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Review article

The toxicology of climate change: Environmental contaminants in a warming world

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ABSTRACT

Climate change induced by anthropogenic warming of the earth's atmosphere is a daunting problem. This review examines one of the consequences of climate change that has only recently attracted attention: namely, the effects of climate change on the environmental distribution and toxicity of chemical pollutants. A review was undertaken of the scientific literature (original research articles, reviews, government and intergovernmental reports) focusing on the interactions of toxicants with the environmental parameters, temperature, precipitation, and salinity, as altered by climate change. Three broad classes of chemical toxicants of global significance were the focus: air pollutants, persistent organic pollutants (POPs), including some organochlorine pesticides, and other classes of pesticides. Generally, increases in temperature will enhance the toxicity of contaminants and increase concentrations of tropospheric ozone regionally, but will also likely increase rates of chemical degradation. While further research is needed, climate change coupled with air pollutant exposures may have potentially serious adverse consequences for human health in urban and polluted regions. Climate change producing alterations in: food webs, lipid dynamics, ice and snow melt, and organic carbon cycling could result in increased POP levels in water, soil, and biota. There is also compelling evidence that increasing temperatures could be deleterious to pollutant-exposed wildlife. For example, elevated water temperatures may alter the biotransformation of contaminants to more bioactive metabolites and impair homeostasis. The complex interactions between climate change and pollutants may be particularly problematic for species living at the edge of their physiological tolerance range where acclimation capacity may be limited. In addition to temperature increases, regional precipitation patterns are projected to be altered with climate change. Regions subject to decreases in precipitation may experience enhanced volatilization of POPs and pesticides to the atmosphere. Reduced precipitation will also increase air pollution in urbanized regions resulting in negative health effects, which may be exacerbated by temperature increases. Regions subject to increased precipitation will have lower levels of air pollution, but will likely experience enhanced surface deposition of airborne POPs and increased run-off of pesticides. Moreover, increases in the intensity and frequency of storm events linked to climate change could lead to more severe episodes of chemical contamination of water bodies and surrounding watersheds. Changes in salinity may affect aquatic organisms as an independent stressor as well as by altering the bioavailability and in some instances increasing the toxicity of chemicals. A paramount issue will be to identify species and populations especially vulnerable to climate–pollutant interactions, in the context of the many other physical, chemical, and biological stressors that will be altered with climate change. Moreover, it will be important to predict tipping points that might trigger or accelerate synergistic interactions between climate change and contaminant exposures.

Contents

Abbreviations: CO, Carbon Monoxide; CO_2, Carbon Dioxide; DDT, Dichlorodiphenyltrichloroethane; FMO, Flavin-Containing Monooxygenases; HCB, Hexachlorobenzene; HCH, Hexachlorocyclohexane; HLC, Henry's Law Constant; IPCC, Intergovernmental Panel on Climate Change; NOx, Nitrogen Oxides; OP, Organophosphate; PCP, Pentachlorophenol; PBT, Persistent, Bioaccumulative, and Toxic; PCB, Polychlorinated Biphenyl; PM, Particulate Matter; POP, Persistent Organic Pollutant; SO_2, Sulfur Dioxide; VTG, Vitellogenin; VOC, Volatile Organic Compound.

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1. Introduction

Climate change is an increasingly urgent problem with potentially far-reaching consequences for life on earth. Humans and wildlife are also exposed to an array of chemical, physical, and biological stressors that arise largely from anthropogenic activity, but also from natural sources. One of the consequences of climate change that has recently attracted attention is its potential to alter the environmental distribution and biological effects of chemical toxicants. There is growing awareness of the importance of anticipating the effects of chemical pollution in the rapidly changing environment, and identifying and mitigating effects in those humans and ecosystems most vulnerable.

The U.N. Intergovernmental Panel on Climate Change (IPCC) has completed four assessments covering the evidence, impacts, and mitigation of climate change (IPCC, 2007a,b,c,d,e). They report unequivocal global warming with evidence of increases in global mean air and ocean temperatures, widespread snow and ice melt, and rising global sea level. Temperature is projected to increase 1.8–4.0 °C by the end of the century under a range of probable greenhouse gas emission scenarios with the greatest warming expected at high latitudes. In addition to global warming, some regions, such as North and South America, northern Europe, and northern and central Asia are projected to experience increased precipitation, while others, including southern Africa and Asia and the Mediterranean, are expected to experience substantial droughts. Heat waves, precipitation and storm events are predicted to be more frequent and intense. Oceanic acidification linked to increasing atmospheric carbon dioxide levels is a growing threat to marine organisms and ecosystems.

This article examines how the environmental parameters, temperature, precipitation, and salinity, as altered by climate change, could affect the environmental distribution and biological effects of chemical toxicants. It is intended to provide a broad perspective on the interactions of climate change and chemical behavior/toxicity based on available research, which in some cases continues to be limited. For example, key aspects of climate change and pollutant interactions that merit further study involve describing effects on vulnerable species and populations and revealing the nature of thresholds that might trigger adverse events. While climate change will affect the environmental distribution and toxicity of numerous chemical toxicants, we focus primarily on three major classes of global significance: air pollutants, persistent organic pollutants (POPs), and other pesticides. Air pollution is a global problem, and here we focus on two compounds, tropospheric ozone and particulate matter (PM), as they are potent toxicants of human health concern. POPs are persistent, bioaccumulative, and toxic (PBT) contaminants found ubiquitously in the environment, humans, and wildlife. At present, twelve chlorinated organic chemicals are listed as POPs under the U.N. Stockholm Convention, including several organochlorine pesticides, such as dichlorodiphenyltrichloroethane (DDT) and toxaphene, as well as the polychlorinated biphenyls (PCBs), dioxins, and furans (UNEP, 2005). Other pesticides, such as atrazine, aldicarb, and chlorpyrifos are of special interest as they are applied in large quantities over a broad area and have a range of toxicological effects. Moreover, pesticide use patterns may change as agriculture and pest species shift in response to climate change.

2. Effects of climate change on contaminant environmental fate and behavior

Climate change will have a powerful effect on the environmental fate and behavior of chemical toxicants by altering physical, chemical, and biological drivers of partitioning between the atmosphere, water, soil/sediment, and biota, including: air-surface exchange, wet/dry deposition, and reaction rates (e.g., photolysis, biodegradation, oxidation in air). Temperature and precipitation, as altered by climate change, are expected to have the largest influence on the partitioning of chemical toxicants. In addition, an array of important processes, such as snow and ice melt, biota lipid dynamics, and organic carbon cycling, will be altered by climate change potentially producing significant increases in fugacity (thermodynamic measure of substance tendency to prefer one phase over another) and contaminant concentrations (MacDonald et al., 2002).

2.1. Altered fate and behavior of air pollutants

It is widely recognized that air quality and climate change are strongly interconnected (IPCC, 2007c,e). Climate change is projected to generally degrade air quality, but for tropospheric ozone and PM, there continues to be uncertainty as to the direction and magnitude of changes in environmental distribution patterns (Aw and Kleeman, 2003; Ebi et al., 2006; IPCC, 2007c,e; Racherla and Adams, 2006).

Tropospheric ozone is generally short-lived and forms in the lower atmosphere from the nitrogen oxide (NOx)-dependent photochemical oxidation of volatile organic compounds (VOCs), carbon monoxide (CO), and sulfur dioxide (SO_2) (Forster et al., 2007). Ozone levels are dictated by emissions of ozone precursors, temperature, water vapor levels, atmospheric circulation patterns, and stratospheric inputs (Denman et al., 2007; Forster et al., 2007; Stevenson et al., 2006). Elevated temperatures generally lead to increased formation of ozone, while increased water vapor generally leads to increased breakdown of ozone. As such, climate change impacts on regional ozone levels will largely be determined by the extent to which temperature, water vapor levels, and air circulation patterns are altered. The interplay of these factors is depicted in Fig. 1, Legend Item A.

While ozone concentrations are projected to increase for many regions, climate change, on a global scale, is expected to generally accelerate tropospheric ozone destruction due to catalyzed photodegradation in the

