Impacts of climate change on the ecotoxicology of chemical contaminants in estuarine organisms

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Abstract   Global climate change effects will vary geographically, and effects on estuaries should be independently considered. This review of the impacts of climate change on the ecotoxicology of chemical contaminants aims to summarize responses that are specific to estuarine species. Estuarine organisms are uniquely adapted to large fluctuations in temperature, salinity, oxygen, and pH, and yet future changes in climate may make them more susceptible to chemical contaminants. Recent research has highlighted the interactive effects of chemical and nonchemical stressors on chemical uptake, metabolism, and organism survival. Assessments have revealed that the nature of the interaction between climate variables and chemical pollution will depend on estuarine species and life stage, duration and timing of exposure, prior stressor exposure, and contaminant class. A need for further research to elucidate mechanisms of toxicity under different abiotic conditions and to incorporate climate change factors into toxicity testing was identified. These efforts will improve environmental risk assessment of chemical contaminants and management capabilities under changing climate conditions [*Current Zoology* 61 (4): 641–652, 2015].

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1   Estuaries and Climate Change

Estuaries are unique habitats where freshwater inputs meet the sea. Estuaries provide critical ecosystem services including water filtration and nutrient recycling (Barbier et al., 2011), nursery habitat for commercially and recreationally important fish and shellfish species (Beck et al., 2003), and act as buffer against storm impacts to coastal communities (Barbier et al., 2011). A diversity of flora and fauna inhabit estuaries, some spending their entire lives in the estuary, some only as juveniles, and some just passing through (i.e. the anadramous fish such as salmon, smelt, shad, striped bass, and sturgeon migrate from seawater into freshwater to spawn). Animals with different life history stages may be differentially impacted by climate change, with some effects potentially occurring after they migrate from the estuary.

With changes in global climate, estuaries in North America are predicted to experience increased storm activity, storm surges and coastal flooding, sea-level rise, and increased runoff and sediment loading (Romero-Lankao et al., 2014). Such alterations may induce a wide range of effects on estuarine habitats, including increased water temperature, water current alteration, increased salinity, increased erosion, and alteration in freshwater runoff patterns (Grabemann et al., 2001; Knowles and Cayan, 2002). Additional changes to the estuarine environment may follow, including changes in flooding zones and brackish water boundaries, water residence times, and water quality (Pethick, 1993; Grabemann et al., 2001; Weiss et al., 2011). Consequences of increased atmospheric CO_2 include increases in oceanic CO_2 and acidification, hypoxia, and upwelling (Pörtner, 2001; Harley et al., 2006; Romero-Lankao et al., 2014). Estuarine systems are considered especially sensitive to climate change, as they are already subject to considerable anthropogenic stress, including land use changes, biological contamination (non-native species and pathogens), and chemical pollution (Kennish, 2002). Climate change effects may intensify the negative effects of anthropogenic stressors in estuaries (Romero-Lankao et al., 2014).

1.1   Water quality variability

Extensive ranges in water quality parameters already occur in estuaries worldwide, including large and regular fluctuations in temperature, salinity, oxygen, and pH (Cai and Wang, 1998; Kemp, 1989; Cai et al., 2011; Sunda and Cai, 2012). The National Estuarine Research Reserve System (NERRS) water quality monitoring

database (http://cdmo.baruch.sc.edu/) documents diel temperature, salinity, oxygen and pH ranges for estuaries throughout the U.S. Additional monitoring has demonstrated water quality ranges at specific sites, for example, continuous monitoring data (2007–2008) from Leadenwah Creek, SC, showed temperature ranged from 2.79–37.88°C and salinity ranged from 0–36.83‰. Monitoring from 2009–2012 in similar creeks within the Charleston, SC area, measured pH values from 7.16–8.31 and values as low as 6.9 have been measured in the water column (Ringwood and Keppler, 2002; NOAA Tidal Creek Monitoring Database 2013). Lower pH values have been documented in bottom water, reaching 6.48 and a pCO_2 value of 4.7 kPa in a Charleston, SC tidal creek (Cochran and Burnett, 1996). Tidal creek measures of dissolved oxygen ranged from 26.3%–128.2% air saturation, with an average of 73.4% from 2009–2012 (NOAA Tidal Creek Monitoring Database, 2013). Other studies have measured low oxygen levels in creeks throughout Charleston including 32% (Cochran and Burnett, 1996) and ~10% during early morning low tides (Lerberg et al., 2000).

