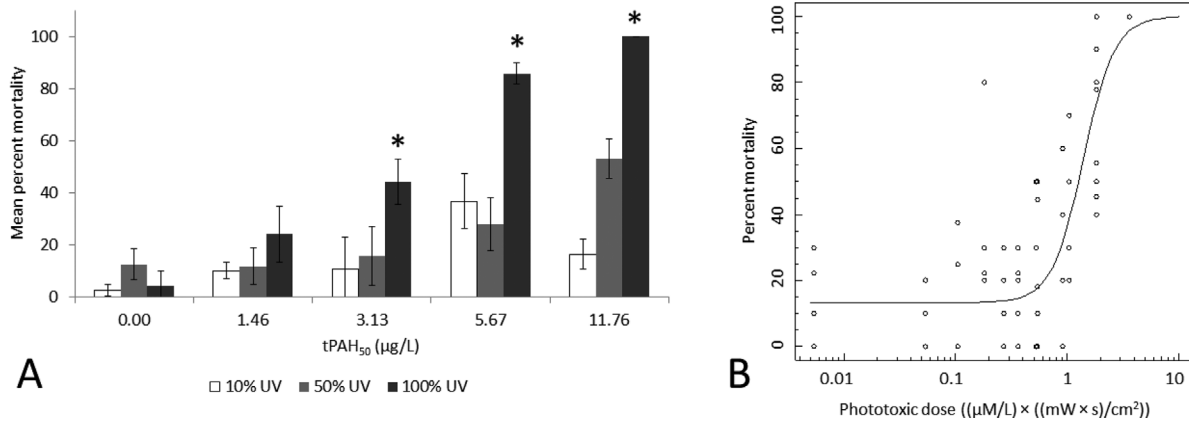


Figure 1. (A) Mean percentage of mortality  $\pm$  1 standard error of larval red drum exposed to slick A high-energy water-accommodated fraction (HEWAF) and 1 of 3 ultraviolet (UV) treatments. Asterisks indicate significant difference from control treatment of the same UV ( $\alpha = 0.05$ ). (B) Phototoxic dose-response model of mortality in larval red drum exposed to slick A HEWAF and UV. The model consists of the 10%, 50%, and 100% UV treatments, and the 5 polycyclic aromatic hydrocarbon (PAH) treatments.

### Statistical analyses

For each toxicity test, percentage survival data was arcsine-transformed to meet analysis of variance (ANOVA) assumptions. A 2-factor ANOVA with a Dunnett's post hoc test was used to determine differences in survival between treatments using UV treatment and tPAH<sub>50</sub> concentration as factors. The ANOVAs were performed using the statistical software JMP (Ver 11, SAS Institute). Statistical significance was determined using  $\alpha = 0.05$ . Median lethal concentrations (LC50s) for whole-test phototoxic dose, and individual UV treatments within toxicity tests were calculated using the *drc* package in R (Ver 3.1.2) [28].

## RESULTS

Red drum larvae were exposed to Slick A HEWAF dilutions containing 0.00 μg/L, 1.46 μg/L, 3.13 μg/L, 5.67 μg/L, and 11.76 μg/L tPAH<sub>50</sub>. Red drum exposed to the 100% UV treatment received a total integrated dose of 705.79 mW × s/cm<sup>2</sup> with a mean intensity ( $\pm$  1 standard deviation [SD]) of 0.038  $\pm$  0.021 mW/cm<sup>2</sup>/s.

Mean survival of all treatments is detailed in Figure 1A. Significant mortality occurred in the 100% UV treatment following exposure to tPAH<sub>50</sub> concentrations  $\geq$  3.13 μg/L

(ANOVA,  $F_{(14,59)} = 11.42$ , Dunnett's post hoc test,  $p < 0.01$ ). Median mortality tPAH<sub>50</sub> concentrations (LC50s) were calculated separately by UV treatment. The tPAH<sub>50</sub> LC50 for the 50% UV and the 100% UV treatments were 12.2 μg/L tPAH<sub>50</sub> (95% confidence interval = 7.37–17.0 μg/L tPAH<sub>50</sub>) and 3.42 μg/L tPAH<sub>50</sub> (95% confidence interval = 2.47–4.37 μg/L tPAH<sub>50</sub>) respectively. A model using phototoxic units was also used to calculate a phototoxic LC50. The phototoxic LC50 for Slick A HEWAF to red drum larvae was 1.41 μM/L × mW × s/cm<sup>2</sup> (95% confidence interval = 1.17–1.65 μM/L × mW × s/cm<sup>2</sup>; Figure 1B).