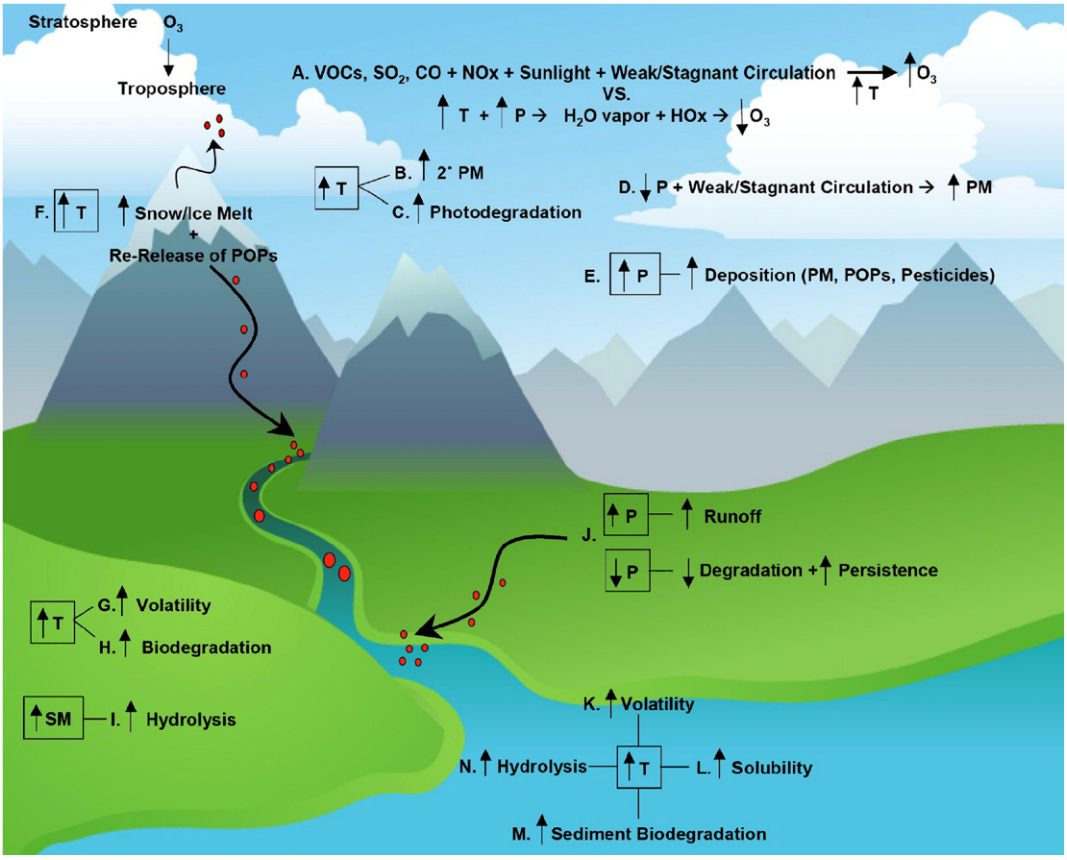

Fig. 1. Effects of climate change effects on the environmental distribution of contaminants. Figure legend: A. Temperature increases coupled with ozone precursors, sunlight, and weak/stagnant circulation will increase the rate of formation of ozone, whereas water vapor will increase ozone destruction. B. Temperature increases may promote the formation of secondary PM. C. Temperature increases may lead to increased photodegradation of POPs. D. Climate change producing increases in precipitation will increase the wet deposition of PM, POPs, and pesticides. E. Declining precipitation coupled with weak/stagnant circulation may increase PM regionally. F. Melting snow, ice, and glaciers will release and remobilize POPs sequestered in these once frozen matrices. G. Temperature increases will enhance the volatility of POPs and other pesticides from soils to the atmosphere. H. Temperature increases will enhance microbial degradation of POPs and pesticides in soils and sediments. I. Enhanced soil moisture will increase hydrolytic degradation of pesticides, but may not affect POPs since they are relatively resistant to hydrolysis. J. Precipitation increases will enhance the potential for pesticide and POP runoff into aquatic system, whereas decreases in precipitation will ameliorate chemical runoff but may increase persistence. K. Water temperature increases will enhance the volatility of POPs and other pesticides from water to the atmosphere. L. Water temperature increases will increase the solubility of POPs making them more apt to be retained in water. M. Water temperature increases will enhance microbial activity increasing the degradation of POPs and pesticides in soils and sediments. N. Water temperature increases will increase the hydrolysis of pesticides to less or more bioactive degradates. 2°PM = Secondary PM; CO = Carbon monoxide; HO*x* = HO_2 + OH; NO*x* = Nitrogen oxides; O_3 = Ozone; P = Precipitation; PM = Particulate Matter; POP = Persistent Organic Pollutant; SM = Soil Moisture; T = Temperature; VOCs = Volatile organic compounds.

presence of increased atmospheric water vapor. For example, Racherla and Adams (2006) project a 5% decline in *global* tropospheric ozone concentrations in the 2050s from 1990s levels using present day pollutant emission scenarios. Dentener et al. (2006) and Stevenson et al. (2006) estimated future ozone concentrations for 2030 based on current levels of emissions. They calculated that climate change could reduce *global* ozone by 0.5–1.0 ppb over the continents and 1–2 ppb over the oceans.

However, despite estimates of net global declines, several studies project regional scale increases in ozone pollution linked to climate change (Aw and Kleeman, 2003; Cheng et al., 2007; Hogrefe et al., 2004; Langner et al., 2005; Stevenson et al., 2006). For example, tropospheric ozone concentrations are predicted to increase in Southern California as a result of accelerated gas phase reaction rates associated with rising temperatures (Aw and Kleeman, 2003). Similarly, using the IPCC A2 high CO_2 emission scenario, Hogrefe et al. (2004) estimate increases in summertime average daily maximum 8-hour ozone concentrations over the eastern U.S. of 2.7 ppb by the 2020s, 4.2 ppb by the 2050s, and 5.0 ppb by the 2080s.

Models of the New York metropolitan area have been used to estimate average summertime ozone increases from 0.3 ppb in the 1990s to 4.3 ppb by the 2050s (Knowlton et al., 2004). Cheng et al. (2007) modeled future concentrations of various air pollutants (ozone, NO*x*, SO_2, and suspended particulates) in four south-central Canadian cities (Montreal, Ottawa, Toronto, Windsor) using end of 20th century emission scenarios. They found that a warming climate would increase the number of days in the high ozone category (concentrations ≥81 ppb) by 40–100% by the 2050s and 70–200% by the 2080s, from the current average of eight days. In Europe, increases in tropospheric ozone are projected over central and southern regions predicted to experience precipitation declines (Langner et al., 2005). In contrast, ozone decreases are projected to occur over northern Europe due to increased precipitation.

Climate change-induced shifts in precipitation patterns will also affect PM fate and behavior (Aw and Kleeman, 2003; Forster et al., 2007; Racherla and Adams, 2006). PM consists of both natural and anthropogenic sources of soils, dusts, acids, organic chemicals, and metals. It enters the atmosphere through direct emissions or is formed as secondary particles through atmospheric chemical reactions (Forster et al., 2007). Much of the research on PM fate and behavior focuses on PM_{10} (particles with an aerodynamic diameter ≤10 μm) and more recently on $PM_{2.5}$ (fine particles with an aerodynamic diameter of ≤2.5 μm) as these particle sizes are inhalable and have been shown to be potent toxicants (Forster et al., 2007; USEPA, 2004).

Decreased concentrations of atmospheric fine PM are projected in regions that experience increases in precipitation due to enhanced scavenging of PM by water molecules. Racherla and Adams (2006) estimate that increases in precipitation and wet deposition loss rates could decrease the global burdens and atmospheric residence times of $PM_{2.5}$ by 2–18% by the 2050s (Fig. 1, Legend Item E). However, changes in

other climate variables may also affect PM concentrations. Global warming could increase the formation of secondary PM by catalyzing *in situ* gas phase reactions (Fig. 1, Legend Item B). Aw and Kleeman (2003) modeling of PM interactions with climate change in the Southern California region indicate that non-volatile secondary PM may increase with rising temperatures, but that semi-volatile secondary PM could increase or decrease.

PM concentrations are highly affected by regional emissions, and atmospheric transport of these pollutants can be driven by synoptic-scale (i.e., low or high pressure systems of the lower atmosphere that range on the order of 1000 to 2500 km) weather patterns. For example, Buchanan et al. (2002) investigated the influence of regional weather patterns on PM_{10} concentrations in Edinburgh, Scotland and demonstrated that PM can move well beyond its point source due to these large area dynamics. As climate change is predicted to affect synoptic-scale weather patterns, regional distribution of air pollutants may be affected. In contrast, increases in the frequency of stagnant air events in polluted urban, rural, or industrial settings could enhance the intensity of air pollution (Fig. 1, Legend Item D) (Denman et al., 2007).

2.2. Altered fate and behavior of POPs

Climate change will influence the environmental fate and behavior of POPs by altering the fundamental mechanisms of solvent switching and solvent depletion, and by enhancing contaminant degradation (Brubaker and Hites, 1998; Ma et al., 2004; MacDonald et al., 2002; Meyer and Wania, 2008; Sinkkonen and Paasivirta, 2000; Sweetman et al., 2005; Wania, 1999). Solvent switching involves contaminants partitioning into different chemical phases (solid, liquid, gas) in the direction of thermodynamic equilibrium. While this process can increase concentrations of a contaminant in an environmental compartment (water, sediment, biota, etc.), it cannot produce contaminant concentrations that exceed the thermodynamic equilibrium (MacDonald et al., 2002; Wania, 1999). The effects of global warming on solvent switching can be predicted by considering temperature-driven changes in partitioning constants of POPs, such as Henry's Law Constants (HLC).

In contrast to solvent switching, solvent depletion is a complex process that requires energy and increases fugacity and often contaminant concentrations as solvent concentrations continually decline. Thus, contaminant concentrations in a given environmental compartment can exceed the thermodynamic equilibrium (MacDonald et al., 2002). Examples of solvent depletion processes that may be influenced by climate change include contaminant biomagnification, trophic structure alterations, hydrological processes, and organic carbon cycling. Many transport processes and spatial and temporal variables can influence solvent depletion processes making them difficult to predict (Macdonald et al., 2003; Wania, 1999).

Enhanced volatility and partitioning of POPs to the atmosphere by solvent switching is likely with global warming, as are increases in the rate of contaminant degradation. The warming climate may produce a minor reduction in POP exposure to aquatic biota because of enhanced partitioning from water to the atmosphere as contaminant HLCs rise with increasing water temperatures (Ma et al., 2004; Macdonald et al., 2003). Supporting this hypothesis, elevated air temperatures from 1990–2000 linked to fluctuations of the North Atlantic Oscillation, El Niño-Southern Oscillation, and Pacific North American patterns increased the volatility and atmospheric concentrations of the POPs, hexachlorobenzene (HCB), and PCBs, in the Great Lakes Region, USA (Ma et al., 2004).

Observed temperature increases due to climate change are most pronounced at higher latitudes. The IPCC reports that average arctic temperatures have increased at nearly twice the global average rate in the past 100 years (IPCC, 2007e). POPs are unique in that they can move thousands of miles from their point of release and are often observed at higher latitudes. This observation is explained by the concept of global fractionation (Braune et al., 2005; Breivik et al., 2004; Wania and Mackay, 1996). Most POPs are semi-volatile enough to evaporate at temperate or tropical latitudes, existing as gases or adsorbed to aerosols in the atmosphere. Global atmospheric circulation transports these air masses, containing POPs, to higher latitudes in short jumps coinciding with the seasons. As temperature gradients between high and low latitudes become less pronounced, the temperature-induced global fractionation of POPs to high latitudes could decline (Beyer et al., 2003).

In addition, a decline in atmospheric partitioning and transport of POPs to the poles may result from temperature- and precipitation-accelerated increases in degradation, particularly in the atmosphere and soil (Dalla Valle et al., 2007; Macdonald et al., 2005; Sinkkonen and Paasivirta, 2000; Sweetman et al., 2005; Wania and Mackay, 1996). Dalla Valle et al. (2007) predict that increasing temperatures in Venice Lagoon, Italy will accelerate the degradation of PCB 118 and PCB 180 congeners, 2,3,7,8-tetrachlorodibenzofuran, and 1,2,3,4,7,8-hexachlorodibenzofuran in most environmental compartments. However, these authors note that while elevated temperatures are expected to decrease the fugacity capacity (i.e., indicator of compartment capacity to store a chemical) of most environmental compartments, the fugacity capacity of the air compartment is projected to decline only negligibly. Thus, enhanced atmospheric mobility and long-range transport is predicted in this study. Global warming, however, may also accelerate atmospheric photodegradation of POPs, counter-balancing this atmospheric partitioning (Brubaker and Hites, 1998; Sinkkonen and Paasivirta, 2000).