A recent report by the National Estuarine Research Reserve system on Climate Sensitivity highlights the importance of researching and planning for multiple stressor impacts related to climate change in estuaries, such as non-point source pollution (Robinson et al., 2013[1]). While estuarine organisms are uniquely adapted to tolerate a wide range of water quality conditions, changes in these abiotic parameters can cause physiological stress and adaptations come with an energetic cost (Fernandes and Rantin, 1994; da Silva Rocha et al., 2005; Lannig et al., 2010). Chemical toxicity may provide additional stress and increase mortality rates. Alternatively, the environmental parameters themselves may not cause mortality, but they may modify the chemical toxicity. For example, membrane structure and fluidity is also known to change in response to temperature, which may increase permeability and chemical uptake (Quinn, 1988). Metabolic activity is known to be affected by temperature, which may impact an organism's ability to biotransform and eliminate xenobiotics. Climate-driven variations in water quality may impact the toxicity of pollutants, both because of altered chemical fate and transport, and as a consequence of changes in physiological response.

1.2 Effects of climate stressors on chemical exposures

Climate change will likely affect chemical use. Sea level rise, increased storm frequency and intensity, coastal flooding, and increased temperatures will yield increases in mosquito populations and vector-borne diseases (Khasnis and Nettleman, 2005), thus requiring greater mosquito-control pesticide use. Similarly, warmer and wetter conditions will exacerbate agricultural weed, fungal and insect pests, also requiring increased pesticide use (Kattwinkel et al., 2011). The spread of agricultural pests into higher latitudes will also serve to increase pesticide applications (Rosenzweig et al., 2001). The concentration and location of chemical contaminants present in the atmosphere and soils, such as metals and persistent organic pollutants, are also expected to increase (Hooper et al., 2013). The movement of these chemicals is predicted to be altered by climate change, through increased atmospheric deposition and surface run-off (Noyes et al., 2009; Kattwinkel et al., 2011).

2 Effects of Climate Stressors on Contaminant Toxicity

Chemical contaminants are one of the stressors that estuaries face, and as climate changes occur, we need to understand what the combined impacts will be on estuarine organism health.

2.1 Pesticides

Pesticide pollution in estuaries is an important issue because estuaries serve as sinks for chemical contaminants delivered by rivers from larger watersheds. Pesticides enter estuaries via runoff and drift from applications to crops, mosquito control, turf grass, and home lawns, and leaching from antifoulant paints. A number of studies have investigated the effects of climate stressors on the toxicity of pesticides in estuarine organisms. DeLorenzo et al. (2013) evaluated herbicide toxicity in a marine phytoplankton, *Dunaliella tertiolecta*, including interactive effects of increasing temperature and salinity. Four common photosystem II inhibiting herbicides (irgarol, diuron, atrazine, and ametryn) were tested in static 96 h algal bioassays, under four exposure scenarios: standard temperature and salinity (25°C, 20‰), standard temperature and elevated salinity (25°C,

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[1] Robinson P, Leight AK, Trueblood DD, Wood B, 2013. Climate sensitivity of the National Estuarine Research Reserve System Report to NOAA's Climate Program Office, pp79.

40‰), elevated temperature and standard salinity (35°C, 20‰), and elevated temperature and elevated salinity (35°C, 40‰). The endpoints assessed included algal cell density at 96 h, growth rate, chlorophyll *a* content, lipid content, and starch content. Herbicide effects on growth rate, cell density and starch content were more pronounced under elevated salinity and temperature conditions. The effect of the herbicides on cellular chlorophyll *a* and lipids generally was not altered by exposure conditions. It was suggested that herbicides may cause starch to be accumulated as a result of impaired photosynthesis, and that at higher temperatures and salinities there is an increase in the rate of chemical diffusion across the cell membrane, resulting in greater herbicide toxicity and greater starch content. These results indicate that changes in climate that result in warmer temperatures and increased salinities within estuarine ecosystems could increase the sensitivity of marine phytoplankton to pesticide pollution. Predicting climate change effects on estuarine phytoplankton will be further complicated by the likelihood for spatial expansion and increased frequency of harmful algal blooms with increased water temperatures and increased nutrient loading (Miller and Harding, 2007; Moore et al., 2008). There may also be shifts in phytoplankton community structure due to salinity and temperature tolerances of individual taxonomic groups (Quinlan and Phlips, 2007) and changes in microbial food web interactions (bacteria, zooplankton) with changing climate factors (Petchey et al., 1999; Sarmento et al., 2010). As primary producers contributing to the base of the estuarine food web, it will be important to investigate all aspects of climate change on phytoplankton species.

Similarly, interactive effects of climate and contaminant stress on aquatic macrophytes could have significant impacts on estuarine structure and function. Hall et al. (1997) studied salinity effects on atrazine toxicity using a species of submerged aquatic vegetation native to the Chesapeake Bay, Sago pondweed *Stuckenia pectinata*. They tested a range of salinities (1 to 12 ‰) and showed that atrazine was more toxic at the higher salinity based on plant dry weight. The multistressor effect of salinity and atrazine was thought to be due to decreased physiological functions, such as contaminant metabolism and detoxification processes, at higher salinities. This was supported by lower control growth rates at the higher salinity (Hall et al., 1997). Additional studies with the herbicide atrazine also found interactive effects of temperature, salinity, and herbicide toxicity. Hall et al. (1994) determined the influence of a range of

salinities on the acute toxicity of atrazine to nauplii of the copepod *Eurytemora affinis*. Decreasing atrazine toxicity was observed with increasing salinity for the copepod. Toxicity values (96h LC50) for the copepod were 0.5 mg/L, 2.6 mg/L, and 13.2 mg/L at 5 ‰, 15 ‰, and 25‰ salinity, respectively. A chronic (8 d) exposure with atrazine and *E. affinis* also showed toxicity to be altered by salinity, but the response was not linear (Hall et al., 1995a).