Red drum larvae were exposed to Slick B dilutions containing 0.00 μg/L, 2.27 μg/L, 5.11 μg/L, and 8.25 μg/L tPAH<sub>50</sub>. Red drum larvae within the 100% UV treatment, exposed to Slick B HEWAF, received a total integrated dose of 846.9 mW × s/cm<sup>2</sup> with a mean intensity of 0.032  $\pm$  0.015 mW/cm<sup>2</sup>/s. Mean survival in all treatments is detailed in Figure 2A. Significant mortality occurred in the 50% and 100% UV treatments following exposure to tPAH<sub>50</sub> concentrations  $\geq$  5.11 μg/L (ANOVA,  $F_{(11,48)} = 80.47$ , Dunnett's post hoc test,  $p < 0.01$ ) and  $\geq$  2.27 μg/L, respectively (Dunnett's post hoc test,  $p < 0.01$ ). The tPAH<sub>50</sub> LC50 in the 50% and 100% UV treatments were 5.79 μg/L tPAH<sub>50</sub> (95% confidence interval = 5.45–6.14 μg/L tPAH<sub>50</sub>) and 1.99 μg/L

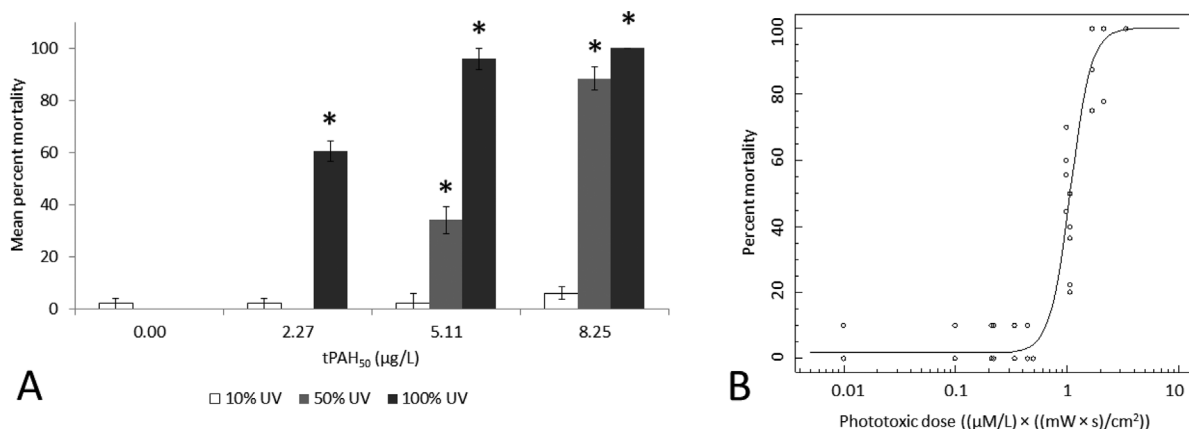


Figure 2. (A) Mean percentage of mortality  $\pm$  1 standard error of larval red drum exposed to slick B high-energy water-accommodated fraction (HEWAF) and 1 of 3 ultraviolet (UV) treatments. Asterisks indicate significant difference from control treatment of the same UV ( $\alpha = 0.05$ ). (B) Phototoxic dose-response model of mortality in larval red drum exposed to slick B HEWAF and UV. The model consists of the 10%, 50%, and 100% UV treatments, and the 5 polycyclic aromatic hydrocarbon (PAH) treatments.