While atmospheric partitioning of POPs and enhanced degradation are generally predicted with climate change, regional patterns of increased precipitation and ice/snow melt are expected to enhance wet deposition of POPs to aquatic and terrestrial ecosystems (Macdonald et al., 2003; Meyer and Wania, 2008; Wania and Mackay, 1996). Macdonald et al. (2003) note that increases in precipitation will be an important variable driving the distribution of some POPs, such as hexachlorocyclohexane (HCH) and toxaphene, to aquatic systems. Both HCH and toxaphene have HLCs that favor water partitioning. Moreover, snow and snowmelt are powerful drivers in solvent switching and solvent depletion processes that may increase contaminant levels (Macdonald et al., 2003; Meyer and Wania, 2008). Falling snow provides a solvent switching condition under which contaminants can be readily adsorbed to snow surfaces and transported to the ground. As the climate warms and snow melts or sinters, the loss of surface area results in a solvent depleting condition that increases the concentration of contaminant in meltwater. Macdonald et al. (2003) estimate that this process might result in a loss of 10^5 to 10^6 m^2 of surface area for every 1000 kg of snow, which may lead to a substantial increase of POPs in meltwater.

Melting sea ice coupled with expanded open water may also accelerate the rate of exchange of some POPs from air to water. Macdonald et al. (2005) provide a summed PCB congener budget of gas exchange into the Arctic Ocean of 20 metric tons/year, and estimate that reduced Arctic sea ice cover of 50% could result in a proportionate doubling of PCB air to sea exchange. Glaciers have also acted as long-term sinks for POPs and melting of this ice is expected to remobilize these archived pollutants (Fig. 1, Legend Item F) (Blais et al., 2001). However, pollutant remobilization from glaciers may not be a major influence on the overall POP budget in Arctic ecosystems. One exception is DDT, for which Arctic glacial melt is projected to be a significant climate-modulated source (Blais et al., 2001).

Organic carbon cycles in terrestrial and aquatic systems will also be altered by climate change, which will in turn alter POP distributions (Macdonald et al., 2003; Magnuson et al., 1997; Schindler et al., 1997). Declines in dissolved organic carbon (DOC) were observed between 1970 and 1990 in boreal lakes in northwestern Ontario during an extended period of climate warming and drought coupled with increased forest fires (Schindler et al., 1997). The declines in DOC were

attributed to decreased stream flow to lakes caused by drought and increased evaporation from warming. POPs will readily partition from water to carbon-rich particles, such as DOC. Thus, reduced DOC levels due to climate change could reduce the capacity of waters to bind these contaminants thereby making them more bioavailable for uptake by aquatic species (Magnuson et al., 1997; Schindler et al., 1997).

Temperature-induced acceleration of organic carbon metabolism by soil and sediment biota could also increase contaminant concentrations and promote partitioning to water and aquatic biota (Macdonald et al., 2003). However, biodegradation rates of POPs will also increase with rising soil and sediment temperature (Fig. 1, Legend Items H, M), which may ameliorate POP increases from this solvent depleting process (Sinkkonen and Paasivirta, 2000; Sweetman et al., 2005). Increased temperatures will also increase the volatilization of POPs from soils to air (Fig. 1, Legend Items G, K) where they will be subject to photodegradation and transport (Fig. 1, Legend Item C) (Beyer et al., 2003; Brubaker and Hites, 1998; Ma et al., 2004; Scheyer et al., 2005). For example, the loss of permafrost associated with rising temperatures will re-release pollutants from these once frozen soils making them available for atmospheric partitioning or runoff to aquatic systems (Macdonald et al., 2005).

In addition to the many abiotic factors that can influence contaminant behavior, altered species migration patterns linked to climate change could be an important factor modulating the transport of POPs (Blais et al., 2007). Migratory species, particularly fish, birds, and marine mammals, may be exposed to contaminants in one location and transport these contaminants in substantial quantities to other locations. This biotic transport of contaminants may be similar in magnitude to atmospheric and oceanic transport (Burek et al., 2008). There is evidence, for example, that Arctic and Antarctic birds may act as vectors transporting persistent contaminants from oceans to terrestrial systems via their guano (Blais et al., 2005). In Canadian coastal ponds under the nesting cliffs of northern fulmars (*Fulmarus glacialis*), concentrations of HCB, DDT, and mercury were 10 to 60 times higher than contaminant concentrations in sediments from unaffected ponds. Similar results have been observed for Antarctic seabirds, whereby elevated DDT and HCH levels have been measured in sediments at locations where penguins historically migrated (Blais et al., 2007). These studies provide some evidence that climate-induced fluctuations in the migratory patterns of birds could play an important role in altering the local and global transport of POPs (Burek et al., 2008). In addition, PCB fluxes are up to eight times higher in sub-Arctic lakes receiving the greatest sockeye salmon (*Oncorhynchus nerkus*) returns than in lakes receiving atmospheric inputs of PCBs alone (Krummel et al., 2003). Since temperature is an important controller of anadromous and freshwater fish migrations, temperature increases linked to climate change could alter POP fate through changes in fish spawning behavior (Wrona et al., 2005).

2.3. Altered fate and behavior of pesticides

Like the POPs, climate change will influence the environmental fate and behavior of pesticides by altering fundamental mechanisms of environmental partitioning primarily through mechanisms of increased volatility, wet deposition, and enhanced degradation. While additional research is needed, many pesticides may prove to be less susceptible to solvent depleting processes than POPs since they are generally less persistent and more likely to degrade with climate change. Specifically, global warming may reduce soil and aquatic concentrations of pesticides due to a combination of increased volatilization and degradation (Bailey, 2004; Benitez et al., 2006; Van den Berg et al., 1999) (Fig. 1, Legend Items G, H, I, K, M, N). Conversely, increases in the intensity and frequency of rain and storm events will promote the wet deposition of pesticides to terrestrial and aquatic systems (Bollmohr et al., 2007; Burgoa and Wauchope, 1995; Chiovarou and Siewicki, 2007; Dabrowski et al., 2002; Presley et al., 2006; Vu et al., 2006). Independent of these distribution processes, climate change may alter the frequency and amount of pesticides used as agriculture shifts in response to the rapidly changing climate (Chen and McCarl, 2001; Reilly et al., 2001, 2003).

Volatilization is a key factor in the environmental partitioning of pesticides, and global warming could lead to enhanced volatilization of pesticides relative to soil and water. Van den Berg et al. (1999) notes that volatilization processes may be responsible for the loss of up to 50% of the applied dose of a pesticide, depending on the properties of the pesticide, application technique used and environmental conditions. With this atmospheric partitioning, pesticides may be dispersed from areas of high concentrations to areas of lower concentrations, possibly exposing new populations to the toxic effects of the pesticides (Beyer et al., 2003). In addition to enhanced volatility, climate change could have an important effect on accelerating pesticide degradation (Bailey, 2004; Benitez et al., 2006; Bloomfield et al., 2006). Bailey (2004) examined residues of the pesticide isoproturon in soils over a twenty-year period and found that from 1997–2001 increased degradation in warmer soils caused pesticide concentrations to fall too low to control weed growth 30 days earlier than in years before 1997. Additionally, increased water temperature was found to increase the photodegradation rate of several phenyl-urea pesticides (Benitez et al., 2006). Given the potential increase in the loss of applied pesticides due to enhanced volatility and degradation, a compensatory increase in pesticide applications may be necessary to be efficacious against target pests. Bloomfield et al. (2006) report on the findings of the European Food Safety Authority's 2005 Scientific Panel that for every 10 °C increase in temperature, it is predicted that the half-life of pesticides in soils may decrease by 60%.

The IPCC (2007e) reports that precipitation events and extremes are very likely to become more frequent, widespread, and intense during the 21st century. Moreover, a range of climate models supports a likely increase in the intensity of typhoons and hurricanes with heavier precipitation and higher peak wind speeds (IPCC, 2007e). Precipitation scavenges gases and aerosols, with adsorbed chemical particles, from the atmosphere and deposits them to surfaces (Fig. 1, Legend Item E). As storms and rainfall events become more intense and frequent, increasing amounts of contaminants will be deposited to surfaces and lost in runoff, predominantly as pulse releases, exposing humans and wildlife to these chemicals (Fig. 1, Legend Item J) (Bollmohr et al., 2007; Burgoa and Wauchope, 1995; Chiovarou and Siewicki, 2007; Presley et al., 2006; Vu et al., 2006). Bollmohr et al. (2007) examined the exposure and toxicity of a variety of pesticides, including chlorpyrifos and endosulfan, in arthropods and fish in the Lourens River and estuary in Western Cape, South Africa. No detectable amounts of the pyrethroids cypermethrin and fenvalerate were measured in the upper Lourens River, but these pesticides were found in the estuary at levels likely to pose acute and chronic risk to aquatic life. Pesticide concentrations in a rice paddy watershed at the Sakura river basin in Japan were monitored for 3 years starting in 2002 (Vu et al., 2006). Sixteen different herbicides were detected in the stream water, and surface drainage significantly increased during rainfall events greater than 1.5 cm per day. Elevated soil moisture associated with increased precipitation could also enhance the degradation of pesticides to differentially toxic and environmentally mobile degradates (Fig. 1, Legend Item I) (Van den Berg et al., 1999). Conversely, the hydrolytic degradation of these chemicals may be limited in regions with reduced precipitation and lower soil moisture levels (Bailey, 2004; Van den Berg et al., 1999).

In terms of the links between storm intensity and chemical contamination of aquatic systems, Chiovarou and Siewicki (2007) modeled the transport and fate of the six pesticides, atrazine, carbaryl,

diquat dibromide, imidacloprid, and fipronil, in water bodies in Volusia County, Florida and Portland, Oregon under different storm intensities. Concentrations of all six contaminants were found to increase with increasing storm intensity. Consistent with these results, Presley et al. (2006) investigated pollutant and pathogen levels in New Orleans, Louisiana shortly following Hurricane Katrina. They measured soil and sediment concentrations of several contaminants, including the POP aldrin and other semi-volatile organic pollutants, as well as several metals, and found levels that exceeded U.S. EPA human health soil screening levels, which are used to identify hazardous waste sites that merit further evaluation under Superfund law. Burgoa and Wauchope (1995) also found a five-fold increase in applied pesticide loss to runoff during extreme rainfall events. These studies provide evidence that the influence of climate change on increasing storm intensity and frequency could lead to episodes of heightened contamination of water bodies and surrounding watersheds.

