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Perspectives
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Review

Aquatic ecosystem dynamics following petroleum hydrocarbon perturbations: A review of the current state of knowledge

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A B S T R A C T

Petroleum hydrocarbon spills in aquatic environments are among the worst ecological disasters resultant of global trade and commerce. History has shown that despite taking measures to minimize their frequency, large spills still occur. Crude oil spilled in aquatic environments poses a significant threat to aquatic life, as toxic effects cascade across trophic levels, affecting phytoplankton, zooplankton, fish, aquatic birds, mammals, and benthic organisms. The literature shows much work has been done detailing the toxicity of crude oil at each of the aforementioned trophic levels, but very little of this knowledge has been incorporated into modelling studies. Instead, the majority of contemporary models focus on the abiotic fate of spilled crude oil, driven by factors such as evaporation, dissolution, dispersion, sinking, and sedimentation. In this study, we present a thorough review of the role of crude oil toxicity on aquatic organisms from a food web point of view, followed by an overview of the modelling literature, and finally outline a modelling plan in which we aim to fill the biological/ecological gap in contemporary oil spill models. We conclude with a North American viewpoint, emphasizing the importance of robust ecological management tools, as the Laurentian Great Lakes hub is vital to shipping and industry, but at high risk for petroleum hydrocarbon spills.

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Introduction

The Deepwater Horizon oil rig explosion on April 20th, 2010 ignited public fury, and brought to the forefront the ongoing debate of our dependence on oil extraction, transport, and use as a primary fuel. Described as an ecological disaster and the greatest oil spill in US history, the BP (British Petroleum) oil rig leak is estimated to have spewed 4.9 million barrels of crude oil into sea before the capping of its underwater well. This figure dwarfs the Exxon Valdez oil spill (EVOS; acronyms used are listed in the Appendix A) that totalled 10.9 million gallons (Council, 2003; Paine et al., 1996). In its time, however, the EVOS was remarkable for its environmental damage (Shaw, 1992). It is estimated that 100,000–300,000 birds died from exposure to oil (Paine et al., 1996; Piatt et al., 1990). Pre- and post-spill data revealed that sea otter (*Enhydra lutris*) survival in the oiled portion of Prince William Sound (PWS) was significantly lower in the years following the spill (Peterson, 2003). In addition, high mortality rates were observed in animals at various trophic levels with chronic exposure to crude oil (Peterson, 2003).

Crude oil is comprised of a complex mixture of petroleum hydrocarbon and non-hydrocarbon compounds. Mixtures vary among different crude oils (see Table 1), resulting in multiple physical and chemical properties (Council, 2003). Monocyclic aromatic hydrocarbons (e.g., benzenes, toluenes, and xylenes) and phenols comprise the most acutely toxic components of fresh crude oil, but their high volatility limits their toxic effects to aquatic organisms (Council, 2003; Neff et al., 2000). Weathering crude oil forms 3–5 ringed polycyclic (or polynuclear) aromatic hydrocarbons (PAHs), which become the primary source of persistent toxicity at spill sites (Boehm and Page, 2007; Neff et al., 2000). Weathered oil in aquatic environments poses a significant threat to lifeforms, as toxic effects can cascade across trophic levels (Council, 2003; Gin et al., 2001; Peterson, 2003). Ecologically, crude oil can alter the structure and function of both freshwater and marine food webs (e.g., through mortality, retarded succession, and retrogression) (Paine et al., 1996; Shaw, 1992).

The Laurentian Great Lakes hold a significant portion of the world's fresh water. The Great Lakes–St. Lawrence Seaway (GL–SLS) is a busy trade artery, serving mining, farming, manufacturing, and commercial interests from the western prairies to the eastern seaboard (The St. Lawrence Seaway Management Corporation, 2013). Over 180 million metric tons are moved along the GL–SLS annually, and dominant commodities include iron ore, coal, limestone, grain, machinery, cement, and aggregates of salt and stone (The St. Lawrence Seaway Management Corporation, 2013). The economics of the GL–SLS are staggering, with an estimated total of $375 billion in exports from Canada and the United States (The St. Lawrence Seaway Management Corporation, 2013). Despite the size and complexity of the GL–SLS, the system has maintained a strong record of trouble-free navigation. With the sheer number of transit ships using this route annually, however, the threat of an oil spill is ever present, putting both vital shipping routes and the ecology of the Laurentian Great Lakes at constant risk.

While there are extensive reviews on the toxic effects of crude oil on individual trophic levels in the aquatic food web (e.g., Berrojalbiz et al., 2009; Burns et al., 1993; Carls et al., 2002; Poulton et al., 1997; Rice et al., 2001), few reviews have attempted to reconcile and integrate the knowledge gained at an ecosystem scale. Of particular interest, are the issue of environmental persistence, bioaccumulation, and trophic transfer of PAHs in aquatic food webs and their possible consequences. In this review, we adopt an integrated ecosystems approach to delve into the consequences of oil spills on aquatic food web interactions, highlighting the short- and long-term effects on the integrity of these ecosystems (see Fig. 1). We review the commonly exerted effects of crude oil on the structural and functional integrity of phytoplankton and zooplankton assemblages, pelagic and demersal fish, bacteria, and benthic communities. In addition, we present a thorough review of the modelling literature, highlighting the abiotic and biotic representations of oil spills in aquatic environments. We emphasize the lack of explicit biological representation in contemporary oil spill models, and make suggestions on how to fill this gap in the knowledge. We conclude with a Laurentian Great Lakes-centric discussion, underscoring the importance of, and need for robust oil spill management tools.

Phytoplankton

Phytoplankton are unicellular primary producers—often referred to as algae—which collectively form the base for the most spatially extensive food webs in nature. Negative anthropogenic stressors affecting algae, can propagate throughout the food web and impact aquatic life at all trophic levels. Existing knowledge on the toxic effects of crude oil and its constituents on phytoplankton assemblages is unclear and sometimes contradictory (Banks, 2007; Batten et al., 1998; Fiala and Delille, 1999; González et al., 2009; Sargian et al., 2005). Toxicity of crude oil is species-specific (Council, 2003; Fiala and Delille, 1999; Ostgaard et al., 1984; Varela et al., 2006), and varies with oil composition (see Table 1 for the physical properties and compositions of common crude oils). In the following sections, we review the impacts of crude oil and its constituents on phytoplankton growth, photosynthetic activity, and potential structural/functional alterations.

Biochemical effects of crude oil on algae growth and photosynthetic activity

There are numerous studies determining the effects of crude oil exposure on phytoplankton growth (Bate and Crafford, 1985; Kong et al., 2010; Pérez et al., 2010; Sargian et al., 2005; Singh and Gaur, 1988; Tukaj, 1987). In both laboratory cultures and natural phytoplankton assemblages, exposure to high concentrations of petroleum water-soluble fraction (WSF) has been observed to be toxic, while stimulatory effects have been reported at lower concentrations (El-Sheekh et al., 2000; Parab et al., 2008; Pérez et al., 2010; Siron et al., 1991). Time series data reported in Sheng et al. (2011) indicate chlorophyll-a concentrations decreased in the Northwest Shelf of Australia in the month following the Montara oil spill, but a strong resurgence (up to 1.5 times above average) in subsequent months. The authors hypothesized the initial chlorophyll-a reduction may have resulted from slick-induced solar radiation blockage, allowing for the proliferation of bacteria that eventually cleared up the hydrocarbons via decomposition. The combination of solar radiation and nutrients released from dead fauna may have sparked the phytoplankton resurgence (Sheng et al., 2011). Observations from the northeastern Gulf of Mexico three weeks after the Deepwater Horizon well was capped also indicate significantly higher phytoplankton biomass (Hu et al., 2011).

Algal response to oil spill events is both dose-dependent and species-specific (González et al., 2009; Pérez et al., 2010; Tukaj, 1987). For example, the effective concentration for 50% growth reduction (EC50) in *Phaeodactylum tricornutum* (diatom) and *Dunaliella tertiolecta* (green flagellate) in batch cultures was 16.4 and 36.0 mg L^{-1} WSF (fraction of hydrocarbon readily soluble in water), respectively (Siron et al., 1991). Romero-Lopez et al. (2012) tested strains of *Scenedesmus intermedius*, *Microcystis aeruginosa*, and *D. tertiolecta* to increasing levels

Table 1
Physical properties, hydrocarbon composition, and volatile organic compound composition of common crude oils. Fields denoted with asterisk were not measured.

	Physical properties						% Weight hydrocarbons					Volatile organic compounds (µg/g oil)						
	API gravity	Sulphur content (weight %)	Water content (volume %)	Flash point (°C)	Density (g/mL @ 15 °C)	Dynamic viscosity (cP @ 15 °C)	Saturates	Aromatics	Resins	Asphaltenes	Waxes	Benzene	Toluene	Ethylbenzene	Xylenes	c3-Benzenes	Total BTEX	Total BTEX and c3-Benzenes
Alaska North Slope Crude Oil	30.89	1.11	<0.1	<−8	0.8663	11.5	75	15	6	4	3	2866	5928	1319	6187	5620	16300	21920
Alberta Sweet Mixed Blend	35.72	0.63	<0.1	−4.3	0.8404	6.1	77.3	16.8	4.2	1.7	3.2	2261	5308	1646	8954	1240	18170	30570
Arabian Light	31.3	1.93	<0.1	<−10	0.8776	13	75.5	15.2	5.7	3.6	2.7	979	3050	1995	4927	8620	10950	19570
Sockeye Sour	19.32	4.51	0.8	−4	0.9465	761	49.2	17.2	15.1	18.5	1.6	1343	2031	974	3880	5810	8230	14040
South Louisiana	32.72	0.49	<0.1	<−10	0.8562	10.1	80.8	12.6	5.9	0.8	1.7	1598	3552	891	6164	6680	12210	18890
West Texas Intermediate	34.38	0.86	<0.1	<−10	0.8594	8.6	78.5	14.8	6	0.7	2.8	4026	7395	4845	7105	10190	23370	33560
Diesel	37.52	0.09	<0.1	54	0.831	2.76	88.2	10.2	1.7	0	1.7	136	1024	619	3774	13780	5550	19330
Bunker Fuel	11.55	1	3.1	94	0.9883	1410	44.2	39.5	8	8.4	2.3	0	149	124	612	1750	890	2640
Heavy Fuel Oil 6303	11.47	1.48	0.1	111	0.9888	22800	42.5	29	15.5	13	2.5	40	136	58	396	940	630	1570
Orimulsion-400	8.63	2.8	28	>320	1.0093	256	32.1	19.7	9.6	10.6	*	16	29	22	29	80	100	180

of petroleum and diesel exposure. Short term exposure effects were mainly limited to rapid photosynthetic inhibition. In the long term, however, exposure resulted in massive destruction of sensitive cells—though some cultures were able to grow due to selection of toxin-resistant cells. The authors concluded micro algae are able to survive petroleum contamination with physiological acclimatization with no genetic changes while contamination is under a physiological threshold. Once this threshold is surpassed, however, survival may depend exclusively on mutations conferring resistance, and selection of these mutations (Romero-Lopez et al., 2012). Dose dependent observations were also reported in studies testing oil sensitivity in *P. tricornutum* (Bate and Crafford, 1985), and *Chlorella vulgaris* (El-Sheekh et al., 2000). Karydis (1981) linked diatom oil sensitivity to the presence of silica in frustules. He found crude oil to exert less severe impacts in silicon limited media, suggesting the adsorptive properties of silica in diatom frustules modulate the uptake and intracellular distribution of hydrocarbons. More recently, Hook and Osborn (2012) compared the toxicity of WSF, chemically enhanced WSF mixture (i.e., oil mixed with dispersant), and dispersant on *P. tricornutum*, and found a significantly higher toxic response in cells exposed to chemically enhanced oil and dispersant. Exposure to chemically-enhanced WSF and dispersant caused membrane damage, but WSF exposure did not (Hook and Osborn, 2012).