Atrazine exposure to two estuarine fish species demonstrated increased atrazine toxicity with increasing salinity. For larval sheepshead minnows *Cyprinodon variegatus*, 96 h LC50 values were 16.2 mg/L, 2.3 mg/L, and 2.0 mg/L at salinities of 5 ‰, 15 ‰, and 25 ‰, respectively (Hall et al., 1994). Fortin et al. (2008) studied the interactive effect of salinity and atrazine with the larval mummichog *Fundulus heteroclitus*. In the absence of atrazine, fish were able to maintain osmotic balance at high salinity (35 ‰), or low salinity (3‰), but atrazine exposure (5 µg/L) combined with salinity stress resulted in loss of osmoregulatory function (Fortin et al., 2008). Waring and Moore (2004) exposed Atlantic salmon *Salmo salar* smolts to atrazine in freshwater and then assessed survival after transfer to seawater (33 ‰). All fish without prior atrazine exposure survived transfer to seawater, but atrazine concentrations (2.0–22.7 µg/L) which were sublethal in freshwater caused acute mortality (14%–43%) within 24 h after the seawater transfer (Waring and Moore, 2004). The atrazine studies cited here demonstrate that the herbicide-salinity interaction will depend on exposure factors including timing, duration, acclimation, species, and life stage.

Greco et al. (2011) examined the influence of temperature on the toxicity of a mixture of three chlorophenoxy herbicides (2,4-D, dicamba, and mecoprop) in the estuarine soft shell clam, *Mya arenaria*. Increased temperature was found to inhibit antioxidant defenses (measured by malondialdehyde content, cytochrome C oxidase and superoxide dismutase activity) in herbicide exposed clams. Temperature enhanced effects were also seen on gametogenesis, hemocyte suppression, catalase and acetylcholinesterase activities (Greco et al., 2011).

The mixture toxicity of two pesticides, an organophosphate (OP), chlorpyrifos, and an organochlorine, DDT, were tested with an estuarine harpacticoid copepod *Microarthridion littorale* under various salinities (Staton et al., 2002). There was a significant interactive effect of pesticide and salinity on copepod survival after 24 h, but the response was not linear. The greatest pesti-

cide toxicity was observed at 3‰ salinity, with 4.05% survival, compared to 21.4% survival at 12‰ and 7.5% survival at 35‰ (Staton et al., 2002). Increased salinity also served to increase toxicity of an OP insecticide (dimethoate), a carbamate (aldicarb), and an insect growth regulator (tebufenozide) to brine shrimp (*Artemia* sp.), while toxicity of imidacloprid (a neonicotinoid insecticide) was not affected by salinity (Song and Brown, 1998).

The interactive effects of two pesticides (a synthetic pyrethroid, permethrin, and an insect growth regulator, methoprene) and salinity were examined in the fiddler crab *Uca pugnax* (Stueckle et al., 2009). Chronic, sublethal effects on limb regeneration and molting were measured. Salinity variation (both low of 10‰ and high of 40‰) was found to decrease the effective concentration of permethrin that caused abnormal limb regeneration in male crabs (Stueckle et al., 2009).

The effect of temperature and salinity on the toxicity of several pesticides was investigated using the grass shrimp *Palaemonetes pugio*. The insecticide Scourge® is a synthetic pyrethroid product with the active ingredient resmethrin and the synergist piperonyl butoxide, and is commonly used as a mosquito control agent. Chlorothalonil is a widely used chlorinated fungicide applied for agriculture, golf course maintenance, and lawn care. Larval and adult grass shrimp were exposed to Scourge® and chlorothalonil with standard test conditions of temperature 25°C and salinity 20‰, a 10°C increase in temperature, a 10‰ increase in salinity, and a combined increased temperature and salinity exposure (DeLorenzo et al., 2009). Both pesticides were found to be more toxic at the higher temperature than under standard testing conditions (DeLorenzo et al., 2009). It was speculated that since metabolic rate is increased at higher temperatures, the rate of water movement across the gills would increase and hence increase pesticide uptake. The effect of increasing salinity was not consistent across pesticides. Increased salinity increased chlorothalonil toxicity, but decreased Scourge® toxicity. Scourge® may become less bioavailable in higher salinity due to binding. The effect however, was limited to adult shrimp, indicating potential physiological difference between life stages. Combining increased salinity with increased temperature resulted in increased toxicity of both chlorothalonil and Scourge® to *P. pugio* (DeLo-