It is not possible to fully consider the effects of climate change on pesticide distributions in the environment without also considering anticipated shifts in agriculture. Climate change is likely to affect agriculture by shifting the location and type of crops grown and the range and magnitude of crop pests. Pesticide use will shift in response to these altered cropping patterns and crop pest distributions. Although most investigations have focused on the U.S. and Europe, growers are expected to be able to expand crop production to higher latitudes and altitudes not currently suitable for farming (Bloomfield et al., 2006; MAFF, 2000; Reilly et al., 2003; Tubiello et al., 2002).

Tubiello et al. (2002) predict that both wheat and corn production will migrate north in the U.S. due to increased temperature and precipitation, while hotter and drier climates in the south will experience decreased crop production. Warmer temperatures in northern regions will also lead to longer growing seasons, potentially allowing increased farming and increased pesticide use. Farmers will also be able to grow new crops in areas currently under cultivation with other crops. Increased temperatures may make the currently temperate south of England favorable for growing sunflower, grapes, peaches (Fuller et al., 2001), and grain maize (Bloomfield et al., 2006). These types of expanded cropping patterns will likely result in new pesticide uses on naïve ecosystems, as well as potential increases in the volume and array of pesticides used.

Another route by which climate change is likely to affect pesticide use is by altering the distribution and abundance of crop pests. Climate change may influence crop pest populations by reducing generation times and over-wintering mortality, increasing the number of generations and population growth rates, and altering crop–pest synchrony (Cannon, 1998; Olfert and Weiss, 2006; Patterson et al., 1999; Porter et al., 1991). Studies show that the main drivers of pest distribution and abundance are temperature, rainfall, and CO_2, all of which are being altered with climate change (Gutierrez et al., 2006; Porter et al., 1991; Rafoss and Saethre, 2003).

One early study modeled the potential distribution of the European corn borer (*Ostrinia nubilalis*) and found an estimated northward shift in the pest's European range of up to 1220 km with a temperature increase of 3–6 °C (Porter et al., 1991). Olfert and Weiss (2006) made a similar prediction for three pest species of beetles in Canada. In a more regionally based analysis, Gutierrez et al. (2006) examined the distribution and abundance of pink bollworm (*Pectinophora gossypiella*) in cotton in Arizona and California. Their model predicts that the bollworm is currently unlikely to reach pest status in the Central Valley of California, but that its range is likely to expand into the Central Valley with temperature increases of 1.5–2.5 °C. Rafoss and Saethre (2003) predict that the codling moth (*Cydia pomonella*) will extend its range and abundance in Norway with increasing temperatures, and that the Colorado potato beetle could migrate into Norway where it is not currently established. In contrast to the studies described above, Newman (2005) predicted that climate change would reduce the abundance of aphid species in southern Britain. These varied results demonstrate that while pests may generally increase in number and distribution, changes are likely to be species and region specific.

Some studies have examined how pesticide use could shift in response to these expected climate change-induced alterations in pest distributions and intensity (Chen and McCarl, 2001; Reilly et al., 2001, 2003). For example, Reilly et al. (2003) focus modeling on the decades of the 2030s and 2090s and assess climate change impacts on pesticide use by measuring pesticide expenditures. They project climate-linked increases in pesticide expenditures in the U.S. ranging from 10–20% on corn, 5–15% on potatoes, and 2–5% on soybeans and cotton, but variable shifts in pesticide expenditures on wheat of $\pm$15% depending on the region and climate change scenario. No delineation is provided concerning the difference in pesticide expenditures between the decades studied. In addition, this modeling applies the IPCC's IS92A emissions scenario, which has since been updated by the IPCC under its “Special Report on Emission Scenarios” (IPCC, 2000). Despite these limitations, the results from this study are generally consistent with findings by Chen and McCarl (2001) in which increases in U.S. pesticide expenditures are projected in 2090 for corn, cotton, potatoes, and soybeans pests, with variable changes in wheat-related pesticide expenditures.

Expanded cropping patterns and increased pest pressures are expected to increase the variety and amount of pesticides used. Moreover, increased pesticide usage may be necessitated as climate change enhances chemical volatilization, degradation, and runoff. Taken together, these climate change-induced shifts in agriculture may increase human and wildlife exposures to pesticides.

3. Effects of climate change on contaminant-linked human health effects

The IPCC projects that climate change is likely to affect the health of millions of people, and that the effects will be mostly negative (Confalonieri et al., 2007). The elderly, infants, children, and urban poor are expected to be most vulnerable to the rapidly changing climate (Confalonieri et al., 2007; Ebi et al., 2006; Patz et al., 2000a, 2005). Notable adverse consequences of climate change on human health include increased death and injury associated with more severe and frequent heat waves, extreme weather events, and enhanced vector-borne and allergic disease transmission. While adverse health outcomes are projected to be greatest in low-income countries, more severe, frequent, and widespread heat waves and storm events will also impact developed countries unprepared to cope with these events (Confalonieri et al., 2007).

There continues to be a lack of data describing the effects of contaminant exposures on human health and vulnerable subpopulations under projected climate change scenarios. However, a number of studies suggest that the toxicity of ozone and PM will be exacerbated with global warming, and some of these data support that older adults will be especially vulnerable (Bell et al., 2007; Confalonieri et al., 2007; Dominici et al., 2006; Fiala et al., 2003; IPCC, 2007c; Katsouyanni et al., 1993; Knowlton et al., 2004; Koken et al., 2003; Mauzerall et al., 2005; Ordonez et al., 2005; Rainham and Smoyer-Tomic, 2003; Ren and Tong, 2006). Other potential interactions between climate change and toxicant exposure include increased susceptibility to pathogens (Abadin et al., 2007; Nagayama et al., 2007; Smialowicz et al., 2001) and aeroallergens (D'Amato et al., 2002; Diaz-Sanchez et al., 2003; Epstein, 2005; Janssen et al., 2003). Table 1 summarizes important interactions between climate change, toxicant exposures, and human health.

3.1. Vulnerable subpopulations

Elucidating the relationship between humans and the climate is complicated by the interactive nature of the many environmental,

Table 1
Climate change-induced effects of contaminants on human health.

Climate change-induced effect	Relationships/Interactions	References
Increased cardio-respiratory disease	▪ ↑ temperature exacerbates the adverse effects of ozone and PM ▪ The elderly and individuals with pre-existing cardio-respiratory disease may be more vulnerable to these effects	(Bell et al., 2007; Confalonieri et al., 2007; Dominici et al., 2006; Fiala et al., 2003; IPCC, 2007a; Katsouyanni et al., 1993; Knowlton et al., 2004; Koken et al., 2003; Mauzerall et al., 2005; Ordonez et al., 2005; Rainham and Smoyer-Tomic, 2003; Ren and Tong, 2006)
Altered exposure and risk	▪ Some populations may experience increases or decreases in POP exposures and health risks depending on the region and diet of exposed individuals ▪ Pesticides may impair mechanisms of temperature regulation especially during times of thermal stress	(Bard, 1999; Gordon, 1997; McKone et al., 1996; Watkinson et al., 2003)
Increased susceptibility to pathogens	▪ Toxicants can suppress immune function, and climate-induced shifts in disease vector range will result in novel pathogen exposure ▪ Immune system impairment linked to toxicants may increase human vulnerability to climate shifts in pathogens ▪ Low-income populations, infants, children, and the chronically ill may be more susceptible	(Abadin et al., 2007; Haines et al., 2006; Lipp et al., 2002; Nagayama et al., 2007; Patz et al., 2005; Rogers and Randolph, 2000; Smialowicz et al., 2001)
Increased allergenicity potential	▪ Air pollution and allergen exposures linked to climate change can exacerbate allergic disease and asthma incidences ▪ Climate change enhanced allergen production coupled with POP exposures may sensitize individuals to allergic disease ▪ Low-income populations, infants, children, and the chronically ill may be more susceptible	(D'Amato et al., 2002; Diaz-Sanchez et al., 2003; Epstein, 2005; Janssen et al., 2003)

biological, and socioeconomic conditions that can influence human health (Epstein, 2005; Haines et al., 2006; McMichael et al., 2006; Patz et al., 2005). The nature of negative health outcomes linked to climate change and the ability of populations to acclimate will depend on many conditions. These conditions include the age distribution and prevalence of inherited disease across the population, the surrounding physical and biological environment, and the many social and economic variables that influence population health (e.g., education, health care infrastructure, economic development) (Haines et al., 2006; McMichael et al., 2006).

Assessments of the U.S. population have identified the very young (<1 year), older adults (>65 years), and immuno-compromised individuals as more vulnerable to climate change because they have a reduced capacity to acclimatize to extreme heat and are also more vulnerable to vector-, food-, and water-borne disease (Ebi et al., 2006; Patz et al., 2000b). Ebi et al. (2006) note that there will be 100 million more Americans that are aged 65 or older in 2100 than in 2000, leading to generally increased vulnerability of the U.S. population to climate sensitive health outcomes. The effects of contaminants on vulnerable subpopulations warrant further study, although there is evidence that older individuals will be more susceptible to climate–air pollutant interactions (Fiala et al., 2003; Koken et al., 2003; Ordonez et al., 2005). In addition, low-income populations, infants, children, and chronically ill individuals may be especially susceptible to climate sensitive outcomes linked to interactions between pollutant exposures and changes in vector-borne and allergic disease (D'Amato et al., 2002; Diaz-Sanchez et al., 2003; Epstein, 2005; Haines et al., 2006; Janssen et al., 2003).

3.2. Air pollutants and cardio-respiratory disease

Studies examining interactions between climate change, air pollution, and human health have focused largely on tropospheric ozone and PM (Confalonieri et al., 2007; IPCC, 2007c). Generally, heat appears to render people more vulnerable to the adverse effects of air pollution. Climate change-induced increases in tropospheric ozone and PM, as is projected for many regions, coupled with global warming may exacerbate human vulnerability to cardio-respiratory disease especially among older adults.