Hydrocarbon toxicity has been shown to affect phytoplankton cell size. Pérez et al. (2010) observed increased average cell diameters and growth rate reductions in *Isochrysis galbana*, following oil exposure. The authors suggest fuel exposure may affect cell division mechanisms, but not the production of new cell material. They further suggest reduced growth rates may be resultant of cell cycle prolongation, rather than increased mortality (Pérez et al., 2010). In studying the impacts of crude oil on natural estuarine phytoplankton assemblages, Sargian et al. (2005) drew similar conclusions, suggesting reduced growth rates and increased mean cell sizes resulted from cell cycle perturbations. Studying the algal nucleic acid response following crude oil exposure, El-Sheekh et al. (2000) observed increases in both RNA and DNA at low oil concentrations. These trends quickly reversed as fuel concentrations increased, eliciting a stronger negative response from DNA than RNA. Similarly, Parab et al. (2008) observed higher than normal RNA: DNA ratios, and high protein production in algae exposed to low oil concentrations. They point out that a high RNA:DNA ratio is indicative of cell/organism growth, and suggest the algal response at low levels of oil exposure may be stimulatory, or carcinogenic/mutagenic (Parab et al., 2008).

The inhibitory effects of petroleum hydrocarbons and their constituents on phytoplankton photosynthetic activity is well documented (Armstrong and Calder, 1978; Ostgaard et al., 1984; Pérez et al., 2010; Singh and Gaur, 1988). Exposure of oceanic and coastal marine phytoplankton assemblages to various concentrations of crude oil WSF yielded reduced photosynthetic activity and chlorophyll-a concentrations (González et al., 2009). Marwood et al. (1999) argue algal PAH toxicity may manifest in the form of membrane damage. Hydrocarbon-driven destruction of plasma membranes can disturb ion balances, reduce intracellular pressure, and decrease phycoerythrin and chlorophyll concentrations (Stepaniyan, 2008). Additional toxicity was observed in the form of photo-driven production of free radicals (Kelly et al., 1998; Sikkema et al., 1995). A potent oxidant, the hydroxyl (OH⁻) free radical reacts indiscriminately with lipids, DNA, and proteins, leading to the oxidation of these molecules (Kelly et al., 1998). PAH toxicity is further accentuated by solar radiation, as photomodified PAHs are more soluble than their parent compounds (Duxbury et al., 1997; McConkey et al., 1997). Furthermore, select PAHs may react with light to produce phytotoxic oxygenation compounds such as quinones, hydroxylated quinones and benzoic acids (Huang et al., 1997; Marwood et al., 2003; McConkey et al., 1997; Sargian et al., 2005). It is important to note that complex mixtures of PAHs can yield convoluted and, often times, difficult to quantify reactions to light. Armstrong and Calder (1978) hypothesized exposure of microalgae to oil may impair
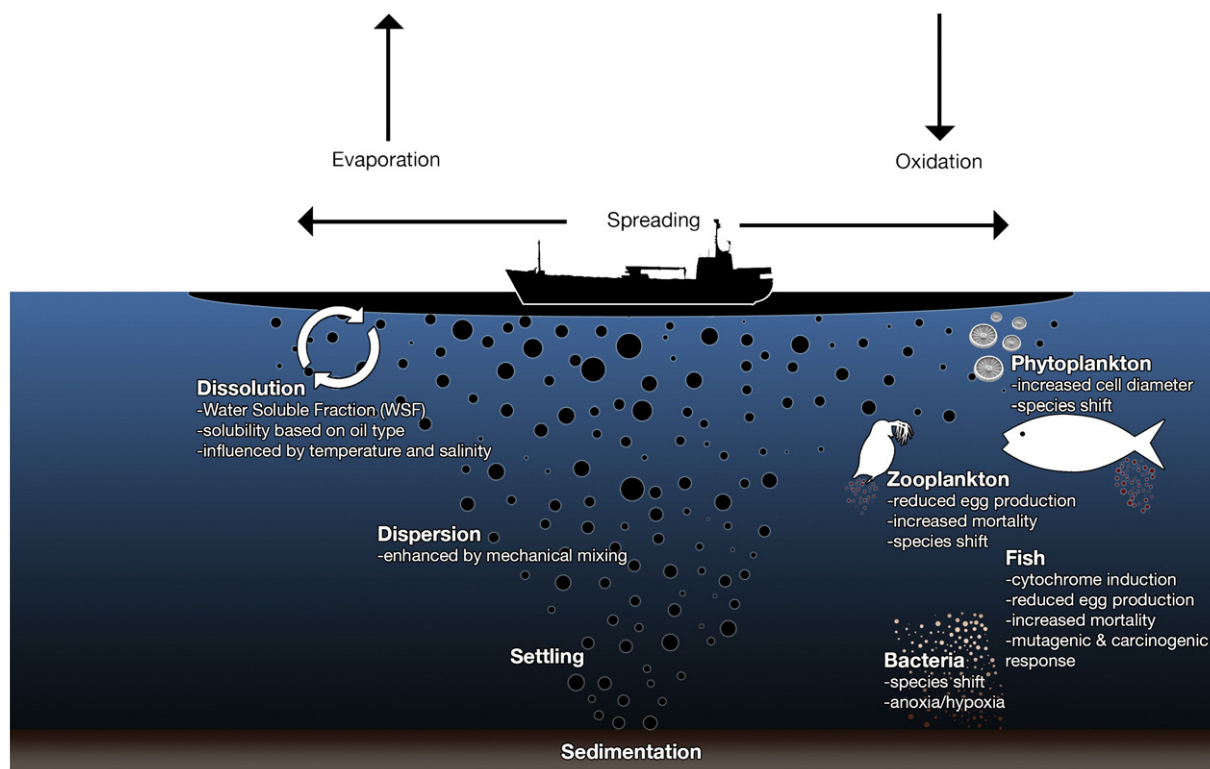
Fig. 1. Conceptual diagram of physical and ecological processes following a petroleum hydrocarbon spill in open waters.

energy-yielding metabolic processes through electron transport system interference. Photomodified anthracene (a PAH consisting of three fused benzene rings) was found to inhibit photosynthesis both in vivo and in vitro (Huang et al., 1997). More specifically, photosystem I (PSI) was directly inhibited by photomodified anthracene, and electron transport from photosystem II (PSII) was blocked, causing excitation pressure in PSII (Huang et al., 1997). Under normal circumstances, PSII absorbs photons of 680 nm wavelength to oxidize two molecules of water into one molecule of molecular oxygen. The 4 electrons removed from the water molecules are transferred by an electron transport chain to ultimately reduce 2NADP$^+$ to 2NADPH. This creates a proton gradient across the thylakoid membrane and drives ATP (adenosine triphosphate) generation, and transports electrons to PSI via intermediary proteins. Exposure of *Lemna gibba* plants to photomodified anthracene, however, yielded reduced photosynthesis and chlorophyll-a fluorescence kinetics (photons re-emitted after being absorbed by chlorophyll) indicating electron transport interference at or near PSI and PSII (Huang et al., 1997).

Structural and functional responses to crude oil exposure

Exposure to crude oil elicits variable responses from phytoplankton, potentially altering both structure (i.e., physical construction of cells), and function (i.e., the role in the food web) (Shaw, 1992). In oil exposure microcosm studies, nanoflagellate biomass of an oceanic assemblage increased, while picophytoplankton biomass decreased. Specifically, this was driven by severe reductions and the eventual disappearance of *Prochlorococcus* and *Synechococcus* from both low and high WSF concentration treatments (González et al., 2009). In microcosm studies examining the effects of oil from the DHOS, Gilde and Pinckney (2012) observed decreasing total phytoplankton biomass with increasing crude oil concentrations. Prasinophytes and cryptophytes showed a significant negative response, while diatoms, euglenophytes, and chlorophytes remained relatively resistant at the concentrations tested; cyanophyte relative abundance, however, increased (Gilde and Pinckney, 2012).

Several studies suggest cell size as an important factor in PAH toxicity (Echeveste et al., 2010; Fan and Reinfelder, 2003; González et al., 2009). In culture and natural phytoplankton assemblages, the picocyanobacteria *Prochlorococcus* and *Synechococcus* were found to be highly sensitive to the hydrocarbons pyrene and phenanthrene, compared to the larger *Thalassiosira* species (Echeveste et al., 2010). Fan and Reinfelder (2003) observed uptake of phenanthrene among the *Thalassiosira* species to also be size dependent. Namely, uptake was 2–3 times greater in the smaller *T. pseudonana* than in *T. weissflogii*, reflecting the 2.8-fold difference in surface area-to-cell volume ratio. These observations support Del Vento and Dachs (2002), who found shape was secondary to size in microorganism persistent organic pollutant (POP) uptake.

Hjorth et al. (2007) used a food web approach to comprehend the direct and indirect effects of pyrene on the structure and function of bacteria, phytoplankton, and zooplankton assemblages in a mesocosm environment. While direct and immediate effects were observed in the phytoplankton community, no immediate discernible effects were observed in zooplankton, and a bacterial lag of approximately 48 h was observed. Phytoplankton function (i.e., primary production) was least affected, attributed to functional redundancy, whereby opportunistic phytoplankton species took over the roles of negatively affected species (see Table 2 in Hjorth et al. (2007), showing changes in dominant phytoplankton species across different days at different pyrene exposure concentrations). Manifestations of phytoplankton stress response to toxicants may also be nutrient-dependent (Hjorth et al., 2008; Interlandi, 2002; Karydis, 1981; Kong et al., 2010). Roessink et al. (2008) divided nutrient–organic micro-pollutant interactions in aquatic ecosystems into four categories. The first interaction is dilution of toxicant by biomass. The environmental fate of a toxicant in conjunction with a nutrient level shift may result in higher biomass organisms, lowering internal exposure (Skei et al., 2000). The second interaction is nutrient impact on toxicant transport. An increase in nutrient levels may lead to increased phytoplankton production, and possibly eutrophication.