renzo et al., 2009). Breken-Folse et al. (1994) also examined the effect of temperature and salinity on pesticide toxicity in *P. pugio* and *C. varietagus*. The insecticides tested included 4-nitrophenol and 2,4-dinitro-phenol (used to manufacture drugs, pesticides, and dyes, and used as pesticides) and the OP insecticides terbufos, and trichlorfon. Increasing salinity decreased toxicity of the nitrophenols in both test organisms, except for 2,4-dinitrophenol toxicity to grass shrimp which had the opposite response. Increasing salinity increased toxicity of the OP compounds. Increasing temperature generally decreased toxicity of nitrophenols, except for 2,4-dinitrophenol toxicity to sheepshead minnow which had the opposite response. Increasing temperature increased the toxicity of the OP compounds (Breken-Folse et al., 1994).

A study with carbaryl, a carbamate compound used on agricultural crops and for home pest control, examined the effects of temperature on the toxicity in grass shrimp. Changes in survival and sublethal effects on activity of the nervous system enzyme, acetylcholinesterase, were found (Johnson and DeLorenzo, 2011[2]). In the 96h LC50 toxicity test, carbaryl concentrations of 25 µg/L and greater caused significant mortality at 25°C, whereas carbaryl concentrations of 12.5 µg/L and greater caused significant mortality at 32°C, indicating increased toxicity with increased temperature. There was no significant interactive effect of temperature on carbaryl inhibition of acetylcholinesterase activity (Johnson and DeLorenzo, 2011).

While pesticide toxicity generally increases with temperature, pyrethroid insecticide toxicity typically decreases with temperature (Di Giulio and Hinton, 2008). There are exceptions to this trend, however, and DDT, an organochlorine insecticide, has also been shown to be less toxic to estuarine organisms at higher temperatures (e.g blue crabs; Koenig et al., 1976). The differences in temperature effects are thought to be driven by biotransformation activity that can yield either more toxic or less toxic degradation products, depending on the pesticide (Harwood et al., 2009). Increased biotransformation rates at higher temperatures have been shown to yield less toxic pyrethroid degradation products, but in the case of chlorpyrifos, increased production of the more toxic oxon-analog occurs at higher temperatures (Harwood et al., 2009).

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[2] Johnson SE, DeLorenzo ME, 2011. Effects of increased temperature on pesticide toxicity in the grass shrimp *Palaemonetes pugio*. NOAA Office of Education, Science and Education Symposium, 2–4 August 2011, Silver Spring, MD.

Salinity has also been shown to affect pesticide biotransformation rates. Wang et al. (2001) compared hybrid striped bass *Morone saxatilis chrysops*, which tolerate salinities ranging from freshwater to full strength seawater, and rainbow trout responses to the carbamate insecticide aldicarb at 1.5, 7, 14, and 21‰ salinity. Aldicarb mortality was significantly greater at higher salinities for rainbow trout, but hybrid striped bass mortality was not affected by salinity. Cholinestase activity in the muscle, while not altered by increased salinity alone, decreased significantly with increasing salinity in aldicarb-exposed rainbow trout. Increasing salinity was shown to enhance expression of flavin-containing monooxygenase (FMO) in the rainbow trout, increasing bioactivation of aldicarb to the more potent cholinesterase inhibitor, aldicarb sulfoxide. Salinity did not induce FMO expression and subsequently alter aldicarb toxicity in hybrid striped bass, suggesting that salinity-mediated enhancement of aldicarb toxicity is species-dependent (Wang et al., 2001). Salinity modifications to pesticide toxicity have been shown to vary across compounds, even within a species. Katz (1961) tested 13 pesticides at 5‰ and 25‰ salinity with the three-spined stickleback and observed mostly minor changes in toxicity. Increasing salinity increased pesticide toxicity to the fish for aldrin (OC), dieldrin (OC), DDT (OC), methoxychlor (OC), guthion (OP), malathion (OP), CoRal (OP) and toxaphene (OC), decreased toxicity of endrin (OC), chlordane (OC), and lindane (OC), and had no effect on sevin (carbamate) and heptachlor (OC) (Katz, 1961).

Garcia et al. (2014) assessed the effects of low oxygen (hypoxia) and elevated CO_2 /low pH (hypercapnia) on pesticide toxicity in a common estuarine bivalve species, the hard clam *Mercenaria mercenaria* with the pyrethroid insecticide, resmethrin. Larval clams were exposed to a control (no resmethrin), 0.79 mg/L resmethrin (half of the LC50 value determined for larval clams under standard test conditions; approximately 70% air saturation or approximately 6 mg/L and a pH of 7.9–8.0), and 1.59 mg/L resmethrin (the LC50 value determined under standard test conditions) with and without oxygen and pH stress. Resmethrin toxicity was tested under hypoxia (defined as approximately 20% air saturation or approximately 2 mg/L), hypercapnia (defined as 1%–2% CO_2 or a pH of 6.9–7.0), and a mixture of hypoxia and hypercapnia. Resmethrin was more toxic to larval clams under hypoxia, hypercapnia and a combination of hypoxia and hypercapnia. Changes in clam filtration rates and metabolic processes under oxygen

and pH stress may have influenced resmethrin uptake, degradation, and subsequent pesticide toxicity (Garcia et al., 2014).