Rising temperatures appear to increase susceptibility to cardio-respiratory disease linked to air pollution exposures. Epidemiological evidence suggests that heat exacerbates mortality and morbidity from cardio-respiratory disease in humans exposed to ozone and PM (Fiala et al., 2003; IPCC, 2007c; Koken et al., 2003; Ordonez et al., 2005; Rainham and Smoyer-Tomic, 2003). During the European heat wave of 2003, there was a surge in respiratory illnesses that was associated with increased concentrations of particulates and ozone especially among the elderly (Fiala et al., 2003; Ordonez et al., 2005). In another study illustrating the effects of climate sensitive outcomes on vulnerable older populations, males in Denver, Colorado aged 65 and older were found to be at increased risk for hospitalization for acute myocardial infarction, coronary arteriosclerosis, and pulmonary heart disease when co-exposed to higher temperatures and ozone (Koken et al., 2003). More recently, Bell et al. (2008) examined confounding factors, including air pollution levels, on heat-related mortality in three Latin American cities: Mexico City, Mexico, Sao Paulo, Brazil, and Santiago, Chile. They found that ozone and PM_{10} enhanced heat-related mortality, and that susceptibility was associated with increasing age in all three cities.

Modeling studies also show increased mortality and morbidity with increased ozone exposure coupled with global warming (Bell et al., 2007; Knowlton et al., 2004; Mauzerall et al., 2005; Rainham and Smoyer-Tomic, 2003). For example, using the IPCC A2 climate scenario (i.e., high growth of CO_2), a 4.5% increase in ozone-related deaths in the U.S. from climate change was modeled for the mid 2050s compared to the 1990s (Knowlton et al., 2004). Similarly, Bell et al. (2007) estimated elevated ozone in 50 U.S. cities applying the IPCC A2 climate scenario and found a corresponding increase in daily total mortality of 0.11% to 0.27%. By examining cardio-respiratory mortality in Toronto, Canada from 1980 to 1996, Rainham et al. (2003) detected a small, but consistent effect of air pollution (ozone, NOx, SO_2, CO, and PM_{10}) on temperature/humidity-related mortality. Recently, Ren et al. (2008) modeled the modulating effects of temperature and ozone interactions on mortality from 1987 to 2000 in 60 large eastern U.S. cities, and found that temperature had a synergistic effect on ozone-related mortality in the northeast. Specifically, for each 10 ppb increase in ozone, low, medium, and high temperatures increased mortality by 2.22%, 3.06%, and 6.22%, respectively. However, in the southeast U.S., the effects of temperature on ozone mortality were less robust than in the northeast. This suggests that regional differences (e.g., geography, population age structure, culture) may contribute to altering the effects of climate change and air pollution on adverse health outcomes.

Increasing temperatures may also modify the associations between PM and cardio-respiratory disease. Qian et al. (2008) found a synergistic effect of PM_{10} and high temperatures on daily cardio-respiratory

mortality in Wuhan, China. The PM_{10} effects were strongest on extremely high temperature days (daily average temperature 33.1 °C) and weakest during normal temperature days (daily average temperature 18 °C). Epidemiological data collected in Brisbane, Australia from 1996 to 2001 shows that respiratory- and cardiovascular-related hospital admissions and mortality were elevated when both temperature and PM concentrations increased (Ren and Tong, 2006). Katsouyanni et al. (1993) examined the interaction of smoke (PM), SO_2, and ozone with deaths in Athens, Greece during a July 1987 heat wave versus deaths in the 6 previous Julys (1981–1986). They found a significant positive association between SO_2 concentrations and temperature on the number of deaths when the average daily temperature was at or above a threshold of 30 °C. Dominici et al. (2006) constructed a database of hospital admission rates from 1999 to 2002 using U.S. Medicare data for cardiovascular and respiratory outcomes and injuries, ambient $PM_{2.5}$ concentrations, and temperature. They identified an association between $PM_{2.5}$ and hospital admission rates for respiratory outcomes that was positively correlated with temperature. Moreover, a comparison of regions with average temperatures that differed by 1 °C showed that the warmer regions had an additional nine hospital admissions per 10,000 individuals for respiratory tract infections per 10 $\mu g/m^3$ increase in $PM_{2.5}$. In contrast to the body of evidence showing positive associations between temperature and air pollution on death and disease, Samet et al. (1998) found little relationship between temperature and particulate matter on mortality upon examination of mortality data for Philadelphia, U.S. from 1973–1980.

3.3. Altered effects of POPs and pesticides

Questions concerning climate change impacts on the toxicity and risks to humans exposed to POPs and pesticides has received scant attention. McKone et al. (1996) conducted a study to model the effects of a 5 °C increase in temperature on human health risks in western U.S. populations exposed to HCB. Their analysis concluded that this global warming scenario would have little negative impact on health risk associated with HCB among these populations. In fact, exposures to humans might decline because of enhanced environmental degradation and the tendency of HCB to partition to the atmosphere with rising temperature (Ma et al., 2004). This atmospheric partitioning would remove it from water, thereby reducing exposures to aquatic biota, and in turn, potentially reducing human dietary exposures (Macdonald et al., 2005). However, under this scenario, this compound could then be subject to atmospheric transport to northern latitudes, where wet deposition to aquatic systems may lead to potentially elevated dietary exposures and health risks among exposed northern and indigenous communities (Bard, 1999).

Chemical toxicant exposures may also affect homeostatic temperature regulation in humans and other endotherms. Organophosphate and carbamate insecticides are known to elicit a fever in humans. Conversely, acute exposures in the rat lead to an acute reduction in core temperature followed by a delayed elevation in the core temperature (Gordon, 1997; Watkinson et al., 2003). In additional experiments, rats have been chronically exposed to dietary chlorpyrifos, and then subsequently challenged with a larger dose of chlorpyrifos (Gordon and Padnos, 2002). The ensuing hypothermic response was observed to be greater than for a normal acute dose, indicating that chronic exposure may sensitize the thermoregulatory response. Intoxication by these classes of pesticides may make it even more difficult for humans (and other endotherms) to maintain normal core temperatures, especially during times of thermal stress, such as heat waves.

3.4. Increased vulnerability to disease vectors

The potential for adverse human health impacts extends beyond those direct effects linking climate change to augmented exposures and toxicity. Climate change-induced shifts in disease vector range and severity coupled with contaminant exposures could increase human vulnerability to disease by impinging on the ability of individuals to mount an effective immune response to new pathogen exposures.

The distribution and emergence of vector-borne diseases, such as malaria and cholera, are predicted to be dependent on temperature, humidity, and precipitation (Lipp et al., 2002; Patz et al., 1996, 2005; Rogers and Randolph, 2000). As such, climate change is predicted to facilitate the reemergence or expansion of endemic vector-borne diseases or might promote the migration of these diseases to new regions. For example, cholera incidences in south Asia are linked to weather patterns (Patz et al., 2000a) and are predicted to increase with shifts in precipitation patterns (IPCC, 2007c). Likewise, malaria is predicted to migrate into higher latitudes and altitudes, particularly in Africa and South America, where it is endemic, although regions of Africa are also predicted to see declines due to high temperatures and desertification (IPCC, 2007b).

Evidence supports a link between contaminant exposures and suppressed immune system function (Abadin et al., 2007; Nagayama et al., 2007; Smialowicz et al., 2001). Immunotoxicity is a sensitive endpoint for several POPs, including heptachlor, PCBs, and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Exposures to POPs may decrease the ability of humans (and other animals) to fight infection (Abadin et al., 2007). Young rats exposed to heptachlor were observed to have suppressed antibody-mediated immune response as adults (Smialowicz et al., 2001). A study of Japanese infants found that perinatal exposure to dioxins, PCBs, and organochlorine pesticides altered the ratios of lymphocyte subsets, potentially leading to increased autoimmune disease and immune suppression later in life (Nagayama et al., 2007). While further study is need, immune system impairment linked to toxicant exposures may increase human vulnerability to climate-induced shifts in vector borne and infectious diseases. Populations living in lower income countries may be especially vulnerable to these pathogen–pollutant interactions as they may lack the resources to prevent and manage disease (Haines et al., 2006).

3.5. Allergenicity

In addition to changes in vector-borne disease, the incidences and severity of allergic disease are rising, especially in industrialized countries (D'Amato et al., 2002; Diaz-Sanchez et al., 2003). Asthma prevalence has quadrupled in the United States since 1980. Air pollution and higher concentrations of CO_2-induced allergens linked to climate change may be contributing to increased rates of allergic disease and asthma (Epstein, 2005; Shea et al., 2008).

Shifts in plant populations have already been documented as a result of climate change (Rogers et al., 2006; Root et al., 2003; Singer et al., 2005). For example, studies show that increasing concentrations of CO_2 enhance the production of Amb a 1 allergen and pollen from ragweed (*Ambrosia artemisiifolia* L.) (Rogers et al., 2006; Singer et al., 2005). In addition, diesel exhaust, which contains numerous pollutants, including PM, NOx, VOCs, CO, and polycyclic aromatic hydrocarbons (PAHs), has been shown to enhance allergenicity and asthma symptoms in adults and children by acting synergistically with allergens (D'Amato et al., 2002; Diaz-Sanchez et al., 2003; Janssen et al., 2003). For example, Janssen et al. (2003) found that Dutch children aged 7–12 from 24 schools within 400 m of a major roadway had increased sensitization to outdoor allergens. This relationship between adverse symptoms and traffic-related air pollution was largely restricted to children with pre-existing bronchial hyperresponsiveness (common among asthmatics) and allergen sensitivity. Thus, the combination of enhanced air pollution and allergen production linked to climate change may exacerbate allergic disease and asthma incidences in vulnerable individuals, especially children, infants, and asthmatics (Epstein, 2005).

4. Effects of climate change on contaminant toxicity to wildlife

There is substantial evidence that climate change is affecting the phenology of organisms, the range and distribution of species, and the composition and dynamics of communities (Lovejoy and Hannah, 2005; Penuelas and Filella, 2001; Root et al., 2003; Walther et al., 2001). While species have historically acclimated or adapted to changes in climate, the rapid rate of current climate change coupled with increasingly fragmented and impaired habitats present unprecedented challenges for modern species (Boone et al., 2007; Fisk et al., 2005; Occhipinti-Ambrogi, 2007; Rohr et al., 2004; Schiedek et al., 2007).