This phytoplankton bloom increases the flow of detritus to the sediment, increasing the sedimentation of toxicants (Skei et al., 2000). The third interaction covers direct toxicant–food interactions. Pieters et al. (2005) observed insecticide toxicity was 2–3 times higher in *Daphnia magna* under low nutrient status (and low phytoplankton abundance) than under high nutrient status. Finally, the fourth interaction covers indirect toxicant–food interactions. Jak et al. (1998), for example, showed eutrophication symptoms can be triggered at lower nutrient levels in the presence of organic micro-pollutants, due to the reduction of plankton grazers. Specific examples of nitrogen– and phosphorus–toxicant interactions are provided below.

Environmental nitrogen availability may affect the bio-concentration of hydrophobic organic compounds (HOCs). Halling-Sørensen et al. (2000) found a disproportionate increase in HOCs as algal lipid concentrations increased in response to reduced nitrogen availability. Biological concentration factors (BCF represents the concentration of a particular chemical in biological tissue per concentration of the same chemical in the surrounding water) for HOCs increased 9-fold as the total algal lipid content of the green algae *Selenastrum carpriconutum* increased from 17 to 44% of algal dry weight due to nitrogen-starvation (Halling-Sørensen et al., 2000). Previous studies had also linked nitrogen starvation to altered lipid synthesis. Tornabene et al. (1983), for example, observed lipid fractions of 36–54% dry cell weight in nitrogen starved *Neochloris oleoabundans*, with up to 80% triglyceride lipid fraction. Increasing nitrogen availability resulted in the formation of polar lipids, dominated by polyunsaturated C16 and C18 fatty acids, although species-specific differences were noted (Piorreck et al., 1984). Other studies report nutrient deficiencies may be lethal in combination with crude oil. Karydis (1981), for example, observed inhibitory effects in the diatom *Skeletonema costatum* occurred faster when Tunisian crude oil was introduced to phosphorus rather than nitrogen deficient media. In the same study, phosphorus deficiency more strongly affected cellular chlorophyll-a content than nitrogen deficiency. Nutrient additions made minimal impact to algal community structure, but the addition of pyrene led to a short-term increase in diatom abundance (68% of total phytoplankton biomass) and decreases in both dinoflagellate and cryptophyte abundances (Hjorth et al., 2008). These results, however, contradict previous findings in which diatoms were observed to be negatively sensitive to PAH exposure (Sargian et al., 2005).

Summary of phytoplankton effects

The literature shows that in addition to physical interference (e.g., surface slick induced light attenuation, gas exchange interference), a floating mass of crude oil and its constituents, especially PAHs, can significantly impact phytoplankton. The application of chemical dispersants has been shown to yield more toxic effects than naturally weathering crude oil. In addition, response to crude oil is species dependent, varies with oil composition, and can be stimulatory to growth in small concentrations. As concentrations increase, regardless of chemical profile, the toxic impacts become apparent in the forms of increased cell diameter and reduced cell division, lower chlorophyll-a concentrations, and reduced photosynthetic activity resultant of electron chain transport interference in PSI and PSII. In addition, exposure to oil may induce changes in community structure, but not necessarily function, depending on community complexity and functional redundancy. Finally, interactions between nutrients and toxic oil constituents may alter lipid biosynthesis and partitioning, influencing bioaccumulation of hydrophobic oil constituents and eventual transfer of these toxicants to higher trophic levels.

Zooplankton

Zooplankton link primary producers to higher organisms, and are critical vectors in marine and freshwater food webs (Saiz et al., 2007). Zooplankton grazing, growth, and mortality influence the biogeochemical cycling of nitrogen and phosphorus (Alcaraz et al., 2010; Turner, 2002). Contaminants interfering with these processes may significantly disrupt the flows of mass and energy in aquatic ecosystems. Several studies have reported high microfaunal mortality rates resultant of exposure to crude oil and its constituents (e.g., *Oithona davisae*: Barata et al., 2005; nematodes: Danovaro et al., 1995; *Daphnia middendorfiiana*: Federle et al., 1979; O'Brien, 1978; *Temora longicornis*: Teal and Howarth, 1984). In the following sections, we discuss the toxic effects of oil contaminant uptake, elimination, and trophic transfer in zooplankton communities.

Uptake and elimination

Initial exposure of zooplankton to oil constituents is followed by rapid dilution, until an equilibrium state is established (Landrum et al., 2003; Lotufo, 1998). Smaller species (e.g., *Eurytemora affinis*) were found to accumulate more ^{14}C-1-naphthalene than larger *Calanus helgolandicus* species (Harris et al., 1977). While both passive diffusion (e.g., Sobek et al., 2006, 2010), and feeding (e.g., Corner et al., 1976; Magnusson and Tiselius, 2010; Magnusson et al., 2007) have been observed as HOC uptake mechanisms, the literature remains divided on the relative importance of each. Cailleaud et al. (2009) illustrated diffusive uptake, showing a significant somatic PAH increase in the estuarine copepod *E. affinis* when exposed to dissolved PAHs in a flow through experiment (613 ng g^{-1} vs. 9.5 ng g^{-1} dry weight in non-exposed individuals). Berrojalbiz et al. (2009) used *Rhodomonas salina* (cryptophyte) and *Paracartia grani* (copepod) in laboratory experiments to study the accumulation and cycling of PAHs in zooplankton. They concluded passive partitioning dominated PAH accumulation in zooplankton, regardless of uptake mechanism. Jensen et al. (2012) performed laboratory exposure experiments on *Calanus finmarchicus* studying the diffusive uptake of phenanthrene and benzo(a)pyrene. They found the lighter PAH compound (i.e., phenanthrene) accumulated more quickly, and reached steady state within 96 h. Benzo(a)pyrene—a heavier compound—accumulated more slowly, reaching steady state after 192 h. This bias towards uptake of lighter molecules may have contributed to the observations of Froehner et al. (2011), who found an absence of low molecular weight PAHs, and an abundance of high molecular weight PAHs in sediment samples collected from southern Brazil.

In addition to PAH consumption via contaminated food and diffusive uptake, zooplankton have also been observed to directly ingest oil (Conover, 1971; Council, 1985). Following the Arrow tanker oil spill (Nova Scotia, Canada, 1970), it was estimated that 10% of the Bunker C oil in the water column was consumed directly by zooplankton (Conover, 1971). *C. finmarchicus* ingested approximately 5×10^{-4} g of oil per day, per individual, sedimenting approximately 3 tons of oil per day within an area of 1 km^2 of oceanic waters (Muschenheim and Lee, 2002). Zooplankton species in the Gulf of Mexico following the DHOS were observed to ingest dispersed oil droplets (1–30 μm in diameter) (Lee et al., 2012). Subsequently released fecal pellets contained numerous oil droplets, carrying an estimated 200 μg m^{-3} of oil to the sediments (Lee et al., 2012).

The predominant modes of PAH elimination from zooplankton are diffusive depuration, metabolism, fecal pellets, and egg production. Berrojalbiz et al. (2009) suggest that up to 90% of the PAHs accumulated by *C. helgolandicus* may rapidly depurate once the copepod is transferred to uncontaminated water. Fecal pellets are an elimination pathway contributing significantly to the packaging, elimination, and sedimentary flux of petroleum hydrocarbons in both marine and freshwater ecosystems (Prahl and Carpenter, 1979). Elimination via defacation may be more predominant when PAH uptake is through contaminated food, rather than diffusion (Berrojalbiz et al., 2009). Some researchers, however, downplay the net fecal PAH flux to the sediments, arguing zooplankton fecal pellets are mostly recycled by bacteria, and organic rain is comprised mainly of aggregated phytoplankton (Turner, 2002). Another important elimination pathway is egg production, as female copepods have been observed to eliminate PCBs twice as fast as males

(McManus et al., 1983). Similarly, fluoranthene (a PAH) somatic concentrations in *Coullana* sp. changed dramatically during reproductive cycles, such that 50% of the total fluoranthene was stored in lipid-rich maturing eggs (Lotufo, 1998). Dachs et al. (1996) observed lower molecular weight compounds covaried with organic carbon and fecal pellets fluxes, suggesting lower molecular weight compounds are more readily eliminated than heavy compounds. Thus, the fate of PAHs following intake by zooplankton depends on multiple factors, including PAH form (i.e., particulate or dissolved), bacterial involvement, and zooplankter gender/sexual maturity (egg contamination).

Toxicity of oil on eggs

The extent of crude oil toxicity in zooplankton is modulated by species, and developmental stage. The early stages of zooplankton development (i.e., fertilization, embryonic stage, hatching, and larval phases) are particularly susceptible (Council, 1985; George et al., 1998). For example, hydrocarbon contamination of *Acartia pacifica* resting eggs reduced the number of emerging nauplii by 3.8–100% when Fuel Oil #0 concentrations were increased from 50 mg kg^{-1} to 5000 mg kg^{-1} (Jiang et al., 2008). Exposure of starved *Calanus* spp. to pyrene yielded no reduction in egg production (Jensen et al., 2008). However, in the same experiment, feeding individuals experienced significant reductions in both grazing and egg production, indicating limited pyrene uptake through passive diffusion. Exposure to 10–80 parts per billion (ppb) of south Louisianna crude oil did not significantly reduce the rate of egg production in *Centropages hamatus*, but had deleterious effects on the hatching success of the eggs (Cowles and Remillard, 1983). Olsen et al. (2013) found *C. finmarchicus* egg production to be very low with high dispersed oil concentrations, and subsequent improvement with the removal of the dispersed oil. Though the removal of dispersed oil improved egg production rate, only a small portion of the exposed females participated in egg production (Olsen et al., 2013). Bellas and Thor (2007) reported both lethal and sublethal effects on egg production rate, hatching, recruitment and survival of *Acartia tonsa* exposed to different types and levels of PAHs, and concluded that egg production rate was a more sensitive and appropriate toxicity endpoint measure in zooplankton than mortality.

It has been suggested that lack of hydrocarbon accumulation in zooplankton and the loss of egg viability could be associated with: (i) incorporation of toxic hydrocarbon components or their altered metabolites into oocytes, or (ii) altered biosynthetic pathways involved in oogenesis as a result of exposure to hydrocarbons (Cowles and Remillard, 1983). Capuzzo et al. (1984) observed modifications in lipid biosynthesis in larval lobsters exposed to sub-lethal concentrations of crude oil, whereby oil-exposed lobster larvae had lower levels of major energy storage lipids (triacylglycerols), and higher levels of sterols. This pattern was linked to the energetic disruptions in lobster larvae observed in the study, and the authors concluded oil exposure may trigger developmental abnormalities (Capuzzo et al., 1984). Similarly, increased mobilization of energy stores was reported in decapod *Microbrachium borellii* eggs, following exposure to sublethal crude oil concentrations (Lavarias et al., 2006). Studies with *C. finmarchicus* suggest PAH toxicity could affect egg and sperm production via lipid peroxidation (Hansen et al., 2008; Saiz et al., 2009). Further, reduced egg hatching success following PAH exposure could be linked to lipid peroxidation (Bellas and Thor, 2007; Jensen et al., 2008), while reduced fecundity may be the result of maternal malnutrition (e.g., toxicity-mediated reduction in feeding; see Saiz et al., 2009).