2.2 Metals

Estuaries often serve as sinks for metal pollution, as river sediments are deposited and metal concentrations can accumulate. Changing climate may alter the toxicity of metals to estuarine organisms since metal solubility and speciation state in seawater varies with abiotic parameters including salinity, temperature, oxygen, and pH.

Salinity alterations to metal toxicity have been observed with several estuarine organisms. Decreases in salinity tend to increase metal bioavailability and bioaccumulation (e.g. in the blue mussel *Mytilus*; Fischer, 1986). Toxicity of cadmium (Cd) to larval *C. variegatu*s, and copepod, *E. affinis*, nauplii was examined at salinities of 5, 15 and 25‰ (Hall et al., 1995b). Cd toxicity to *C. variegatu*s increased significantly with decreasing salinity, with a 96h LC50 value of 495.5 µg/L at 25‰, compared to 312.14 µg/L at 15‰ and 180.3 µg/L at 5‰ salinity. Cd toxicity to *E. affinis*, however, was not linearly affected by salinity. Toxicity values for *E. affinis* exposed to Cd were 82.9 µg/L, 213.2 µg/L, and 51.6 µg/L at salinities of 25, 15, and 5‰, respectively (Hall et al., 1995b). Salinity also affected Cd toxicity to *P. pugio*, with the direction of effect dependent on the original salinity regime of the shrimp collected (Oguma et al., 2013). Cd toxicity increased with increasing temperature and decreasing salinity in the fiddler crab, *Uca pugilator* (O'Hara, 1973). Cd was least toxic at 10°C and 30‰ (240 h LC50 of 47 mg/L), and most toxic at 30°C and10‰ (240 h LC50 of 2.9 mg/L). Temperature had a greater influence on Cd toxicity than salinity in the fiddler crab (O'Hara, 1973). Wright and Roosenburg (1982) noted a similar response of increased metal toxicity with increasing temperature and decreasing salinity with larval *U. pugilator* exposed to trimethyltin at temperatures of 10°C, 20°C and 28°C and salinities of 10‰, 20‰ and 30‰.

Temperature tends to increase metal uptake (Fritioff et al., 2005) and subsequent metal toxicity. The toxicity of Cd, Cu, Zn, and Pb to the juvenile crayfish *Orconectes immunis* was significantly increased under elevated temperature (Khan et al., 2006). A 3–4 degree change in temperature caused three-fold increases in metal-induced oxygen consumption (Khan et al., 2006). Increasing temperature from 15°C to 20°C increased metal uptake in the Mediterranean fish *Solea senegalensis* and altered metallothionein levels and metal par-

titioning into liver and kidney (Siscar et al., 2014). Ectotherms have been shown to have reduced aerobic metabolism capacity at elevated temperatures (Pörtner, 2001), and oxygen limited effects have been demonstrated with cadmium and the eastern oyster *Crassostrea virginica* (Lannig et al., 2006; Lannig et al., 2008). Increasing temperature from 20°C to 28°C significantly increased cadmium toxicity as exhibited by increased oyster mortality and lipid peroxidation, and decreased condition index (Lannig et al., 2006). Hypoxemia occurred more rapidly in Cd-exposed oysters at high temperature (Lannig et al., 2008). In addition, metal detoxification mechanisms (glutathione levels and rate of protein synthesis) decreased at the higher temperature, reducing capacity for metal metabolism (Lannig et al., 2006). Based on these data, environmental warming will increase the toxicological risk of metals to estuarine species.

Areas of hypoxic and anoxic conditions or "dead zones" are generally projected to increase with increasing seawater temperatures (IPCC, 2014) and have also been associated with increased metal toxicity. For example, oysters were not able to maintain energetic balance, as exhibited by depletion of ATP, decreased adenylate energy, and increased ADP/ATP ratios, under the combined stress of anoxia and cadmium exposure (Ivanina et al., 2010). Acclimation to moderate hypoxia, however, was associated with decreased manganese (Mn) uptake in silver catfish and reduced Mn toxicity (Dolci et al., 2014).