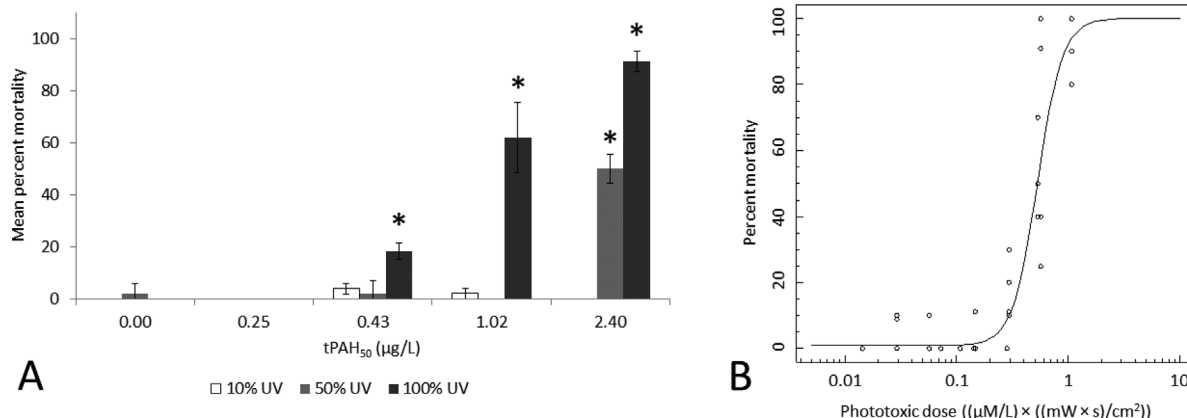


Figure 3. (A) Mean percentage of mortality  $\pm$  1 standard error of larval speckled seatrout exposed to slick A high-energy water-accommodated fraction (HEWAF) and 1 of 3 ultraviolet (UV) treatments. Asterisks indicate significant difference from control treatment of the same UV ( $\alpha = 0.05$ ). (B) Phototoxic dose–response model of mortality in larval speckled seatrout exposed to slick A HEWAF and UV. The model consists of the 10%, 50%, and 100% UV treatments, and the 5 polycyclic aromatic hydrocarbon (PAH) treatments.

tPAH<sub>50</sub> (95% confidence interval = 1.78–2.19 μg/L tPAH<sub>50</sub>), respectively. The phototoxic LC<sub>50</sub> for Slick B to red drum larvae was 1.06 μM/L × mW × s/cm<sup>2</sup> (95% confidence interval = 0.993–1.12 μM/L × mW × s/cm<sup>2</sup>; Figure 2B).

Speckled seatrout larvae were exposed to Slick A dilutions containing 0.00 μg/L, 0.25 μg/L, 0.43 μg/L, 1.02 μg/L, and 2.49 μg/L tPAH<sub>50</sub>. Speckled seatrout larvae in the 100% UV treatment exposed to Slick A HEWAF received a total integrated dose of 1108.9 mW × s/cm<sup>2</sup> with a mean intensity of 0.042 ± 0.019 mW/cm<sup>2</sup>/s. Mean survival in all treatments is detailed in Figure 3A. Significant mortality occurred at tPAH<sub>50</sub> concentrations  $\geq$  2.40 μg/L in the 50% UV treatment (ANOVA,  $F_{(14,60)} = 24.89$ , Dunnett's post hoc test,  $p < 0.01$ ), and  $\geq$  0.43 μg/L in the 100% UV treatment (Dunnett's post hoc test,  $p < 0.01$ ). The tPAH<sub>50</sub> LC<sub>50</sub> in the 50% and 100% UV treatments was 2.41 μg/L tPAH<sub>50</sub> (95% confidence interval = 0.741–4.07 μg/L tPAH<sub>50</sub>) and 0.827 μg/L tPAH<sub>50</sub> (95% confidence interval = 0.626–1.03 μg/L tPAH<sub>50</sub>), respectively. The phototoxic LC<sub>50</sub> for Slick A to speckled seatrout larvae was 0.516 μM/L × mW × s/cm<sup>2</sup> (95% confidence interval = 0.478–0.553 μM/L × mW × s/cm<sup>2</sup>; Figure 3B).

Speckled seatrout larvae were exposed to Slick B dilutions containing 0.00 μg/L, 0.08 μg/L, 0.18 μg/L, 0.56 μg/L, and 1.63 μg/L tPAH<sub>50</sub>. Speckled seatrout larvae in the 100% UV

treatment exposed to Slick B HEWAF received a total integrated dose of 1439.73 mW × s/cm<sup>2</sup> with a mean intensity of 0.056 ± 0.019 mW/cm<sup>2</sup>/s. Mean survival in all treatments is detailed in Figure 4A. Significant mortality occurred at 1.63 μg/L tPAH<sub>50</sub> in the 50% UV treatment (ANOVA,  $F_{(14,60)} = 26.53$ , Dunnett's post hoc test,  $p < 0.01$ ), and tPAH<sub>50</sub> concentrations  $\geq$  0.18 μg/L in the 100% UV treatment (Dunnett's post hoc test,  $p < 0.01$ ). The tPAH<sub>50</sub> LC<sub>50</sub> in the 50% and 100% UV treatments were 1.01 μg/L tPAH<sub>50</sub> (95% confidence interval = 0.802–1.21 μg/L tPAH<sub>50</sub>) and 0.182 μg/L tPAH<sub>50</sub> (95% confidence interval = 0.159–0.206 μg/L tPAH<sub>50</sub>), respectively. The phototoxic LC<sub>50</sub> was 0.287 μM/L × mW × s/cm<sup>2</sup> (95% confidence interval = 0.239–0.335 μM/L × mW × s/cm<sup>2</sup>; Figure 4B).