Trophic transfer of crude oil constituents

The lower aquatic food web was traditionally thought to accumulate high concentrations of PAHs and HOCs that are eventually transferred to higher consumers (Wan et al., 2007; Xinhong and Wen-Xiong, 2006). Lotufo (1998), however, argues that the link between contaminated zooplankton and adverse effects in higher predators is debatable. In a study investigating the accumulation and transfer of PAHs from

sediments to bluegill (*Lepomis macrochirus*) via *Chironomus riparius*, Clements et al. (1994) observed rapid PAH accumulation in *C. riparius*, but very low bluegill somatic PAH concentrations. This may be indicative of highly inefficient PAH transfer from invertebrates to fish (Niimi and Dookhran, 1989). In a related study, however, Woodin et al. (1997) reported more complex results when physiological responses were examined, with the most profound being related to cytochrome activity. Cytochromes are rate-limiting factors in oxidative metabolic processes, and are responsible for approximately 75% of all metabolic reactions in animals (Guengerich, 2008). The authors subjected *Anoplarchus purpurescens* to controlled laboratory conditions approximating oiled sites following the EVOS. Woodin et al. (1997) found a 49-fold increase in cytochrome P4501A in individuals subjected to oiled conditions, but this rapidly fell to baseline levels when placed in non-oiled surroundings. Along the same lines, DuLacoste et al. (2013) tested multiple exposure pathways, and observed no PAH bioaccumulation in juvenile turbot (*Scophthalmus maximus*). Individuals were exposed for four days, followed by a six-day depuration period. PAH concentrations in the liver and muscles peaked at the onset of exposure, but fell to background levels by the end of the experiment. James et al. (2001) found the catfish *Ictalurus punctatus* to readily conjugate PAHs (specifically 3-OH-BaP; BaP: benzo(a)pyrene) in the intestine. The conjugated metabolites were secreted into the intestinal contents, and very little unchanged 3-OH-BaP was found in the blood stream. The authors assert this may be evidence of the preferential uptake of conjugated metabolites into the blood stream (James et al., 2001). Further, the Woodin et al. (1997) study reported varied biological responses based on route of exposure. For example, the authors observed CYP1A induction in intestinal mucosal epithelial and endothelial cells when individuals were fed oiled food, with relatively low levels of CYP1A in liver, gill, and gonadal cells. (CYP1A induction is a response in fish exposed to xenobiotics, including petroleum hydrocarbons). Conversely, when individuals were in close proximity to oiled sediments, CYP1A was strongly induced in endothelial cells, and all examined organs. Jönsson et al. (2006) suggest the apical membrane of gill epithelial cells minimize the uptake of waterborne organic compounds, yielding a first pass metabolism that protects intracellular environments—including the CYP1A system.

While PAHs are transferred to higher trophic organisms via consumption of PAH laden organisms, the literature suggests this form of exposure is likely at levels that can be dealt with through first pass metabolism. First pass metabolism would greatly reduce xenobiotic concentration during absorption before it reaches the circulatory system. More recently, Wan et al. (2007) reported significant negative relationships between trophic level and lipid-normalized concentrations for ten PAH compounds. The authors concluded PAH attenuation up the food chain can be attributed to low assimilation efficiencies and efficient metabolic transformations in higher species. In his review, Livingstone (1998) related faster BaP metabolization in fish (relative to invertebrates) to higher levels of total cytochrome P450. Additional attenuation may stem from biotransformations in lower organisms. Harris et al. (1977) found copepods exposed to radio-labeled naphthalene for several days contained a considerable proportion of radioactivity that was no longer identifiable as naphthalene, suggesting biotransformation. Similar findings have been reported in other species (see Lotufo, 1998). *E. affinis* have been found to exhibit PAH compound selection, whereby higher molecular weight PAHs are accumulated while lower molecular weight compounds are preferentially eliminated (Cailleaud et al., 2009). Other studies have drawn similar conclusions, reinforcing the notion that the elimination rates were inversely proportional to compound hydrophobicity (Landrum, 1988; Lydy et al., 1992).

Summary of trophic transfer in lower food web animals

Zooplankton and zoobenthos are important intermediate links in the lower food web, connecting primary producers to higher trophic levels. The deleterious impacts of hydrocarbon spills, such as increased

zooplankton mortality and reduced egg production/viability, can potentially alter food web structure. The use of chemical dispersants was found to significantly reduce zooplankton community abundance, highlighting the damaging nature of chemical dispersants. The literature is split between passive diffusion and feeding as being the primary toxin uptake pathways. Several studies report high mortality rates with hydrocarbon contamination, but some researchers argue that zooplankton egg production is more sensitive, and therefore a more appropriate endpoint measure to toxicity than mortality. Hydrocarbons can inhibit zooplankton egg production by incorporating toxins into oocytes, and altering the biosynthetic pathways involved in oogenesis. Toxic elimination mechanisms include diffusive depuration, metabolism, and defecation. Studies show low molecular weight PCBs and PAHs are preferentially eliminated, and that elimination is inversely proportional to compound hydrophobicity.

Fish

Numerous studies have demonstrated the toxic effects of crude oil and its constituents on fish (Balch et al., 1995; Carls et al., 2005, 2008; Hose et al., 1996; Lee and Anderson, 2005; Marty et al., 2003; Rice et al., 2001). Fish take up petroleum hydrocarbons at the gills, through food intake, and other body epithelia (Council, 1985; Lee et al., 1972). In pink salmon (*Oncorhynchus gorbuscha*), hydrocarbon exposure triggers CYP1A induction in embryos, and has been linked to adverse cellular, organism, and population level effects (Brannon et al., 2006; Carls and Thedinga, 2010; Carls et al., 2005). Similar findings have been reported in other species (see Hose et al., 1996 and references therein). In the following sections, we review the impacts of crude oil on fish well-being across species and developmental stage, and look into the metabolic breakdowns of PAHs in fish digestive systems.

Uptake and accumulation of hydrocarbons

Fish accumulation of soluble hydrocarbons is affected by various biological, chemical, and physical factors, continuing until a metabolic equilibrium in the liver is reached (Collier et al., 1995; Lee et al., 1972). Natural stressors (e.g., salinity, pH, temperature, and food abundance) may predispose fish to heightened hydrocarbon sensitivity (Council, 1985). Ramachandran et al. (2006) observed PAH exposure varied inversely with salinity, suggesting limited bioavailability and uptake of PAHs in highly saline environments. The authors note that highly saline environments can reduce the solubility of PAHs and the effectiveness of chemical dispersants. Similar findings were reported by Whitehouse (1984). These observations suggest sites of low salinity (e.g., estuarine and nearshore habitats that serve as spawning ground for many fish) may be more sensitive. In addition to salinity, studies show temperature also modulates PAH solubility. Temperature affects the persistence of hydrocarbons in water, and exerts physiological stress on fish in environments outside their normal temperature range (Council, 1985). Whitehouse (1984) observed PAH solubility increased with temperature, and noted that solubility was more sensitive to changes in temperature than salinity. These physicochemical interactions can impact all trophic levels, affecting fish primarily through the gills, food intake, and other body epithelia (Council, 1985; Lee et al., 1972). The metabolic breakdown of PAHs can damage fish, as PAH metabolites can be more damaging than their parent compounds (Livingstone, 1998). Specifically, PAHs metabolites can form covalent bonds with somatic molecules such as proteins, DNA and RNA, resulting in cellular damage, mutagenesis, teratogenesis, and carcinogenesis (Tuvikene, 1995). As such, deposit feeding polychaetes with the capacity to biotransform PAHs could result in more soluble toxins. Juvenile English sole (*Pleuronectes vetulus*) feeding on *Armandia brevis* (deposit-feeding polychaete) exposed to BaP showed reduced growth, increased expression of CYP1A, and evidence of hepatic PAH-DNA adduct formation (i.e., pre-carcinogenic activity) (Rice et al., 2000). When the predatory polychaete *Nereis virens* fed on other closely-related polychaetes (i.e., *Capitella* sp. *I* and *Capitella* sp. *S*), *N. Virens* accumulated significantly more fluoranthene through *Capitella* sp. *I* (a high biotransformer), compared to *Capitella* sp. *S* (a limited biotransformer) (Palmqvist et al., 2006). In a study gauging the trophic transfer potential of PAH metabolites from infaunal organisms to bottom-feeding fish, McElroy and Sisson (1989) observed PAH metabolites accumulated from a diet of polychaetes were further biotransformed by fish. Biotransformation may explain the trophic dilution (i.e., reduced bioconcentrations with increasing trophic level) reported by Wan et al. (2007).

Toxicity of crude oil constituents to fish

The literature shows a great deal of variety in determining the effects of crude oil on fish developmental stages. Early studies suggested fish eggs and embryos were tolerant to the toxic effects of oil (Moles et al., 1979). Brannon et al. (1995) for example, report equally high pink salmon (*O. gorbuscha*) egg viability in both oiled and reference streams following the EVOS. The authors also report high survival rates for fry and juveniles in both oiled and reference streams during the spill year. More recently, Brannon et al. (2006) highlighted the importance of oil concentration in determining the toxicity to early life stages of fish. The authors observed no evidence of toxicity in pink salmon embryos exposed to laboratory or naturally weathered oil until total PAH concentrations exceeded 1500 and 2250 parts per million (ppm), respectively. Along the same lines, other studies have also found early developmental stages of fish to be the most vulnerable to oil contamination (Carls and Thedinga, 2010; Carls et al., 2005; Heintz et al., 1999; Rice et al., 2001; Short, 2003). Hose et al. (1996) for example, observed significantly higher morphological deformities and cytogenic abnormalities among newly hatched Pacific herring (*Clupea pallasi*) larvae from oil-exposed eggs, than those from un-oiled areas. Shen et al. (2012) also concluded early life stages were significantly more sensitive to contamination when they tested the effects of crude and fuel oil exposure on three *Acanthopagrus schlegelii* life stages. They concluded the life-stage dependent response may be due to the different body structures and behaviors associated with the different stages tested (Shen et al., 2012). Carls et al. (2002) estimated that 25–32% of Pacific herring embryos were damaged in PWS after the EVOS (based on an effects threshold of 0.4–0.7 μg L^{-1} total PAHs). Developmental delay was also observed in pink salmon following exposure to dissolved PAHs in the forms of delayed hatching and yolk absorption. Other symptoms included mortality, edema and anemia (Carls and Thedinga, 2010). Zhang et al. (2012) also observed (pericardial) edema, in addition to cardiac looping defects in pyrene treated *Danio rerio*. They concluded embryonic exposure to even low level environmental pyrene can disrupt cardiac development (Zhang et al., 2012).