The effect of increased CO_2 and subsequent decreased pH generally serves to increase metal bioavailability and toxicity to aquatic organisms. At lower pH, metals bound in sediments are also released into free ions, increasing bioavailability to sediment-dwelling species (Ardelan et al., 2009). Metals solubility increases and the metallic ion becomes the dominant species at lower pH, although speciation is complicated in seawater, where cations can form organic complexes that decrease metal toxicity (Ardelan et al., 2009; Millero et al., 2009). A study with the hard clam *Mercenaria mercenaria* found hypercapnia (decreased pH due to elevated PCO_2) modifications to metal toxicity were metal-specific, such that cadmium uptake decreased while copper uptake increased under hypercapnic conditions (Ivanina et al., 2013). Pascal et al. (2010) also found increased CO_2/decreased pH resulted in increased Cu and Cd toxicity in the meiobenthic copepod, *Amphiascoides atopus*. Toxicity values (96 h LC50) for Cu were 0.65 mg/L at pH 8.2, 0.32 mg/L at pH 6.37, and 0.26

mg/L at pH 6.23; and 96-h LC50 values for Cd were 1.38 mg/L at pH 8.2, 1.41 mg/L at pH 6.30, and 0.98 mg/L at pH 6.23 (Pascal et al., 2010).

Low pH was shown to be protective against negative effects of Cu and Cd on bivalve mitochondrial respiration in *M. mercenaria* and *C. virginica* because the rate of proton leak was slower at low pH, thus compensating for the Cd-induced inhibition to respiration (Ivanina and Sokolova, 2013). The interactive effects of increasing CO_2 concentration/decreasing pH and increasing temperature on metal bioaccumulation were examined in the cuttlefish, *Sepia officinalis* (Lacoue-Labarthe et al., 2009). Changes in temperature and pH had effects on osmoregulation and egg permeability of this cephalopod that altered metal accumulation for Ag (greater uptake at lower pH and increased effect at lower temperature), Cd (greater uptake at higher pH and not affected by temperature), and Zn (uptake not linear with pH) (Lacoue-Labarthe et al., 2009).

2.3 Oil, Hydrocarbons, and Oil Spill Dispersants

Oil spills, shipping activity, marinas, municipal and industrial effluents, and other anthropogenic activity release a variety of hydrocarbon compounds into the estuarine environment. Chemical dispersants applied to mitigate off shore oil spills may also contaminate the estuarine environment. Zanette et al. (2011) studied the influence of salinity (35, 25, 15 and 9‰) on diesel oil toxicity in the oyster, *Crassostrea gigas*. Exposure to 1 ppm diesel oil at 35‰ salinity resulted in 53% oyster mortality, whereas no mortality was seen at any of the lower salinities. Salinity alone did not significantly affect glutathione S-transferase activity (GST), but biomarker activity in diesel-exposed oysters was affected by salinity. At 25‰ salinity, but not at the other salinity levels, diesel oil exposure caused a significant increase in GST activity. Lipid peroxidation activity was affected by salinity alone, with highest activity observed at 25‰ in control oysters. Lipid peroxidation activity increased in diesel exposed oysters at 35, 15 and 9‰, but not at 25‰ salinity (Zanette et al., 2011). Similarly, diesel oil caused greater toxicity to the brackish water crustacean *Gammarus* spp. at salinities lower and higher than iso-osmotic conditions (Tedengren et al., 1988). Lower salinity increased toxicity of diesel oil to the marine bivalve *Mytilus edulis*, as measured by negative effects on respiration and ammonia excretion (Tedengren and Kautsky, 1987). Salinity (ranging from 6.9 to 28.6‰) did not alter bioaccumulation or toxicity of fluoranthene in the estuarine grass shrimp *P. pugio* (Weinstein, 2003). Increasing salinity, did however, disrupt osmoregulation

and increase fluoranthene toxicity to the tubificid oligochaete worm *Monopylephorus rubroniveus* (Weinstein, 2003). Salinity has also been shown to affect oil dispersant toxicity (National Research Council, 2005). Oil dispersant efficacy and oil degradation rates were reduced at lower salinity (Kuhl et al., 2013). Toxicity of the oil dispersant Corexit and Corexit-dispersed Sweet Louisiana crude oil to the gulf killifish *Fundulus grandis* was significantly greater at 4‰ than at 12 or 18‰, but decreasing salinity did not alter toxicity of crude oil alone (Kuhl et al., 2013).

Interactions between temperature and benzo[a] pyrene (BAP) were observed with the mussel *Mytilus galloprovincialis* (Kamel et al., 2012). Increasing temperature was correlated with a decrease in detoxification enzyme activity and an increase in BAP accumulation and toxicity (Kamel et al., 2012). Linden et al. (1980) examined effects of water accommodated No. 2 fuel oil on development in *F. heteroclitus*. Oil exposure decreased yolk utilization, and the effect was intensified under increased temperature (Linden et al., 1980). Increasing temperature has been shown to increase oil dispersant toxicity (National Research Council, 2005). The dispersant Corexit 9500 was more toxic to the scallop *Argopecten irradians* at higher temperature, with 6 h LC50 values decreasing from 2500 at 2°C to 200 at 20°C. Similarly, grass shrimp LC50 values for Corexit decreased from 840 at 17°C to 640 at 27°C (National Research Council, 2005).