The bioavailability and toxicity of POPs and pesticides in wildlife is likely to increase in response to rising temperatures and salinity (Boone and Bridges, 1999; Capkin et al., 2006; Gaunt and Barker, 2000; Heugens et al., 2001; Moore et al., 2003; Schiedek et al., 2007; Silbergeld, 1973; Song and Brown, 1998; Tachikawa and Sawamura, 1994; Wang et al., 2001; Waring and Moore, 2004). An underlying mechanism of this interactive toxicity is that temperature alters the toxicokinetics of chemical pollutants in exposed biota (Buchwalter et al., 2003; Lydy et al., 1999; Maruya et al., 2005). Another mechanism probably influencing this enhanced toxicity is that increasing temperature can alter homeostasis and other key physiological mechanisms, thereby exacerbating the adverse effects of contaminants (Anderson and Peterson, 1969; Broomhall, 2002, 2004; Gordon, 2003; Heath et al., 1994; Patra et al., 2007).

Some populations, particularly those living at the edge of their homeostatic or physiological tolerance range, may be more vulnerable to the to the dual stresses of climate change and contaminant exposures (Anderson and Peterson, 1969; Gordon, 2003; Heath et al., 1994; Patra et al., 2007). Moreover, the rapidity of climate change-induced shifts in habitats and trophic food webs could affect contaminant toxicity by altering exposure pathways and increasing susceptibility of some populations, especially those already under stress (AMAP, 2004; Breivik et al., 2004; Brook and Richardson, 2002; Gaston et al., 2003; Gilbertson et al., 2003; Macdonald et al., 2005; Olafsdottir et al., 1998; Sagerup et al., 2000). A limitation of studies investigating the interactive toxicity of climate change and contaminant exposures is that observed biological effects may prove to have a non-linear relationship to the stressors. That is, an incremental increase in temperature or contaminant may be less important than thresholds or tipping points that trigger potentially major synergisms in adverse effects across species, populations, and communities. Table 2 summarizes important climate change–contaminant interactions in wildlife.

4.1. Altered uptake and elimination

Increasing temperatures will generally increase the uptake and excretion of toxicants. For example, Buchwalter et al. (2003) observed enhanced uptake of the organophosphate (OP) pesticide chlorpyrifos with increasing temperatures among three aquatic insect species: *Notonecta kirvyi*, *Pteronarcys californica*, and *Dicosmoecus gilvipes*. Likewise, uptake of the pesticides chlorpyrifos and methyl-parathion, and the POP pentachlorobenzene, increased at 20 °C and 30 °C compared to 10 °C in the midge, *Chironomus tentans* (Lydy et al., 1999). Decreased body burdens of chlorpyrifos and methyl-parathion were also observed at higher temperatures, indicating increased metabolism and excretion. Yet, body burdens did not change for pentachlorobenzene at any of the three temperatures tested. In the estuarine fish, *Fundulus heteroclitus*, warmer temperatures (25 °C) contributed to a rate of elimination of toxaphene congeners that was two-fold higher than in cooler water (15 °C) (Maruya et al., 2005). Similarly, Paterson et al. (2007) monitored elimination of 72 PCB congeners in perch under typical northern latitude annual temperature cycles, and found that elimination occurred only during the spring and summer months when water temperatures were near or above 20 °C.

4.2. Increased toxicity

The toxicity of contaminants may be enhanced with increasing temperatures (Boone and Bridges, 1999; Capkin et al., 2006; Gaunt and Barker, 2000; Silbergeld, 1973). While the exact mechanisms underlying this relationship are not fully understood and the majority of research focuses on aquatic species, studies indicate that temperature-induced shifts in metabolism are one controlling factor (Buckman et al., 2007; Lydy et al., 1999; Monserrat and Bianchini, 1995).

The lethality of the POP dieldrin to the freshwater darter (*Etheostoma nigrum*) increased with increasing temperatures (Silbergeld, 1973). In the green frog (*Rana clamitans*), the toxicity of the insecticide carbaryl increased with temperature increases from 17 °C to 22 °C to 27 °C (Boone and Bridges, 1999). Gaunt and Barker (2000) found that the toxicity of the herbicide atrazine to catfish (*Ictalurus punctatus*) increased with increasing temperature or decreasing dissolved oxygen. They predicted that changes in these two parameters, which would likely occur simultaneously in climate change scenarios, could greatly enhance the toxicity of atrazine to some aquatic species. Capkin et al. (2006) observed increased mortality in juvenile rainbow trout (*Oncorhynchus*

Table 2
Climate change-induced toxicological effects of contaminants on wildlife.

Climate change-induced effect	Relationships/Interactions	References
Altered uptake and elimination	▪ ↑ temperature = ↑ uptake of toxicants ▪ ↑ temperature = ↑ elimination ▪ ↑ temperature = remobilization of bioaccumulated POPs	(Buchwalter et al., 2003; Lydy et al., 1999; Maruya et al., 2005)
Increased toxicity	▪ ↑ temperature = ↑ toxicity ▪ ↑ temperature = ↑ metabolism and potentially altered metabolite profiles ▪ Toxicant exposure may limit capacity of species and populations to acclimate to altered temperatures. ▪ Pollutant-exposed ectotherms and species at the edge of their physiological tolerance range may be especially sensitive to temperature increases.	(Anderson and Peterson, 1969; Boone and Bridges, 1999; Brian et al., 2008; Broomhall, 2002, 2004; Buckman et al., 2007; Capkin et al., 2006; Gaunt and Barker, 2000; Gordon, 2003; Heath et al., 1994; Lydy et al., 1999; Monserrat and Bianchini, 1995; Patra et al., 2007; Silbergeld, 1973)
Altered environmental salinity	▪ ↓ solubility and ↑ bioavailability of pesticides/POPs ("salting out effect") ▪ ↑ salinity + ↑ POP/pesticide exposure may alter osmoregulation due to altered enzymatic pathways	(Fortin et al., 2008; Heugens et al., 2001; Moore et al., 2003; Schiedek et al., 2007; Schlenk and El-Alfy, 1998; Schwarzenbach et al., 2003; Song and Brown, 1998; Tachikawa and Sawamura, 1994; Wang et al., 2001; Waring and Moore, 2004)
Altered ecosystems	▪ Altered POP sequestration and/or remobilization through shifts in food sources and starvation events ▪ Shifts in disease vector range and severity coupled with toxicant exposure inhibiting immune response may leave wildlife more susceptible to disease ▪ Low level exposures may impair organism acclimation to ecosystem alterations induced by climate change ▪ Climate change induced changes in trophic food webs may alter POP bioaccumulation and biomagnification	(AMAP, 2004; Braune et al., 2005; Furnell and Schweinsburg, 1984; Jenssen, 2006; Macdonald et al., 2005, 2003; Olafsdottir et al., 1998; Ramsay and Stirling, 1982; Schiedek et al., 2007, Stirling et al., 1999)

mykiss) exposed to the insecticide endosulfan as temperature was increased from 13 °C to 16 °C. In contrast to these findings, pyrethroids and DDT are generally thought to be more toxic under low temperature conditions, which may be due to a sodium channel modulated increase in nervous system vulnerability at lower temperatures (Narahashi, 2000). However, others have observed increased pyrethroid toxicity at elevated temperatures in leopard frogs (*Rana* spp.) (Materna et al., 1995) and water fleas (*Daphnia magna*) (Ratushnyak et al., 2005), illustrating the species-specific response to increased temperatures and toxicant exposures.

Temperature-dependent changes in metabolism appear to be one important mechanism modulating the biotransformation and enhanced toxicity observed under elevated temperature conditions. For example, despite the relatively high persistence of POPs in biota, Buckman et al. (2007) observed enhanced biotransformation of PCBs to the toxicologically active hydroxylated PCB metabolites by rainbow trout with rising temperature (8, 12, and 16 °C). Moreover, Lydy et al. (1999) postulate that while body burdens of the OP insecticides decline at higher temperatures, toxicity is ultimately enhanced due to an acceleration of the biotransformation of the OP insecticides to their more toxic *ortho*-analog metabolites. Monserrat and Bianchini (1995) suggested a similar explanation for the increased toxicity they observed when exposing crabs (*Chasmagnathus granulata*) to methyl parathion. There was an approximately ten-fold increase in acute lethality with temperature change from 12 °C to 30 °C. The authors suggest that the higher temperature favors enzymatic activation of the organophosphate over degradation and excretion.

The metabolism studies demonstrate a general concept that is likely to hold true for the effect of temperature on toxicity of many contaminants. While the rates of uptake and excretion may generally increase with increasing temperature, the ultimate toxicity of these contaminants will depend on whether changes in metabolism result in increased bio-activation or detoxification.

4.3. Altered homeostasis and physiological responses

The ability of species and populations to tolerate elevated temperatures may be impaired with toxicant co-exposures. Alterations in climate change parameters, predominantly temperature, will act as co-stressors with chemical toxicants, thereby affecting physiological processes and the ability of wildlife to maintain homeostasis (Broomhall, 2004). Ectotherms, such as fishes, amphibians, and reptiles, may be particularly vulnerable to these temperature–contaminant interactions. Moreover, species living at the edge of their physiological tolerance range may be less able to cope with the dual stressors of climate change and contaminant exposures (Anderson and Peterson, 1969; Gordon, 2003; Heath et al., 1994; Patra et al., 2007).

The generalized stress of maintaining homeostasis under increasing temperatures may potentiate the effects of some pesticides. When eggs of the Australian frog (*Limnodynastes peronii*) were reared under a high and low temperature regimen and exposed to the insecticide endosulfan, there was a negative effect of endosulfan on predator avoidance that was proportionally worse for the tadpoles reared at a higher temperature (Broomhall, 2004). This same effect was observed in a previous study with another amphibian species, *Litoria citropa* (Broomhall, 2002). Upper temperature tolerance limits were also reduced in the following four species of freshwater fish exposed to endosulfan and chlorpyrifos: silver perch (*Bidyanus bidyanus*), eastern rainbow fish (*Melanotaenia duboulayi*), western carp gudgeon (*Hypseleotris klunzingeri*), and rainbow trout (Patra et al., 2007). The ability of brook trout (*Salvelinus fontinalis*) and Atlantic salmon (*Salmo salar*) to acclimate to increasing temperature is impaired by sub-lethal doses of DDT (Anderson and Peterson, 1969). Heath et al. (1994) found that exposure of fathead minnows (*Pimephales promelas*) to low doses of the pyrethroid insecticide cyfluthrin could reduce their zone of temperature tolerance by 30%. Cyfluthrin exposure caused a maximum decrease of 3.3 °C below median heat tolerance levels and a 5.6 °C increase in median cold tolerance levels. They observed effects at concentrations as low as 170 parts per trillion.