Long-term exposure of eggs and embryos to highly weathered oil containing 3 to 4-ringed aromatic hydrocarbons can injure embryos and adversely affect survival (Short, 2003). Rice et al. (2001) found the composition of dissolved PAHs in seawater shifts from small 1- and 2-ringed, to larger 3- and 4-ringed PAH structures, as the smaller molecules biodegrade and undergo dissolution more rapidly. Thus, incubating eggs with long-term PAH exposure may sequester harmful 3- and 4-ring PAHs into lipid stores (Carls et al., 1999; Heintz et al., 1999; Marty et al., 1997; Rice et al., 2001; Short, 2003). In addition, emerging evidence suggests fish exposed to oil in early developmental stages may experience delayed development later in life. In pink salmon, adult individuals exposed to PAHs as embryos showed marked declines in survival rate compared to control individuals (Heintz et al., 2000). Bue et al. (1998) made similar observations in collecting gametes from adult pink salmon returning from contaminated and uncontaminated streams. They found significantly higher mortality in embryos from oil contaminated lineages.

The toxicity of spilled oil may not subside as it weathers and disperses, but rather increase. Whitehead et al. (2012) report results from a field study tracking the effects of the DHOS on killifish (*Fundulus*

grandis). Their data suggests heavily weathered crude oil from the spill imparted significant biological impacts in sensitive Louisiana marshes, some of which remained for over 2 months following initial exposure (Whitehead et al., 2012). A 2001 survey of PWS shorelines revealed over 55,000 kg of weathered oil from the EVOS, indicating a decay rate of 20–26% per year (Peterson, 2003). Blumer et al. (1973) observed consistently rapid initial losses of the lowest boiling point crude oil components. Less volatile components are the high ring number aromatics which are abundant in petroleum. These are virtually unaffected by evaporation, and responsible for long-term toxicity (Blumer et al., 1973). Claireaux et al. (2013) examined and compared the exposure effects of untreated Arabian light crude oil, chemically dispersed Arabian light crude oil, and chemical dispersant on the environmental adaptability of the European sea bass *Dicentrarchus labrax*. In mesocosms approximating conditions under an oil slick in shallow water, they found chemically dispersed oil to result in the lowest growth rate. Similar results were reported by Kuhl et al. (2013), who found dispersed oil and dispersant both to be acutely toxic for 1–4 weeks, with toxicity inversely related to salinity, suggesting reduced biodegradation of toxic components in low saline environments. Complicating matters is the fact that developmental toxicity of complex PAH mixtures is not necessarily additive (Billiard et al., 2008). This is especially troubling for current models and management tools estimating risk using dose- or concentration-dependence.

Exposure to crude oil increases petroleum hydrocarbon metabolization via the AhR pathway in fish livers (Lensu et al., 2011), and triggers increased activity of the mixed function oxidase (MFO) system (Lee and Page, 1997; Marty et al., 2003; Neff, 2002). (The AhR or Aryl hydrocarbon Receptor protein regulates biological response to aromatic hydrocarbons. With its ability to bind to a wide array of chemicals, AhR can facilitate their biotransformation and elimination.) MFO are a family of oxidase enzymes that catalyze a reaction in which each of the two atoms in O_2 is used for a different function in the reaction (National Library of Medicine, 2011). The AhR is a ligand-activated transcription factor which provides a molecular pathway by which endogenous and environmental signals can influence immune response (Quintana, 2012). Genetic research on small mammals suggests the AhR pathway mediates an adaptive toxic response, whereby xenobiotic compounds are metabolized and detoxified (Incardona et al., 2005). The same research, however, also uncovered toxic AhR-mediated responses, where receptor activation yielded negative impacts in exposed individuals. Toxic response in the AhR pathway can occur with AhR ligands that are poor substrates for CYP enzymes. Ahr ligands that are metabolically resistant tend to accumulate in tissues, and continually activate the AhR pathway, potentially yielding genotoxicity, mutation, and tumor initiation (Nebert, 2004). Byproducts of AhR-mediated metabolism accumulate in bile, and are excreted through feces and urine (Lee and Page, 1997). As such, the presence of hydrocarbon metabolites or fluorescent hydrocarbon compounds in fish bile is indicative of hydrocarbon contamination (Aas et al., 2000; Marty et al., 1999, 2003; Rice et al., 2001). Incardona et al. (2009) found crude oil exposure on zebrafish (*D. rerio*) and Pacific herring (*C. pallasii*) to yield an array of cardiac issues, including cardiogenic edema and arrhythmia. The authors concluded that the developing heart is the primary target of crude oil exposure. Exposure of Atlantic cod (*Gadus morhua*) to North Sea crude oil yielded DNA-adduct formation (i.e., carcinogenic activity) at high PAH exposure levels (1 ppm) (Aas et al., 2000). Balch et al. (1995) note that petroleum hydrocarbons can induce both carcinogenic and mutagenic responses in fish. Following the Amoco Cadiz oil spill, histological lesions were observed in the ovaries, kidneys and gills of plaice (*pleuronectes platessa*) in heavily hydrocarbon-contaminated shallow subtidal regions (Lee and Page, 1997). Demersal quillback rockfish (*Sebastes maliger*) livers exhibited abnormal enlargement of liver cell nuclei and also the occurrence of a large number of macrocytes in circulating blood following exposure from the EVOS (Marty et al., 2003).

Summary of fish impacts

The literature contains many studies demonstrating the toxic effects of crude oil on fish. It is generally accepted that soluble hydrocarbon accumulation takes place through the gills, as they have a large surface area and are rich in lipids. But studies of bottom dwelling fish report similarities between gut and sediment toxins, making food intake an equally important pathway, especially given the low water solubility of many PAHs. Exposure to petroleum hydrocarbons and other xenobiotics in fish elicits metabolic activity via the AhR pathway, inducing CYP1A. It has been shown that fish may be able to clear assimilated toxins via these metabolic pathways, but at a rate lower than accumulation. The toxic effects of hydrocarbons on fish include delayed growth, reduced survivorship, caused misdevelopment, and the induced of carcinogenic and mutagenic activity. These responses are accentuated when exposure occurs at early life stages, and are tightly linked with PAH derivatives, metabolites, and chemically dispersed oil.

Impacts on other biota

Following an oil spill, aquatic sediments can act as a sink for oil and its constituents (Lee and Page, 1997). When introduced to water, PAHs are adsorbed to particulate organic matter (POM), allowing them to disperse and sink (Danovaro, 2000). The sedimentation rate of oil and its constituents depends on: oil quantity, bathymetry, and the hydrodynamics of the spill site (Gesteira and Dauvin, 2005; Lee and Page, 1997; Olsen et al., 2007). Bacterial and benthic invertebrate response to sedimenting oil varies considerably among, and within genera and species, and depending on developmental stage (Capuzzo, 1985; Council, 1985). In the following sections, we review bacterial oil spill response, and the factors influencing accumulation of PAHs by benthic organisms, their response to oil exposure, and subsequent alterations in community structure.

Bacteria

There is evidence that the subsurface oil carbon incorporated into planktonic food webs following the DHOS came through bacterial pathways (Graham et al., 2010). Experiments and field studies suggest the impact of spilled crude oil increases both bacterial diversity and abundance (Fefilova, 2011). The application of chemical dispersants in the DHOS presumably accelerated the microbial consumption of oil components, and eventual integration into higher trophic levels (Graham et al., 2010). Using a mesocosm to study oil and oil dispersant impacts on planktonic communities, Jung et al. (2012) found bacterial abundances rapidly increased for two days following exposure to oil and dispersant. In these two days, phytoplankton and zooplankton community abundances decreased, but heterotrophic nano-flagellate abundance increased rapidly, indicating microbial loop activity. The authors also found mesocosms treated with crude oil only (i.e., no dispersant) to be less adversely affected (Jung et al., 2012). In another mesocosm study, Ortmann et al. (2012) found crude oil (taken from the DHOS) increased ciliate biomass, providing a viable pathway to transfer carbon to higher trophic levels. On the other hand, chemical dispersant resulted in increased heterotrophic prokaryote biomass at the expense of ciliates, and the authors hypothesized this may reduce grazing and subsequent transfer of carbon up the food web (Ortmann et al., 2012).

Pre Deepwater Horizon Oil Spill. Prior to the DHOS, the literature contained a fairly limited account for microbial processes following oil spills. A series of recently published articles reported the emergence of specialist marine bacteria following oil spill events. These species are adapted to hydrocarbon degradation, and include *Alcanivorax* spp., *Cyclocalisticus* spp., *Oleiphilus* spp., and *Oleispira* spp., while *Cyclocalsticus* spp. has the added ability to degrade PAHs (Head et al., 2006; Seo et al., 2009; Yakimov et al., 2007). The addition of oil has been shown to induce rapid growth of these bacteria in both laboratory and field settings. Immediately following the Agip Abruzzo oil spill

(Leghorn, Italy, 1991), Danovaro et al. (1996) observed an acute decrease in benthic bacterial abundance, followed by an oil-stimulated surge in biomass. In another study, *Alcanivorax* spp. 16S ribosomal RNA (rRNA)-gene sequences were undetectable in control experiments (Head et al., 2006). Within two weeks of oil treatment, however, they constituted more than 30% of the sequences (Head et al., 2006). The extent to which these specialist bacteria (which are usually at low or undetectable concentrations prior to an oil spill) bloom depends on: latitude, temperature, salinity, redox potential, nutrients, and other physical–chemical factors (Leahy and Colwell, 1990; Yakimov et al., 2007).

Post Deepwater Horizon Oil Spill. The bacterial response following the DHOS was very well studied. Rivers et al. (2013) sequenced marine bacterioplankton following the DHOS, and found the microbial community in plumes to be less taxonomically and functionally diverse than unexposed communities. This was attributed primarily to decreased species evenness resulting from *Gammaprotoebacteria* blooms. *Gammaprotoebacteria* are hydrocarbon degrading bacteria, and accounted for the majority of the bacterial response following the DHOS, boosting bacterial cell counts by two orders of magnitude (Rivers et al., 2013). Throughout the post-DHOS spill conditions, the natural microbial community (i.e., non-*Gammaprotoebacteria*) persisted at pre-spill levels, potentially providing a re-establishment pathway (Rivers et al., 2013).

The DHOS bacterial community was initially dominated by members of *Oceanospirillales*, *Colwellia*, and *Cycloclasticus*—none of which were dominant in surface oil slick samples (Redmond and Valentine, 2012). Gutierrez et al. (2013b) identified 3 classes of bacteria following the DHOS: aliphatic degrading bacteria (*Alcanivorax* and *Marinobacter*), PAH degrading bacteria (*Alteromonas*, *Cycloclasticus*, and *Colwellia*), and hydrocarbon degrading bacteria found in the surface slick and plume waters (*Alcanivorax*, *Alteromonas*, *Cycloclasticus*, *Halomonas*, *Marinobacter*, and *Pseudoalteromonas*). Gene sequencing of the dominant plume bacterial species revealed genes for motility, chemotaxis, and aliphatic hydrocarbon degradation were significantly enriched and expressed (Mason et al., 2012). These genes may have enabled cells to actively aggregate and increase in numbers in the plume (Mason et al., 2012). Further, in studying the role of bacterial exopolysaccharides (EPS), Gutierrez et al. (2013a) observed species producing EPS (e.g., *Halomonas*) to exhibit amphiphilic properties i.e. both hydrophilic and lipophilic properties, allowing macromolecules to interface with hydrophobic substrates (e.g., hydrocarbons). *Halomonas* increased solubilization of aromatic hydrocarbons, enhancing their biodegradation, and is likely to have contributed to the ultimate removal of oil from spill sites (Gutierrez et al., 2013a).