## DISCUSSION

Co-exposure to natural sunlight increased the toxicity of *Deepwater Horizon* spill oil to larval red drum and speckled seatrout in a tPAH<sub>50</sub> and UV dose-dependent manner. All calculated tPAH<sub>50</sub> LC<sub>50</sub>s (0.18–5.78 μg/L) in the present study are within the lower range of PAH concentrations (0–84.8 μg/L) reported in the *Deepwater Horizon* spill area [29]. Other photo-induced PAH toxicity tests, including those using

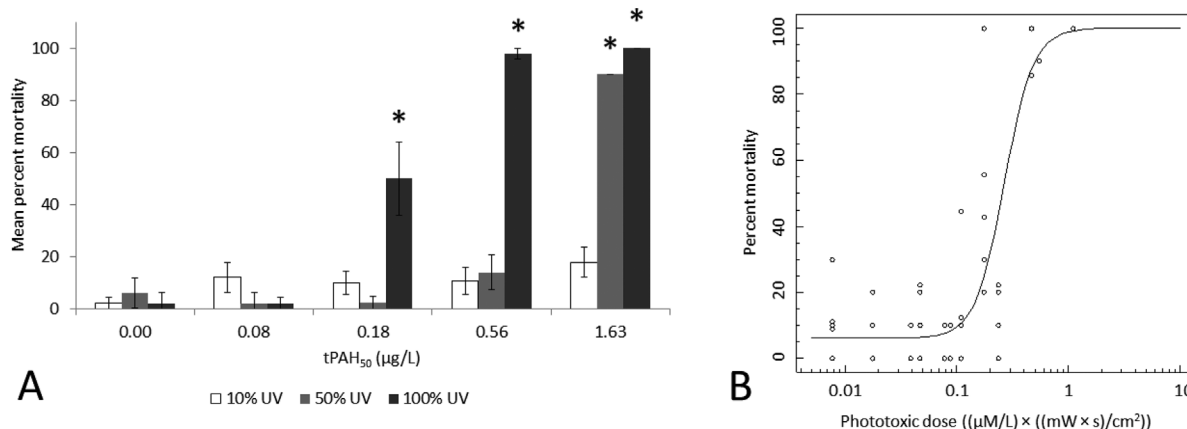


Figure 4. (A) Mean percentage of mortality  $\pm$  1 standard error of larval speckled seatrout exposed to slick B high-energy water-accommodated fraction (HEWAF) and 1 of 3 ultraviolet (UV) treatments. Asterisks indicate significant difference from control treatment of the same UV ( $\alpha = 0.05$ ). (B) Phototoxic dose–response model of mortality in larval speckled seatrout exposed to slick B HEWAF and UV. The model consists of the 10%, 50%, and 100% UV treatments, and the 5 polycyclic aromatic hydrocarbon (PAH) treatments.

*Deepwater Horizon* spill oil, report total PAH median effective concentration (EC<sub>50</sub>) or LC<sub>50</sub> values that are within an order of magnitude of those observed in the red drum and speckled seatrout tests [11,12,16,20]. It is worth noting that the tPAH<sub>50</sub> values in the present study are based on analysis of chemistry samples taken immediately after WAF preparation and do not account for loss of PAH over time in the actual exposures. Given that exposure solutions were not renewed for the duration of the bioassay, the toxicity values reported in the present study are likely higher than if we calculated them using time-integrated doses because of the reduction in PAH concentration in the exposure solutions over time.