Ye and Zou (2008) examined the effect of hypoxia on naphthalene toxicity in the brown shrimp, *Penaeus aztecus*, and reported no significant effect of hypoxia on naphthalene bioaccumulation in either the gills or the hepatopancreas. The lack of effect was attributed to a potential compensatory mechanism of increased metabolic depuration of naphthalene under hypoxic conditions (Ye and Zou, 2008). Hypoxia alone has been linked to endocrine disruption in aquatic species (Hooper et al., 2013). Kraemer and Schulte (2004) found a significant interaction between hypoxia and PCB exposure in *F. heteroclitus*. Prior exposure to PCBs increased fish susceptibility to hypoxia by inhibiting anaerobic glycolysis, which the fish rely on for metabolism during periods of environmental hypoxia.

3   Conclusions, Data Gaps and Future Research Needs

Understanding how climate alters chemical contaminant distribution, fate and effects will be critical to achieving valid environmental risk assessments. Overall,

most studies have shown that increasing temperature causes increased toxicity for contaminants, whereas salinity effects were more variable with type of contaminant and species studied (Table 1). The predictions for climate change effects on salinity patterns also are more variable, since increases may occur with saltwater intrusion, but decreases may occur in areas of increased river discharge. Fewer data were available for the effects of hypoxia and hypercapnia on contaminant toxicity, but trends toward increased toxicity were seen with several compounds. More information was available for estuarine species and modulating effects on pesticide toxicity than was available for metal toxicity, particularly for pH effects and estuarine fish species. Additional research is needed on the interactive effects of multiple abiotic stressors (for example, temperature and salinity; pH and oxygen) on contaminant toxicity to encompass the complex climate modifications anticipated.

Estuaries are dynamic ecosystems with populations of organisms specially adapted to fluctuations in temperature, salinity, oxygen and pH. Climate change may select for more tolerant individuals in estuarine populations. It is possible that future populations of estuarine species could gradually adapt to longer durations of elevated temperatures, higher or lower salinities, hypoxia and acidification. Alternatively, the cumulative stress of abiotic extremes and pollution may overwhelm adaptive capacity of estuarine species, leading to reduced fitness and population growth. The rate of change and potential for acclimation is an aspect of global climate change impacts on organism health that should be addressed in future studies.

While some mechanisms of climate-factor mediation have been identified, more research is needed to elucidate biochemical and physiological pathways associated with changes in toxicity. Hooper et al. (2013) prescribe use of adverse outcome pathways, a predictive approach to characterizing mechanisms of effect for mixtures of chemicals and interactions between chemical and nonchemical stressors. Most studies have examined short-term impacts of climate stress on contaminant toxicity. Future research should include chronic exposures to improve our understanding of the effects of climate-mediated toxicity on population-level endpoints such as growth and reproduction. The impact of climate stress on mixtures of contaminants is another needed area of study.

Chemical risk assessments are typically performed using toxicity data from standardized tests conducted at uniform water quality parameters. The research findings

discussed herein establish that changes in temperature, salinity, oxygen, and pH alter the toxicity of certain environmental pollutants, and that the nature of the effect will depend on the specific species, life stage, and chemical contaminant. Standard toxicity bioassays may not be predictive of actual contaminant toxicity under

Table 1 Trends in effect of temperature and salinity on chemical contaminant toxicity, summarized by taxonomic group