Benthic community

Benthic fauna also show a varied response to oil contamination. Echinoderms, and crustaceans are highly susceptible to contaminant exposure, while polychaetes, oligochaetes, and nematodes tend to be less sensitive (Danovaro et al., 1995; Gesteira and Dauvin, 2005; Peterson et al., 1996). Gesteira and Dauvin (2005) outlined a four phase response of shallow subtidal benthic communities following an oil spill event: (i) rapid mortality of sensitive species such as amphipods, (ii) low number of species and abundance (i.e., empty niches), (iii) increasing abundance of opportunistic species, and (iv) rapid decline in opportunistic species and a concomitant re-colonization of sensitive species (Fig. 2 depicts benthic density response following an oil spill in the Aegean Sea). The rise of hydrocarbon-degrading microorganisms tends to closely follow hydrocarbon release in the water column, and precedes the growth of opportunistic species (Gesteira and Dauvin, 2005). The proliferation of opportunists such as polychaetes, oligochaetes and nematodes, is resultant of their grazing on hydrocarbon-degrading micro-organisms (Gesteira and Dauvin, 2005).
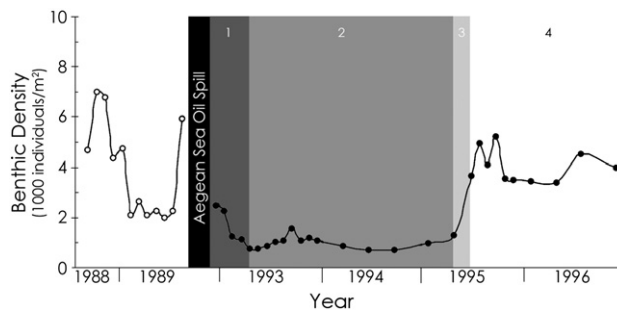
Fig. 2. Changes in benthic organism density following the *Aegean Sea* oil spill from June 1988 to August 1989 and from December 1992 to November 1996 (figure modified from Gesteira and Dauvin, 2005). The benthic response illustrates four distinct phases: 1) A sharp decrease in density (December 1992–May 1993). 2) Persistently low density (Spring 1993–Spring 1995). 3) Increased density attributed to either opportunists, or recolonizing crustaceans (Spring 1995–Summer 1995). 4) Persistently high density, with restored seasonal patterns (Summer 1995–end of survey in 1996).

The literature shows varying temporal responses and recovery times in benthic food webs following oil spills. Ho et al. (1999) observed peak toxicity (measured as mortality in excess of 70%) in the amphipod *Ampelisca abdita* 13 days following the North Cape oil spill. By day 270, toxicity had fallen to near background levels, closely following changes in sediment PAH concentrations (see Fig. 3 in Ho et al., 1999). Strong benthic response was also observed following the World Prodigy oil spill (Rhode Island, USA, 1989). Within two weeks of the spill, *Ampelisca verrilli* abundance decreased by 86% (Widbom and Oviatt, 1994); benthic recovery was not studied. Armstrong et al. (1995) studied several species of crustaceans, molluscs, and finfish at varying depths following the EVOS. They observed that PAHs of petrogenic origin were elevated in oiled bays after the spill, but declined to near background values by 1991. Feder and Blanchard (1998) also observed
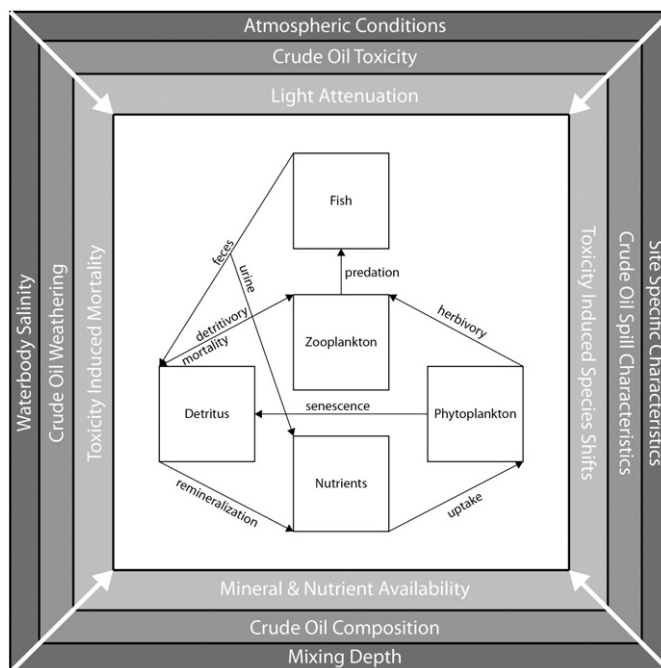
Fig. 3. Proposed paradigm shift towards food-web centric modelling strategies. The food-web (e.g., a nutrient–phytoplankton–zooplankton–detritus–fish structure) is forced externally by abiotic conditions, propagating inward. The outermost layer is representative of hydrodynamic and atmospheric conditions, such as water salinity, temperature, mixing depth, and bathymetric characteristics. The intermediate layer accounts for toxic forcing from crude oil, driven by composition, toxicity, and weathering properties. The innermost layer is indicative of the forcing directly imparted on the food-web, such as shifts in species composition, increased mortality, changes in nutrient regimes, and light attenuation.

near background level PAHs 16 months after the EVOS. More recently, significant reductions of Norway lobster (*Naphrops norvegicus*), Pandalid shrimp (*Plesionika laterocarpus*), and four-spot megrim (*Lepidorhombus boscii*) were observed in the most heavily affected region of the Prestige oil spill (Spain, 2002) (Sánchez et al., 2006). In the same region, however, no reduction was observed in hake (*Merluccius merluccius*), suggesting species-specific responses (Sánchez et al., 2006). Recoveries of *P. laterocarpus* and *L. boscii* were observed two years after the spill.

Oil contamination can also alter benthic community structure. In an experiment designed to test the contrasting roles of oil as both a potential source of organic carbon and a toxicant, Steichen et al. (1996) found that nematode abundance positively covaried with the amount of oil present, while polychaete, oligochaete, bivalve, and ostracod abundances covaried negatively. Similar results were reproduced in field trials, where nematode density was three times higher in enriched sediments compared to clean sediments (Steichen et al., 1996). Following the Agip Abruzzo oil spill, non-selective deposit feeder abundance declined, and meiofaunal structure was immediately affected (Danovaro et al., 1995). Recovery to pre-spill community structure and abundances occurred after two weeks. Immediately following the Amoco Cadiz oil spill, sediment contaminant concentrations of $50 \,\mu g \, g^{-1}$ yielded no change in the subtidal sediment community structure (Dauvin, 1998). But as concentrations increased to $1000 \,\mu g \, g^{-1}$, polychaetes emerged as the dominant class. As sediment concentrations exceeded $10,000 \,\mu g \, g^{-1}$, very low species diversity was observed, with the exception of opportunistic polycheates (Dauvin, 1998). It took in excess of ten years for the originally displaced amphipod *Ampelisca* to regain its dominance in the community, because of its low dispersal, low fecundity, and lack of a nearby unpolluted population from which emigration could occur. Based on these observations, Dauvin (1998) suggests the ecological impacts of oil spills need to be tracked for multiple years.

Summary of other biotic impacts

Bacteria have arguably the strongest response to crude oil spills in aquatic environments. Following the DHOS, bacteria community abundance increased, while both phytoplankton and zooplankton abundances decreased. Crude oil exposure in communities affected by the DHOS yielded increased ciliate biomass, providing a vector for crude oil carbon to be transferred up the food web. Conversely, exposure to both crude oil and chemical dispersant increased heterotrophic prokaryote biomass, limiting carbon transferral. The bacterial response following the DHOS was very well studied. Gene sequencing showed the opportunistic species that thrived in post-spill conditions had similar traits, allowing them to aggregate and increase in numbers. The benthic community's response to crude oil exposure can be broken down into four distinct phases: period of rapid mortality of sensitive species, followed by a period with low species variety and abundance (i.e., empty niches), leading to an increasing abundance of opportunistic species, and finally a rapid decline of opportunists as sensitive species recolonize. It can take decades for this food web normalization to occur, and researchers have suggested the ecological impacts of oil spills need to be tracked for multiple years before we can elicit robust paradigms of benthic community response.

Modelling

Modelling oil spills can be useful in aiding spill response, contingency planning, and evaluation of slick behavior and mass balance. The literature shows a large variety of models, ranging from simple two-dimensional trajectory/particle tracking, to more complex three dimensional fate models (ASCE, 1996). Reed et al. (1999) provide a rigorous review of the classical physical and hydrodynamic formulations (Fay, 1969, 1971; Hoult, 1972; Mackay et al., 1980a) underlying many contemporary models. The majority of contemporary models aim to predict/hindcast spilled oil trajectory, weathering, and fate at or near

the surface (e.g., Abascal et al., 2010; Berry et al., 2012; Marta-Almeida et al., 2013), but very few are designed to address the impacts on organisms and habitats (French-McCay, 2003). In the following sections, we review hydrocarbon-spill modelling practices, contaminant modelling in food webs, and discuss ways to improve the ecological depiction of oil spills in models.

Physical aspects

The advective properties of an oil slick on the water surface are primarily horizontal movement with wind, waves and currents, and oil droplets sinking through the water column (ASCE, 1996). Several studies have illustrated the importance of oil droplet formation and subsequent vertical movements, relating high winds and breaking waves to increased oil dispersion in the water column (Delvigne and Sweeney, 1988; Elliot, 1986; Johansen, 1984; Reed et al., 1994; Singsaas and Daling, 1992). Using the Braer oil spill (Scotland, 1993), Reed et al. (1993) challenged the wind-driven nature of classical two-dimensional surface advection models. Investigation of this spill underscored the importance of entrainment in both mass balance and transport of spilled oil (Ritchie and O'Sullivan, 1994). Coupling oil spill models with hydrodynamic models is becoming a common approach to tracking spills (Martinsen et al., 1994), but the acquisition of real time data remains problematic. Methods such as surface radar require lengthy setup times, but Howlett et al. (1993) recommend surface buoys as an alternate acquisition method.