Another important consideration for climate change and pollutant interactions is the timing of exposures at sensitive life stages inducing responses that in turn alter physiological processes. Brian et al. (2008) measured a transient increase of the yolk precursor protein, vitellogenin (VTG), in male fathead minnows at higher temperatures (30 °C vs. 20 °C) upon exposure to a mixture of endogenous steroidal estrogen, 17β-estradiol, synthetic steroidal estrogen, 17α-ethinylestradiol, and other estrogenic chemicals (4-tertnonylphenol, 4-tertoctylphenol, and bisphenol-A). The temperature-dependent increase in VTG was observed only during the first 24 h of exposure, demonstrating that the effects of elevated temperature were more pronounced early in the exposure period. Increased storm intensity and frequency associated with climate change could lead to episodes of high contaminant exposures due to chemical runoff. High exposure episodes that coincide with sensitive life stages, such as during maturation, spawning, and development, may be detrimental to aquatic species fitness and survival.

4.4. Altered environmental salinity

In addition to global warming, climate change-induced shifts in precipitation and evaporation patterns have resulted in increased salinity in subtropical and tropical oceans and a freshening of mid and high latitude waters (IPCC, 2007e). Sea level rise linked to climate change is projected to lead to salt water intrusion into previously freshwater habitats (IPCC, 2007e). However, salinity could decrease in waters receiving elevated inputs of freshwater due to increases in precipitation or snow and ice melt. In sum, the effects of climate change on salinity patterns are complex and may vary by region as a number of factors can influence this parameter. For example, in brackish water ecosystems, like the Chesapeake Bay, salinity patterns contribute to species distributions and are predicted to shift in response to climate change (Pyke et al., 2008; Rogers and McCarty, 2000). Salinity reductions are expected during winter due to projected increases in tributary flow linked to elevated precipitation. Conversely, increased regional drought frequency and sea level rise are predicted to lead to saltwater intrusion events and elevated salinity for portions of the Bay (Pyke et al., 2008; Rogers and McCarty, 2000).

Salinity–contaminant interactions are made additionally complex because salinity can influence the chemical itself or it may modulate toxicity and physiological functioning of species (Fortin et al., 2008; Heugens et al., 2001; Moore et al., 2003; Schiedek et al., 2007; Schlenk and El-Alfy, 1998; Schwarzenbach et al., 2003; Song and Brown, 1998; Tachikawa and Sawamura, 1994; Wang et al., 2001; Waring and Moore, 2004). Organic compounds are generally less soluble and more bioavailable in saltwater than in freshwater due to the "salting out" effect whereby water molecules are strongly bound by salts making them unavailable for dissolution of organic chemicals (Schwarzenbach et al., 2003). Thus, increased contaminant bioavailability and toxicity is possible in subtropical latitudes experiencing increased salinity, as well as in estuaries and coastal freshwater ecosystems subject to increased saltwater intrusion or droughts. Consistent with this hypothesis, increased mortality to the organophosphate pesticide dimethoate was observed in salt marsh mosquitoes (*Aedes taeniorhynchus*) and brine shrimp (*Artemia* sp.) under hyperosmotic conditions (i.e., 3–4 times the isoosmotic salinity) (Song and Brown, 1998). The authors concluded that the increased toxicity might be attributable to increased dimethoate bioavailability and accumulation at the elevated salinity levels compared to the isoosmotic conditions. They also report that another organophosphate pesticide, malathion, has a higher degradation half-life in seawater (3–5 days) than in freshwater (1 day), supporting the idea of higher persistence due to salting out effects.

Heugens et al. (2001) attribute the increased toxicity observed at elevated salinity to higher physiological costs for organisms to maintain osmoregulation leading to a decline in fitness and elevated sensitivity to contaminant exposures. There is support for this assertion as several studies show that altered salinity profiles coupled with POP and pesticide exposures may alter osmoregulatory function in aquatic organisms (Fortin et al., 2008; Hall et al., 1995; Moore et al., 2003; Schiedek et al., 2007; Schlenk and El-Alfy, 1998; Tachikawa and Sawamura, 1994; Wang et al., 2001; Waring and Moore, 2004).

Spikes in atrazine concentrations may occur after heavy rain events with concentrations reported in North American rivers at up to 108 μg/L and in the Chesapeake Bay at up to 30 μg/L (Fortin et al., 2008). A 96-hour exposure to atrazine at 5 μg/L impaired osmotic control in *F. heteroclitus* larvae, with higher prevalence of dehydrated larvae at isoosmotic (15 ppt) and extreme (35 ppt) salinities and hyperhydrated larvae at low salinities of 3 ppt (Fortin et al., 2008). In the absence of atrazine, salinity had no effect on the prevalence of hyper or hypo hydrated fish. In estuarine copepods (*E. affinis*), high (25 ppt) and low (5 ppt) salinity levels increase mortality in response to high doses of the atrazine (>2.6 mg/L) (Hall et al., 1995). The authors concluded that *Eurytorma* might be more physiologically effective at metabolizing atrazine at intermediate salinities, although impaired osmotic control at these salinity extremes is probably an important contributor to the elevated mortality. Similar results were observed in another study exposing the copepod, *Microarthridion littorale* to chlorpyrifos and DDT (Staton et al., 2002). While the mechanism leading to the impaired osmotic control in fish and altered toxicity in copepods is unknown, alterations in enzymatic pathways have been observed in fish under similar exposures (Moore et al., 2003; Tachikawa and Sawamura, 1994; Waring and Moore, 2004).

In Japanese medaka (*Oryzias latipes*), co-exposure to the pesticide pentachlorophenol (PCP) and elevated salinity resulted in reductions in PCP uptake and increases in clearance (Tachikawa and Sawamura, 1994). Decreased uptake of PCP was associated with decreased water flux across the gills and increased clearance was linked to increased Na^+, K^+-ATPase activity and developing chloride cells. However, pre-exposing Atlantic salmon smolts to atrazine in freshwater at concentrations greater than 1.0 μg/L resulted in mortality upon a 24-hour seawater challenge (Waring and Moore, 2004). Enhanced activity of flavin-containing monooxygenases (FMOs) is another enzymatic pathway that may play a role in potentiating the toxicity of some chemical toxicants under conditions of elevated salinity (Schlenk, 1998). FMOs are induced in the presence of salinity and play a role in maintaining cellular osmotic pressure. Elevated salinity leads to increased FMO activity, which in turn enhances production of a more bioactive metabolite in aldicarb-exposed fish (Wang et al., 2001).

4.5. Altered ecosystems

There is substantial evidence of the ecological impacts of climate change across terrestrial and aquatic environments ranging from polar to tropical regions (Lovejoy and Hannah, 2005; Penuelas and Filella, 2001; Root et al., 2003; Walther et al., 2001). While some species and populations may be especially vulnerable to climate change, it is important to recognize that these impacts will be concomitant with and in some cases exacerbated by other ecosystem stressors, notably chemical pollution, invasive species, over-harvesting, habitat destruction, and pathogens. The superimposition of these increasingly common ecosystem stressors with the rapidly changing climate could further hinder wildlife acclimation and adaptation to climate change (Cook et al., 1998; Fischlin et al., 2007; Macdonald et al., 2005; Occhipinti-Ambrogi, 2007; Scavia et al., 2002). The IPCC projects that ecosystem resilience in many regions is likely to be exceeded this century by an unprecedented combination of climate change disturbances and these many other anthropogenic and natural stressors (Fischlin et al., 2007).

Climate change producing alterations in trophic structures, food sources, migratory patterns, and feeding behavior may influence processes of bioaccumulation and biomagnification in POP-exposed animals (AMAP, 2004; Furnell and Schweinsburg, 1984; Macdonald et al., 2005; Olafsdottir et al., 1998; Ramsay and Stirling, 1982; Stirling et al., 1999). Important solvent switching and solvent depletion processes involve the partitioning of POPs from water to phytoplankton and zooplankton at the base of aquatic food chains followed by bioaccumulation and biomagnification up the food chain (Braune et al., 2005; Macdonald et al., 2005). Apex predators at the top of some food webs may experience significant biomagnification of POPs as a result of these solvent switching and solvent depletion processes. For example, polar bears (*Ursus maritimus*) generally have the highest concentrations of POPs of any Arctic animal (Braune et al., 2005). Stirling et al. (1999) observe that the loss of stable ice flows linked to climate warming are the major factor contributing to Hudson Bay polar bears coming ashore for several months of fasting in progressively poorer condition. Hudson Bay polar bears prey primarily on ringed seals (*Phoca hispida*), the population of which is in decline due to a loss of these stable ice flows (Furnell and Schweinsburg, 1984; Ramsay and Stirling, 1982; Stirling et al., 1999). Polar bears near starvation will use stored fat as an energy source, remobilizing POPs sequestered in these tissues and potentially resulting in the dual stresses of starvation and chemical toxicity (Macdonald et al., 2005).

Climate change-induced POP remobilization scenarios may apply to other species as well, such as migratory salmon, common eider (*Somateria mollissima*), thick-billed murres (*Uria lomvia*), and Arctic Char (*Salvelinus alpinus*) (AMAP, 2004; Macdonald et al., 2005; Olafsdottir et al., 1998). For example, Arctic cod (*Boreogadus saida*) are a primary, high fat forage fish for many Arctic species, and loss of critical sea ice habitat may adversely affect Arctic cod populations and those animals that rely on them for food. Gaston et al. (2003) analyzed the diets of thick-billed murres from 1981–2002, and observed a decrease in consumption of Arctic cod. This shift in diet increased the fat burned to the fat energy gained. These types of shifts in food sources could lead to greater relative biological burdens and remobilization of POPs. However, POP bioaccumulation may be reduced in some predators if they are able to switch to less contaminated food sources (Brook and Richardson, 2002; Macdonald et al., 2005).

Similar to humans, climate change-induced shifts in pathogen and disease vector ranges coupled with toxic contaminant exposures could render wildlife more susceptible to disease by inhibiting their ability to mount an effective immune response (Breivik et al., 2004; Burek et al., 2008; de Swart et al., 1996; Gilbertson et al., 2003; Kajiwara et al., 2002; Sagerup et al., 2000). Glaucous gulls (*Larus argentatus*) had a higher parasitic nematode infection level that was correlated with PCB and organochlorine pesticide concentrations (Sagerup et al., 2000). In laboratory and field studies, northern leopard frogs (*Rana pipiens*) exhibited immune suppression because of DDT or dieldrin exposure (Gilbertson et al., 2003). Harbor porpoises (*Phocoena phocoena*) exhibited a significant correlation between concentrations of PCBs, polybrominated diphenyl ethers (PBDEs), toxaphene, DDT and its metabolites and thymic atrophy and splenic depletion (Breivik et al., 2004). Harbor seals (*Phoca vitulina*) fed POP-contaminated fish collected from the Bering Sea for 2.5 years had higher body burdens of POPs than seals fed relatively uncontaminated fish, and displayed impaired immune responses including suppression of natural killer cell and specific T-cell activity, (de Swart et al., 1996).