Taxonomic Group	Chemical (Class)	Toxicity Change with Increasing Temp.	Reference	Chemical Type	Toxicity Change with Increasing Salinity	Reference
Algae	irgarol, atrazine, diuron, ametryn (herbicides)	∧	DeLorenzo et al., 2013.	irgarol (herbicide)	∧	DeLorenzo et al., 2013.
Macrophytes				atrazine (herbicide)	∧	Hall et al., 1997.
Molluscs	2,4-D, dicamba, mecoprop (herbicides)	∧	Greco et al., 2011.			
	Cd (metal)	∧	Lannig et al., 2006.	Cd (metal)	∨	Fischer, 1986.
	benzo[a]pyrene (PAH)	∧	Kamel et al., 2012.	diesel oil (PAHs)	∨	Tedengren and Kautsky, 1987.
	Corexit 9500 (oil dispersant)	∧	National Research Council, 2005.	diesel oil (PAHs)	∧	Zanette et al., 2011.
Crustaceans				atrazine (herbicide)	∨	Hall et al., 1994.
	DDT (OC)	∨	Koenig et al., 1976.	DDT (OC)	∨	Staton et al., 2002.
				dimethoate (OP)	∧	Song and Brown, 1998.
	terbufos, trichlorfon (OPs)	∧	Breken-Folse et al., 1994.	terbufos, trichlorfon (OPs)	∧	Breken-Folse et al., 1994.
	carbaryl (carbamate)	∧	Johnson and DeLorenzo, 2011.	aldicarb (carbamate)	∧	Song and Brown, 1998.
				tebufenozide (IGR)	∧	Song and Brown, 1998.
				methoprene (IGR)	--	Stueckle et al., 2009.
				imidacloprid (neonicotinoid)	--	Song and Brown, 1998.
	resmethrin (pyrethroid)	∨	DeLorenzo et al., 2009)	permethrin (pyrethroid)	∧	Stueckle et al., 2009.
	chlorothalonil (fungicide)	∧	DeLorenzo et al., 2009)	chlorothalonil (fungicide)	∧	DeLorenzo et al., 2009.
	4-nitrophenol (nitrophenol)	∨	Breken-Folse et al., 1994)	4-nitrophenol & 2,4-nitrophenol (nitrophenols)	∨	Breken-Folse et al., 1994.
	Cd, Cu, Zn, Pb (metals)	∧	Khan et al., 2006)	Cd (metal)	∨	Hall et al., 1995b.
	Cd (metal)	∧	O'Hara (1973)	Cd (metal)	∨	O'Hara, 1973.
	tributyltin (metal)	∧	Wright and Roosenburg, 1982.	tributyltin (metal)	v	Wright and Roosenburg, 1982.
	Corexit 9500 (oil dispersant)	∧	National Research Council, 2005.	diesel oil (PAHs)	∧	Tedengren et al., 1988.
Fish				atrazine (herbicide)	∧	Hall et al., 1994. Fortin et al., 2008, Waring & Moore. 2004.
	terbufos, and trichlorfon (OPs)	∧	Breken-Folse et al., 1994.	terbufos, and trichlorfon (OPs)	∧	Breken-Folse et al., 1994.
				guthion, malathion, CoRal (OPs)	∧	Katz, 1961.
				aldicarb (carbamate)	∧	Wang et al., 2001.
	DDT (OC)	∨	Harwood et al., 2009.	aldrin, dieldrin, DDT, methoxychlor, toxaphene (OCs)	∧	Katz, 1961.
				endrin, chlordane, lindane (OCs)	∨	Katz, 1961.
	4-nitrophenol (nitrophenol)	∨	Breken-Folse et al., 1994.	4-nitrophenol & 2,4-nitrophenol (nitrophenols)	∨	Breken-Folse et al., 1994.
	Cr, Cu, Co, Fe (metals)	∧	Siscar et al., 2014.	Cd (metal)	∨	Hall et al., 1995b.
	No. 2 Fuel Oil (PAHs)	∧	Linden et al., 1980.	Corexit 9500 (oil dispersant)	∨	Kuhl et al., 2013.

variable environmental conditions, for example, pesticide risk assessments for tropical regions rely on toxicity testing with temperate species (Daam and Van den Brink, 2010). Testing environmental contaminants under a wider range of exposure conditions could improve the accuracy of chemical risk assessments.

When addressing abiotic influences in toxicity testing, one must consider several issues. Experimental design challenges include selecting appropriate levels of abiotic stressors, whether to pre-acclimate test organisms to abiotic stressor conditions, pulsed vs. continuous exposures, and proper statistical approach to multifactor data. Long-term monitoring databases may provide useful information on seasonal and decadal ranges in water quality parameters.

Global climate change impacts are complex and far-reaching. Effects on contaminant toxicity are one concern for estuarine organisms, but they may also be challenged by changes in nutrient status, habitat loss, and increases in bacterial and viral pathogens and parasites (Fleming et al., 2006). Nutrient/fertilizer pollution represents an important co-factor to consider because it tends to co-occur with chemical contaminants in agricultural and urban storm water runoff (Hapeman et al., 2002). Since both fertilizer and pesticide applications are predicted to increase with global climate change (Kattwinkel et al., 2011), estuarine organisms will experience the combined effect of nutrient eutrophication (hypoxia, altered trophic states, decreased water clarity) and chemical toxicity. In addition, global warming is predicted to increase upwelling of nutrients into coastal waters (Harley et al., 2006) where nutrient and chemical pollution will mix. The outcome of such complex interactions will be difficult to project. For example, nutrient pollution may cause excessive algal growth which may lead to decreased UV light penetration, which may decrease degradation rates of some chemical contaminants while ameliorating photo-activated toxicity of others. Assessing such multi-stressor impacts is critical to understanding potential ecological consequences. For example, Faxneld et al. (2010) demonstrated synergistic effects of elevated temperature, low salinity, and elevated nitrogen on corals. The negative impacts included a decrease in gross primary production/respiration ratio. In our changing climate scenario, it would be valuable to test a fourth stressor, herbicide exposure, to determine the full extent of toxicity under altered climate, and nutrient eutrophication.

The resilience of estuarine organisms to environmental change will determine the future health of our coastal ecosystems. Improved strategies for pollution reduction and habitat protection may help mitigate multi-stressor effects. By expanding ecotoxicity testing conditions, we may be better poised to protect sensitive estuarine ecosystems; both now and in the face of changing global climate.

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