Changes in oil properties depend on evaporative losses and surface slick thickness, both of which are driven by oil spreading rate (Reed et al., 1999). Many classical models place emphasis on spreading rate as a precursor to natural dispersion and slick persistence. The classical equations (Fay, 1969, 1971; Hoult, 1972) forming the foundation for many contemporary spreading algorithms cannot address elongated slicks, reduced spreading of viscous oils, slick patchiness, and the dependence of spread rate on initial discharge conditions (ASCE, 1996). Subsequent models have been proposed to address these issues (Lehr et al., 1984; Mackay et al., 1980a,b), but these revisions violate other key modeling aspects (e.g., lack of dynamics between thin and thick slick regions, slick thickness variability). It is generally accepted that once gravity spreading has ceased, shear spreading is caused by natural dispersion and the subsequent resurfacing of oil droplets (Reed et al., 1999). Lehr (1996) points out that classical spreading equations are most applicable at the spill epicenter (i.e., the thickest region of the slick) at very early spill stages. As such, these equations may not be suitable for long term approximation of oil spills, as they assume instantaneous release and do not account for subsurface blowouts.

The most widely used hydrocarbon evaporation models utilize simple equations based on distillation data (Fingas, 1998; Fingas et al., 1997). Other commonly used analytical methods are based on questionable assumptions, such as a linear relationship between the liquid phase boiling point temperature and the fraction lost by evaporation as seen in Striver and Mackay (1984). This assumption has been challenged by Reed et al. (1999) as an overestimation of evaporative losses. More computationally intense algorithms, such as the pseudo-component method (Daling et al., 1997), in which the slick is divided into patches distinguished by their boiling point temperatures, are generally accepted as the most robust (Reed et al., 1999).

Natural dispersion of spilled oil is dependent on multiple parameters, including sea-state, slick thickness, density, viscosity, and surface tension (ASCE, 1996). Contemporary models quantify dispersal losses using a variant of the classical equations of Mackay et al. (1980a), whereby a fraction of entrained oil is subject to natural dispersion over time (ASCE, 1996). There has, however, been some debate over the metric used in determining permanent dispersion limit (i.e., droplet size vs. droplet rise time). Permanent dispersion occurs with turbulent motion, as oil droplets are mixed deeper down the water column and rise time increases (Delvigne and Sweeney, 1988). Particle based oil

drift models are forced to address lag time between droplet mixing and subsequent rising, which may lead to an elongated slick, as the surface oil may have sheared away (Elliot, 1991; Johansen, 1987; Reed and Aamo, 1994). Reed et al. (1999) point out that resurfacing oil droplets represent only a small fraction of the slick, but may be significant when integrated over time. Emulsified oil is modelled using the implicit formulas of Mackay et al. (1980b), but the author advocates the use of a simpler form. The equations have been adapted by NOAA in the ADIOS (Automated Data Inquiry for Oil Spills) and SINTEF's OWM (Oil Weathering Model) models, tracking water uptake (emulsification) and maximum water content (inversely proportional to viscosity) parameters, but these parameters vary greatly among oils, and with weathering (Daling and Brandvik, 1988). Daling et al. (1990) highlight a key weakness of these parameters: their dependence on empirical observations under strictly controlled conditions may make prediction based models unreliable.

Ecological aspects

Very few oil spill models address the impacts on organisms and habitats (French-McCay, 2003). The SIMAP (Spill Impact Model Application Package) model includes biological submodels from the Natural Resource Damage Assessment Model for Coastal and Marine Environments, developed for the U.S. Department of the Interior in 1980. The model considers oil spill effects on aquatic organisms, including fish, invertebrates, aquatic plants, plankton, birds, mammals and reptiles (French et al., 1996). Previous efforts geared towards oil spill impacts on wildlife were threshold based, using oil thickness or oil mass as a metric to determine lethality (see French-McCay, 2003 and references therein). There are, however, studies challenging the use of oil mass and slick thickness as the ideal metrics for impact assessment (Mackay, 1980; Mackay and Leinonen, 1977). The use of oil dispersants cause large scale changes, and dissolved oil is more toxic to wildlife than surface slicks (Chapman et al., 2007). Thus, the hydrodynamic properties of the oil slick, and the ecological impact cannot be modelled as mutually exclusive. Further difficulties arise when considering multiple interaction pathways (French et al., 1996; Payne et al., 1987). Biota can, for example, interact with subsurface oil droplets, dissolved hydrocarbons, and floating oil, each of which need to be modelled explicitly.

Biotic modelling is more prevalent in contaminant fate models than ecotoxicology models, but still limited. Most contemporary oil spill models focus exclusively on abiotic factors, but Koelmans et al. (2001) highlight examples of biological integration (e.g., contaminant sorption to algae, contaminant sorption to aquatic plants, integrating of eutrophication modelling into a contaminant fate model). Typical processes considered in food chain bioaccumulation models describe contaminant uptake, depuration, transformation, and trophic transfer (Koelmans et al., 2001). The majority of food chain bioaccumulation models, however, do not consider nutrient or carbon cycling, resulting in limited ecological response as feedback pathways related to fluxes of contaminants and nutrients via organism mortality cannot be captured.

One example of an oil spill–food chain interaction model is that of Gin et al. (2001), who focused on integrating physico-chemical processes with biological uptake mechanisms. Specifically, they combined the Multiphase Oil Spill Model (MOSM) (Huda et al., 1999) and the pelagic food chain model presented in Chapra (1997). The food chain model used consisted of phytoplankton, zooplankton, small fish, large fish, and benthos, and was set such that large fish preyed upon the small fish, the small fish on zooplankton and benthos, and both zooplankton and benthos on phytoplankton. Based on the calculated dissolved and particulate hydrocarbon concentrations in the water column and sediments with MOSM, Gin et al. (2001) used the food chain model to estimate hydrocarbon concentrations at each trophic level. The food chain model assumed a static structure (i.e., fixed trophic biomasses), and was concerned only with trophic hydrocarbon:lipid ratios. Trophic hydrocarbon bioconcentrations were calculated using a *who eats whom* approach, considering the following processes at each

trophic level: oil uptake rate ($m^3 kg^{-1} s^{-1}$), oil loss (s^{-1}), BCF (oil uptake rate/oil loss), oil assimilation efficiency (g oil absorbed per g oil ingested) for each predator feeding on prey, lipid specific consumption rate (kg lipid in prey per kg lipid predator per second), organic carbon ingestion rate (kg organic carbon per kg lipid per second), and prey preference among predator species. While this work improved upon older fisheries-focused biophysical oil-spill models (e.g., Reed, 1980; Reed and Spaulding, 1978; Reed et al., 1984) by reporting hydrocarbon bioconcentrations at multiple trophic levels, the authors noted that it was a *work in progress*, as the lack of field data impeded model verification. In addition, the simplified structure of the model framework limited predictive capability and application. For example, the food chain model only considered dissolved hydrocarbon concentrations in the biotic compartments, with no regard for compartment dynamics. That is, while the hydrocarbon concentrations in zooplankton, for example, change with prevailing conditions, changes in overall zooplankton biomass are not modelled. Another limitation was the unidirectional mass flow inherent in food chain models, as opposed to food web models with explicit recycling and feedback mechanisms, which may modulate hydrocarbon concentrations in the water column. Combined, these limitations yielded a very static biotic framework driven entirely by the abiotic framework (i.e., MOSM). Granted that the goal of Gin et al. (2001) was to create an interactive model combining oil spill dynamics and food chain response in order to predict lethality in aquatic organisms, the lack of biotic–abiotic interaction and fixed food chain structure do not permit model scaling to address ecosystem issues beyond bioconcentrations of hydrocarbons immediately following spill events.

Future directions

French-McCay (2003) suggests ecotoxicological models should include: exposure (severity dependent on oil and biota properties), direct impacts in short term (lethal vs. non-lethal), chronic contamination, indirect effects of reduced food supply/habitat, reduced growth/survival/reproduction success, response time, and population level effects caused by increased mortality. Direct marine organism kills are attributed to oil contact or coating, possibly resulting in asphyxiation, and juvenile life forms are especially sensitive (Chapra, 1997). Sub lethal exposure to organisms can leave them with weakened immune responses and weaker survival, potentially restructuring the food chain (Gin et al., 2001). These acute toxic effects are caused by dissolved, rather than adsorbed or emulsified oil (Landrum et al., 1985; McCarthy et al., 1985; Yapa and Shen, 1994). The impact of any spilled oil on the biota will be a function of the concentration, type, contact duration, geographic location and organism sensitivity (Gin et al., 2001).

Another requirement in modelling biotic response to oil spills is species/trophic specific response. In any spill, plankton are especially at risk, as they reside in their highest concentration near the surface. Direct impacts on zooplankton may be less severe, as Nuzzi (1973) observed weathered oil, void of volatile and water soluble molecules to pass through copepods with no harm, but toxicity through bioaccumulation may still be of concern. In addition, while adult fish can swim and avoid oil, plankton, fish eggs and larvae cannot, increasing their sensitivity (Reed and Spaulding, 1978). Contemporary biophysical models were developed to predict impacts of oil spills and focus on fisheries impacts (Reed, 1980; Reed and Spaulding, 1978; Reed et al., 1984) but do not address impacts of specific oil compounds on key aquatic organisms. This lack of bottom up accounting makes it increasingly difficult to predict disturbances at higher trophic levels triggered by oil-mediated shifts (in species biomass and composition) in the lower food web.

The need for future ecotoxicological models to incorporate biological components is vital, but determining the best method in which to do so remains challenging. Modelling the impacts of a crude oil spill on aquatic food webs in a pelagic environment, for example, requires aspects of fluid dynamics, chemical weathering, nutrient cycling, and a

multi-dimensional food web. While simple models serve as valuable tools for theory building (Perhar et al., 2013), their simple mechanistic frameworks may prohibit them from realistically simulating field data. Likewise, large scale models with complex inter-compartmental dynamics have their own drawbacks (e.g., over-parameterization, convoluted outputs). Below, we outline steps that future studies may wish to take when integrating aspects of food web ecology into oil spill modelling.

Given the entanglement of processes taking place during an oil spill, we suggest starting with a simple approach, and gradually building complexity. As highlighted above, existing contaminant models are driven from an abiotic point of view, and then applied to the biological layer. We suggest the opposite, such that the abiotic factors are treated as boundary conditions, while the food web is the central focus (see Fig. 3). There is ample data available in the literature for parameterization of a relatively simple food web structure. Starting with frameworks that consider the different trophic levels as aggregated entities, such as limiting nutrient–phytoplankton–zooplankton (NPZ), nutrient–phytoplankton–zooplankton–detritus (NPZD), or nutrient–phytoplankton–zooplankton–

fish (NPZF), the basic foundation for studying food web dynamics exposed to petroleum hydrocarbons is set. Importantly, viewing the release of petroleum hydrocarbons as perturbations that can potentially trigger the emergence of alternative attractors (Fig. 4), it is simply unrealistic to expect that the same mathematical formulations and/or parameterization will be adequate to reproduce both pre- and post-oil spill conditions. In this regard, one promising strategy may be the adoption of hierarchical frameworks that accommodate the idea of the existence of three distinct states of the same ecosystem, which share partial (and not complete) commonality in behavior (Fig. 5 top panel). From a mathematical standpoint, the proposed state-specific model parameterization explicitly considers the substantial structural and functional alterations induced by an oil spill, but the hierarchical configuration allows transferring information between the three states and thus avoiding problems of overfitting (Cheng et al., 2010). In doing so, we believe that the hierarchical strategy proposed offers a means to reproduce ecosystem response with a pragmatic (albeit coarser) biotic resolution/complexity relative to ambitious modelling constructs that target simulations of multiple functional groups (genera or species) and a wide range of trophic relationships. This is in stark contrast to tracking sub-trophic level dynamics, such as species successional patterns (see Fig. 5 bottom panel). Such an objective renders a more complicated modelling exercise, and is susceptible to the aforementioned issue of overfitting.