The phototoxic LC<sub>50</sub>s (0.29–1.4  $\mu\text{M/L} \times \text{mW} \times \text{s/cm}^2$ ) are substantially lower than those reported in 2 similar studies on early lifestage mahi-mahi and blue crab [11,12]. The reported EC<sub>50</sub>s (phototoxic dose) for effects on hatching success in embryonic mahi-mahi were between 6.77 and 11.8  $\mu\text{M/L} \times \text{mW} \times \text{s/cm}^2$  (Slick A only). Median lethal concentrations (phototoxic dose) reported for blue crab zoea were 9.5 to 20.6  $\mu\text{M/L} \times \text{mW} \times \text{s/cm}^2$  (Slick A), and 9.9  $\mu\text{M/L} \times \text{mW} \times \text{s/cm}^2$  (Slick B). Although it can be difficult to compare oil photo-induced toxicity across taxa and lifestages because of differences in surface area to volume ratios, maternally transferred pigments and antioxidants, and phototactic behaviors, these data suggest that red drum and speckled seatrout, both members of the drum family (*Sciaenidae*), may be among the more sensitive test organisms to oil photo-induced toxicity. It is unlikely that UV sensitivity alone explains this result because survival in 10%, 50%, and 100% UV controls (no PAH) was >90% in most cases. Regardless of the mechanism, the sensitivity of larval red drum and speckled seatrout lowers the range at which photo-induced PAH toxicity is observed among Gulf of Mexico species.

Both red drum and speckled seatrout are estuarine species. This makes likely exposure scenarios more complex than species that both spawn and live exclusively in open waters. Immediately after hatching, red drum and speckled seatrout larvae at a similar age as those used in the present study can be found in both open and near-shore waters [25,26]. In open water areas of the Gulf of Mexico, UV-A Z<sub>10</sub> (the depth at which only 10% of surface UV-A irradiance remains) can be as deep as 37 m [30]. This suggests that, in open water areas, the likelihood for co-exposure to PAH and UV is quite high. In coastal areas, UV attenuation varies greatly, and Z<sub>10</sub> values as deep as 5 m and as shallow as <1 m have been reported [30]. Thus, the potential for larvae to be exposed to UV in habitats where oil was trapped among seagrasses, salt marsh vegetation, and sediments [31] may be more site- and season-specific. However, the sensitivity of red drum and speckled seatrout larvae to oil photo-induced toxicity may result in significant effects even when UV exposure is relatively low. For example, we observed >80% mortality in speckled seatrout larvae exposed to 1.63  $\mu\text{g}$  tPAH<sub>50</sub>/L of Slick B under 50% ambient natural sunlight in less than 1 d.

Speckled seatrout were more sensitive than red drum to photo-induced PAH toxicity. Tolerance to photo-induced PAH toxicity in fishes may, in part, be related to the robustness of osmoregulatory systems [32]. Mclosky and Oris [33] observed that when dissolved oxygen and temperature were held constant, opercular ventilation rates increased in bluegill fish exposed to both anthracene and UV. This can be the result of accrued damage to the gills, which causes difficulty in gas exchange and maintenance of ion balance [32,33]. Red drum and speckled seatrout are both

euryhaline organisms. However, speckled seatrout is reported to be less tolerant of hypoxic conditions than red drum [34,35]. Because both species are adept osmoregulators, the difference between the 2 species in sensitivity to photo-induced PAH toxicity may be attributable to speckled seatrout's lesser hypoxia coping ability.

Co-exposure to UV as natural sunlight greatly increases the toxicity of *Deepwater Horizon* spill oil to larval red drum and speckled seatrout. Effects were observed during short test durations (<24 h) and were well within the range of tPAH<sub>50</sub> concentrations reported during the spill. Data presented in the present study suggest that these 2 species may be among the most sensitive tested to date to oil photo-induced toxicity. Significant toxicity was observed even under reduced (50% ambient) UV intensity, indicating that effects in the field may occur in more turbid, near-shore coastal waters as well as highly transparent open water.

*Supplemental Data*—The Supplemental Data are available on the Wiley Online Library at DOI: 10.1002/etc.3640.

*Acknowledgment*—The present study was supported as part of the *Deepwater Horizon* Natural Resource Damage Assessment. Data presented in the present study are a subset of a larger toxicological database that is being generated as part of the *Deepwater Horizon* Natural Resource Damage Assessment, and therefore, these data will be subject to additional analysis and interpretation, which may include interpretation in the context of additional data not presented in this publication. Thanks to the Texas Parks and Wildlife's Sea Center for the use of their aquaculture facilities and for providing test organisms.

*Data availability*—Data requests should be addressed to A.P. Roberts at the University of North Texas (Aaron.Roberts@unt.edu). The data presented in the manuscript and related to the present study are part of a larger database related to the United States National Oceanic and Atmospheric Administration's natural resource damage assessment of the *Deepwater Horizon* oil spill. Data requests may have to be reviewed by several parties before being released to the public.

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