POPs, especially PCBs, DDT, dioxins, and furans, have also been investigated as cofactors contributing to recent mass mortality incidences attributed to morbilliviruses among several marine mammal populations (Burek et al., 2008; Kajiwara et al., 2002; Kuiken et al., 2006). For example, a mass mortality incident of 10,000 Caspian seals (*Phoco caspica*) in the spring and summer 2000 was attributed primarily to canine distemper virus, and like other incidences, was preceded by an unusually mild winter. Kajiwara et al. (2002) found

that POP levels, especially for DDT/DDE and PCBs, were higher in animals collected during the Caspian seal epizootic incident than in earlier collections of healthy individuals, suggesting that these contaminants made animals more susceptible to disease. More recently, however, Kuiken et al. (2006) re-analyzed tissue from the same incident but eliminated some specimens from evaluation because they were diagnosed negative for the virus. After this adjustment, the authors found that POP concentrations in diseased seals in 2000 were comparable to concentrations found in seals sampled in previous years. These mixed results are indicative of the need to better understand the interactions of POP body burdens, immune system suppression, and climate-induced changes in pathogenic disease transmission among exposed populations.

In addition to a potentially diminished immune response, other toxic effects linked to chronic, low level POP exposures may impair organism acclimation to ecosystem alterations (Jenssen, 2006). High blood levels of POPs, including HCB, oxychlordane, DDT metabolites, and PCBs, have been associated with a decrease in viable offspring, a decrease in adult yearly survival rate, and an increase in wing feather asymmetry (Bustnes et al., 2002, 2003; Kuenzel, 2003; Leeson and Walsh, 2004). Thyroid hormone deficits during early life stages affect neurodevelopment and subsequent behavior and cognitive ability in vertebrates (Donahue et al., 2004; Jenssen, 2006). Studies of polar bears have shown disrupted thyroid hormone homeostasis induced by POP exposures (Norstrom, 2000; Skaare et al., 2001; Wiig, 1995). Jenssen (2006) hypothesize that since hunting and survival skills are dependent upon behavioral and cognitive abilities, altered thyroid hormone homeostasis associated with POP exposures may be a factor hindering polar bear acclimatization to retreating sea ice. In another example, PCB levels in the glaucous gull (*Larus hyberboreus*), a top predator in the Arctic food web, were significantly related to the proportion of time that adult gulls were absent from the nest (Bustnes et al., 2001). The authors suggested that the gulls required more time to gather food as a result of endocrine disruption or neurological disorders due to high contamination levels.

Climate change may also alter patterns of POP bioaccumulation and biomagnification by altering bottom-up or top-down mechanisms controlling trophic food webs (Braune et al., 2005; Macdonald et al., 2003; Schiedek et al., 2007). Climate change-induced alterations in bottom-up controlling mechanisms, such as altered nutrient and primary production, may lead to the addition or removal of trophic levels (Macdonald et al., 2003). This in turn could shift predators higher or lower in the aquatic food web, leading to a respective increase or reduction of POPs. Top-down alterations in trophic structures elicited by the changing climate, for example, could involve the loss or diminished populations of higher trophic level species leading to consumption further down the food chain and reduced POP biomagnification potential.

5. Conclusions

There is a growing body of evidence that climate change will have broad negative impacts on the distribution and toxicity of environmental contaminants (Bell et al., 2007; Buckman et al., 2007; Confalonieri et al., 2007; Dentener et al., 2006; Fiala et al., 2003; Hogrefe et al., 2004; Knowlton et al., 2004; Macdonald et al., 2005; Patra et al., 2007; Schiedek et al., 2007; Stevenson et al., 2006). However, many areas merit further examination. Direct investigation of climate change impacts on contaminant behavior and toxicity is needed as much of the current literature examines this issue indirectly (e.g., focusing on temperature, salinity, etc.). Research that does focus on climate change directly is of great benefit, but has dealt mainly with predicting pollutant behavior under different climate change scenarios. Less work has been undertaken to describe the toxicological consequences of these altered pollutant distribution patterns. This review also underscores the lack of data describing the effects of climate change and toxicant exposures on human health. While climate change is a global phenomenon, the existing literature has only recently started to explore contaminant interactions outside of North America and Europe (e.g., Bell et al., 2008; Qian et al., 2008). A greater understanding of the biological effects of climate change on chemical toxicity continues to be needed in other parts of the world. This data gap is of special concern since impoverished populations may be particularly susceptible to the interactive effects of climate change and contaminant exposures, as these groups are often exposed to other stressors, such as malnourishment and disease.

Air pollutant concentrations are closely intertwined with climate change, making ozone and PM particularly relevant, as they are influenced by and act on climate change. Air pollution is projected to increase in many regions due to climate change, especially in areas that are urbanized, polluted, and subject to reduced precipitation and stagnant atmospheric circulation patterns. A growing body of epidemiological and modeling evidence supports that global warming coupled with ozone and PM exposures could exacerbate the prevalence and severity of human cardio-respiratory disease and mortality. Given the large segment of the population exposed to outdoor air pollutants, a relatively modest increase in mortality and morbidity estimated from current modeling projections could translate into a substantial number of individuals at risk (Patz et al., 2005; Zhang et al., 2006). Certain subpopulations, especially the elderly, infants and children, and individuals with pre-existing health conditions, such as chronic cardiopulmonary and immunological diseases, may be especially susceptible to these adverse interactions (Ebi et al., 2006; Oberdörster, 2001; Patz et al., 2000a; Pope, 2000). There continues to be uncertainty, however, including that the modeling is based usually on a single emissions scenario and other co-stressors may obscure the interactions between climate, air pollution, and human health. Given the potentially serious consequences of climate–air pollutant interactions on human health, additional research is needed to further refine the modeling projections and describe underlying mechanisms of toxicity.

For POPs and other pesticides, increases in temperature and precipitation will influence environmental distribution through increases in chemical volatility and degradation. Climate change could facilitate a number of solvent depletion processes, including ice and snow melt, altered trophic structures, bioaccumulation and biomagnification, and organic carbon cycling, which could in turn cause substantial POP increases in water, soil, sediment, and biota. While these types of complex climate–POP interactions are not fully understood, they could be more problematic than climate sensitive outcomes leading to thermodynamic forcing and altered environmental partitioning (MacDonald et al., 2002).

Global warming will be expected to enhance partitioning of POPs and other pesticides to the atmosphere, though the increase in atmospheric concentrations of these pollutants may be offset by enhanced degradation (Bailey, 2004; Benitez et al., 2006; Dalla Valle et al., 2007; Sinkkonen and Paasivirta, 2000; Sweetman et al., 2005; Van den Berg et al., 1999; Wania and Mackay, 1996). Moreover, regions subject to increased storm intensity, frequency, and variability could experience pulses of chemical releases or runoff that might present acute risks to human health and wildlife populations (Bollmohr et al., 2007; Burgoa and Wauchope, 1995; Chiovarou and Siewicki, 2007; Dabrowski et al., 2002; Presley et al., 2006; Vu et al., 2006).

As for contamination at higher latitudes, some hypothesize that a reduction of the temperature gradient across latitudes could suppress the long-range transport of POPs (Beyer et al., 2003). Others present evidence that accelerated polar melting of snow, ice, and permafrost, as well as altered organic carbon cycling and metabolism, could remobilize and increase levels of archived pollutants and enhance their air to sea exchange (Blais et al., 2001; Macdonald et al., 2003; Magnuson et al., 1997; Meyer and Wania, 2008; Schindler et al., 1997). Climate change is also expected to result in the greater use of pesticides in regions experiencing increases in arable lands and

expansion of pest pressures (Chen and McCarl, 2001; Reilly et al., 2001, 2003). As a result, human and wildlife pesticide exposures and effects will shift. However, new, more efficacious pesticides and adaptive farming practices, such as altered plant varieties and planting regimens, could offset some of the expected increase in pesticide applications.

Increased temperature and salinity linked to climate change could enhance the toxicity of some POPs and other pesticides in aquatic biota. Altered biotransformation of contaminants to more bioactive metabolites appears to be an important mechanism by which climate change enhances chemical toxicity. Moreover, these climate change and contaminant interactions could compromise homeostasis and physiological responses, potentially impairing species fitness, reproduction, and development (Brian et al., 2008; Heugens et al., 2001; Schiedek et al., 2007).

The complex interactions between climate change and pollutants may be particularly problematic for species living at the edge of their physiological tolerance range. For most species, there are optimum ranges of temperature, salinity, pH, moisture, etc., and organisms living under conditions that approach their tolerance limits are often more vulnerable to additional stressors, such as climate change and chemical pollution (Gordon, 2003; Heath et al., 1994; Heugens et al., 2001; Patra et al., 2007). Species with narrow ranges of tolerance to changing environmental conditions may have difficulty acclimating to climate change. Pollutant exposures may further hinder the ability of organisms to acclimate and make them more susceptible to infectious and vector-borne disease. In addition, species with short generational times, such as microbes and insects, may adapt more successfully to climate change than those species with long generational times. Altered habitats caused by the rapidly changing climate also could trigger species migrations that ultimately push populations into sub-optimal regions where they may experience reduced overall fitness and diminished tolerance to toxicant exposures (Heugens et al., 2001; Schiedek et al., 2007).

Improving our understanding of the effects of multiple stressors on natural systems is an important challenge for environmental scientists. It has taken on more urgency as climate change is not only altering the fundamental structure and function of many ecosystems, but is impacting the distribution and toxicity of chemical pollutants. The vulnerability of human and wildlife populations to climate-sensitive chemical exposures, in the context of the many other stressors that are being altered with climate change, is the paramount question that requires more rigorous study. In addition, the effects of climate change on contaminant toxicity will almost certainly be non-linear, and an important question for future research will be to elucidate thresholds or tipping points in which contaminants as cofactors with other stressors lead to profound effects on ecosystems.

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