While we push for drastic simplifications in our first-approximation of an oil spill–food web hybrid model, we appreciate the complex nature of the processes governing abiotic conditions. Dispersal dynamics may be the most important to track, as they are driven by multiple processes varying with time, and result in a spatially heterogeneous
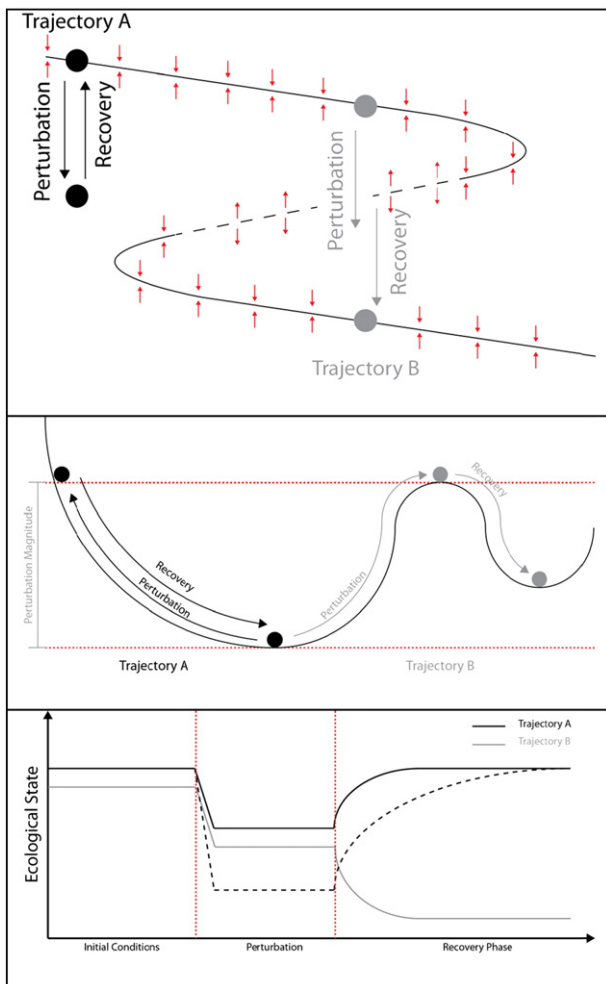

Fig. 4. Ecological attractor diagrams, illustrating hypothetical system shifts triggered by hydrocarbon spills. The expected recovery of a system depends on topological features, initial conditions, and perturbation magnitudes. If there are no nearby attractors (Trajectory A in each of the three panels), it is possible to experience complete system recovery, no matter how strong the magnitude of the oil spill perturbation is. Under a different set of initial conditions, representing the impact of other exogenous stressors (e.g., eutrophication, contamination, climate change), there may be a neighboring attractor, and thus the oil spill could potential push the system into an alternate steady state (Trajectory B in the three panels). In this scenario, the likelihood of full recovery is minimal. Systems with identical initial conditions but different magnitudes of perturbation may recover at different paces as illustrated in the lower most panel.
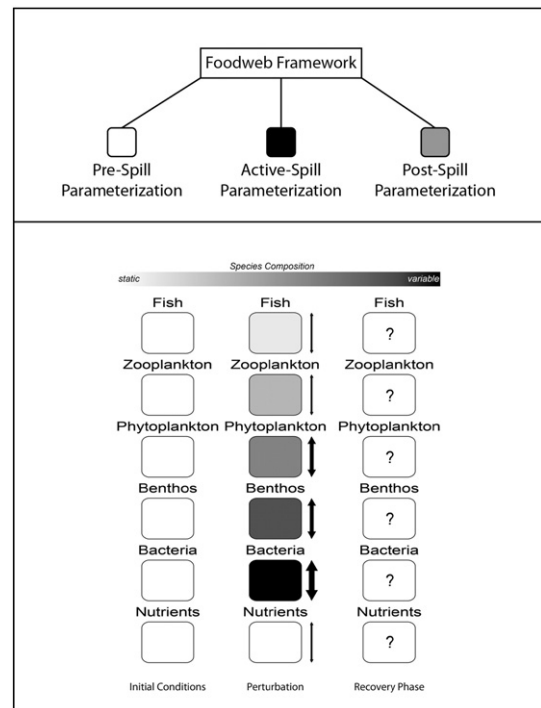

Fig. 5. (top panel) Hierarchical framework to reproduce food-webs experiencing crude oil spills. Three distinct phases (pre-, during- and post-spill) with aggregated trophic levels are parameterized, while the hierarchical structure allows for the transfer of information among the states. (bottom panel) Reductionistic modelling strategy to track trophodynamics in a food-web experiencing crude oil spills. Box shading is indicative of the likelihood of change in species composition (darker boxes are more likely to experience changes in species composition than lighter ones), and arrow thickness indicates the likelihood of biomass fluctuations. Recovery phase dynamics are a function of the initial system conditions and the perturbation magnitude (e.g., hydrocarbon characteristics and volume). The former approach is proposed as a pragmatic modelling tool to oil-spill management, despite its coarser biotic resolution.

environment. One approach towards the development of an *Oil spill–food web* construct may be the implementation of a box strategy, whereby multiple neighboring food web models are concurrently running, in an effort to capture coarse-grained spatial heterogeneity. For example, a box at the epicentre of the spill will be subject to higher hydrocarbon contaminant concentrations, higher light attenuation, and presumably stronger food web shifts than a box closer to the slick periphery. Through cross-boundary exchanges, however, contaminants bioaccumulating in the epicentre may be found in other boxes. Further complications are expected when temporal variability is considered explicitly in the food web, as contaminant uptake, depuration, and mortality rates vary with organism age. In any event, reversing the contemporary approach of using a computationally complex abiotic and relatively simplistic biotic compartment will not only provide a solid foundation for modelling the ecological effects of hydrocarbon spills, but widen the exploration domain, yielding a more sound management tool that can be used to draw more insightful conclusions.

Conclusions

A great deal of the world's freshwater is found in Canada in the Laurentian Great Lakes system. The 3700 km GL–SLS is a vital commercial transportation route linking the Great Lakes economic hub to the rest of the world. With the large number of transit ships using this route annually, the threat of an oil spill and the associated risk to aquatic organisms is real. In 1976, for example, an estimated 300,000 gal of crude oil were spilled in the St. Lawrence River following the grounding of the NEPCO 140 barge (Yapa et al., 1992). Another sizeable portion of Canada's freshwater is locked in Arctic glaciers. In recent years, however, the face of the Arctic has been changing at a higher rate than previously observed. These changes include: warming permafrost, reduction of snow cover extent and duration, reduction in summer sea ice extent, increased mass loss from glaciers, thinning and breakup of remaining Canadian ice shelves (Derksen et al., 2012). The Canadian Arctic Archipelago is exhibiting statistically significant decreases in average total sea ice area (-8.7% per decade) (Howell et al., 2009). As the ice cover recedes in Canada's north, the Arctic Ocean becomes another viable shipping route, exposing the relatively pristine marine Arctic food web to a potential oil spill. Marine spills may be more difficult to contain than freshwater spills due to oceanic currents, swells, and large fetches. Freshwater spills, however, may be more detrimental as PAH solubility is inversely related to salinity (Whitehouse, 1984). In addition, freshwater food webs are generally smaller than their marine counterparts, and the ecological damage may be more extensive due to lack of functional redundancy (Hjorth et al., 2007; Walker, 1995). The DHOS was the most highly organized, and largest scale oil spill response in history. The lessons learned emphasized the need for baseline data. Thus, it may be a good idea to start oil spill experiments in the Great Lakes using *Before-After-Control-Impact* (BACI) sampling designs, in conjunction with developing a holistic oil spill model. Establishing baseline characteristics for potential spill sites is extremely important, and a major hindsight regret in the Gulf coast.

It is only a matter of time until the next petroleum hydrocarbon spill occurs. While this may seem like an overly pessimistic viewpoint, it is necessary, considering the global economy operates on acceptable risk-levels (not ideal risk-levels). Understanding both baseline conditions, and post-spill dynamics is vital to selecting an appropriate clean-up response. The current method of tracking and hind-casting oil spills, however, minimizes the role of the biotic compartment, and focuses instead on the abiotic fates of petroleum hydrocarbons and other toxic compounds. We stress that this method does not capture the whole story. As reviewed earlier, spilled hydrocarbons may initiate structural shifts in food web communities, promoting species that can readily metabolize hydrocarbons. Conversely, hydrocarbons may retard growth, increase mortality, and propagate the toxicity effects up the

food chain. Complicating matters further, are the well documented detrimental effects of commonly used chemical dispersants on both flora and fauna. Each one of these potential outcomes exposes the environment to differential stresses, which are compounded by the abiotic properties of petroleum hydrocarbons. Thus, if the goal of a model is to simply track the abiotic fate of spilled hydrocarbons, the contemporary modelling literature is well stocked. If, on the other hand, the aim is to gauge ecosystem response to an oil spill, and propose holistic remediation strategies focused on minimizing food web disturbances, not only do both abiotic and biotic compartments need to be considered, but their interactive effects must be explicitly formulated. In this study, we have reviewed the literature, and provided a strategy to begin building such a tool.

Acknowledgments

Appendix A

Abbreviations used in alphabetic order:

ADIOS	Automated Data Inquiry for Oil Spills
BaP	benzo(a)pyrene
BCF	bioconcentration factors
CYP1A	Cytochrome P4501A
EC50	effective concentration for 50% growth reduction
EPS	exopolysaccharide
EVOS	Exxon Valdez oil spill
GL–SLS	Great Lakes–St. Lawrence Seaway
HOC	hydrophobic organic compound
MFO	mixed function oxidase
NPZ	Nutrient–Phytoplankton–Zooplankton model
NPZD	Nutrient–Phytoplankton–Zooplankton–Detritus model
NPZF	Nutrient–Phytoplankton–Zooplankton–Fish model
OWM	Oil Weathering Model
PAH	polycyclic/polynuclear aromatic hydrocarbons
PCB	polychlorinated biphenyl
POM	particulate organic matter
POP	persistent organic pollutant
ppb	parts per billion
ppm	parts per million
PSI	photosystem 1
PSII	photosystem 2
PWS	Prince William Sound
SIMAP	Spill Impact Model Application Package
WSF	water soluble fraction

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