

State-of-the-Art and Research Needs for Oil Spill Impact Assessment Modeling

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Abstract

Modeling can be a powerful tool for oil spill impact quantification as part of environmental risk assessments, contingency planning, and natural resource damage assessments. Historically, oil spill models have focused on trajectory and fate in aquatic environments, with impacts being assessed in most cases by evaluating the presence of biota in the area exposed to floating or shoreline oil. A few models have addressed subsurface oil concentrations to which water column biota are exposed, but most of these simply overlay fates model concentration results on maps or grids of biological distributions to assess “impact”. This paper describes the state-of-the-art of biological effects modeling for the most comprehensive approach to date, a model that evaluates dose and resulting impact of oil hydrocarbons on aquatic biota including birds, mammals, reptiles, fish, invertebrates, and plants. The biological effects model is coupled to an oil trajectory and fates model that supplies required spatial and temporal quantification of oil distributions and hydrocarbon component concentrations. Model development and validation studies are reviewed, strategies for applying the model for hindcasts and risk assessments are discussed, and research and data needs are identified.

1 Introduction

There is a growing demand by both government and industry for oil spill fates and biological effects modeling to address the potential environment impacts of spills and oil-related activities. This demand is driven by government regulations, the limits of data collections from field and laboratory work, and the growing power of computers for performing data analyses and modeling calculations. Models use knowledge of physical, chemical, and biological relationships along with environmental data to simulate pollutant transport, fate and biological effects. This analysis is useful for risk assessment, contingency planning, cost-benefit analysis and natural resource damage assessment (NRDA). Modeling may be applied to investigate a single spill event, to evaluate the probable consequences of a hypothetical spill, or to determine impacts of a worst-case spill scenario. Examples of these applications include risk assessment in support of permit application, comparison of response strategies for contingency planning, and analysis of maximum liabilities for accidental spills.

A number of oil trajectory and fates models are available around the world. Several reviews of the state-of-the-art in oil spill trajectory and fate modeling have been performed over the past two decades (Huang, 1983; Spaulding, 1988; ASCE, 1996; Reed et al., 1999) to assess the state of the practice, to summarize key developments, and to project future capabilities. The reader is referred to these papers and the literature (e.g., Mackay et al., 1982; Kirstein et al., 1987; Lehr et al.,

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1995, 2000; Jones, 1997; Galt, 1998; Reed et al., 2000; French McCay, 2004; Boufadel et al., 2007) for descriptions of these and other oil fate models, as well as research needs.

In contrast, few model developers have carried out the analysis to quantitatively address biological impacts of oil spills in aquatic environments. In most cases, impacts are assessed by evaluating the presence or densities of biota in the areas exposed to floating or shoreline oil. In some cases, the oil fate model used for the assessment is three-dimensional, such that subsurface concentrations are considered. However, the assessment of impact is generally performed by establishing a threshold for concern and then mapping or quantifying the area “impacted”. The problem with this approach is that an impact is assumed if there is an intersection of some amount of oil with biological “receptors”, without consideration of dose, uptake, duration of exposure, or measurable effect.

Early modeling efforts for wildlife impact assessment relied on calculating the intersection of oil trajectories with birds or marine mammals, assuming an impact threshold (Reed et al., 1989; Jayko et al., 1990). In some cases, wildlife population and migration models were used to simulate the distribution, behavior and recovery of the affected species, in conjunction with their intersection with oil trajectories (Samuels and Lanfear, 1982; Ford, 1985, 1987, Ford et al., 1982; Brody, 1988; French and French, 1989; French et al., 1989; Seip et al., 1991). In these modeling efforts, the impact threshold for wildlife was appropriately based on a threshold oil thickness or mass for lethal or sublethal effects, although quantitative information definitively indicating what dose would be lethal was not available.

Both subsurface oil droplets and dissolved hydrocarbons must be explicitly simulated (in addition to surface floating oil and associated processes) in an oil fates model in order to be able to evaluate exposure of aquatic biota to oil hydrocarbons and biological effects. A prime case example is the *North Cape* oil spill of January 1996, which occurred during a severe winter storm where 2682 metric tons (828,000 gallons) of home heating oil (No. 2 fuel oil) spilled into the surf zone on the south coast of Rhode Island, USA. Most of the oil was entrained into the water column by heavy surf, resulting in high concentrations of dissolved components in shallow water, which took weeks to disperse, killing millions of water column and benthic organisms (French McCay, 2003). Moreover, because the many hydrocarbons in oil have varying physical-chemical properties (most significantly those related to solubility and volatility), the oil fates model must separately track chemical classes or pseudo-components of the whole oil with characteristics typical of the chemical group to simulate their separate fates (Payne et al., 1984, 1987; Kirstein et al., 1987; French et al., 1996; Jones, 1997; Reed et al., 2000; French McCay, 2004). Most oil fates models employ a Lagrangian particle approach, which enables the modeler to track physical and chemical property changes as oil weathers. This is particularly needed when oil is released over time under varying conditions. The Lagrangian methodology is also useful for biological modeling to track organisms’ movements and exposure to oil (French et al., 1996; French McCay, 2003, 2004).

Potential and documented impacts of oil in aquatic environments have been reviewed by the National Research Council (NRC, 1985, 2002) as well as others (Neff et al., 1976; Neff and Anderson, 1981; Engelhardt, 1983, 1987; Teal and Howarth, 1984; Capuzzo, 1987; Geraci and St. Aubin, 1990; Rice et al., 1996; Sloan,

1999; Kingston, 2002). A biological effects model that considers all impacts of oil should include evaluation of: exposure considering movements and amounts of both oil and biota; duration of exposure and degree of accumulation in tissues; acute effects and direct impacts (lethal and sublethal) in the short-term; sublethal effects of chronic contamination; behavioral changes resulting in reduced growth, survival or reproductive success; indirect effects via reduction in food supply, habitat, or other changes in the ecosystem; impacts of spill response activities; and population level impacts caused by mortality and sublethal effects. Supporting research and information is available to quantify some but not all of these effects, as discussed below.

Herein, the state-of-the-art of biological effects modeling for use in impact and risk analyses is described, as developed by French and French (1989), French et al. (1996), and French McCay (2002, 2003, 2004), and on-going research and case analyses for NRDA and risk assessments. A biological effects model requires physical and environmental inputs including (1) wind data as time- and (optionally) spatially-varying velocities; (2) current data as time- and spatially-varying velocities; (3) environmental conditions such as temperature and salinity; and (4) physical fates model outputs that quantify spatial distributions, physical-chemical characteristics, and concentrations over time of floating oil, entrained oil droplets, dissolved hydrocarbon components, oil in sediments, and oil on shorelines. Typically, a biological effects model is coupled to an oil fates model capable of providing the needed information, such as that described by French McCay (2004). However, in the future, models could be modularized and fates models could provide standardized outputs that could be used as input to biological effects models.

This paper provides background for current research being supported by the Coastal Response Research Center at the University of New Hampshire. Previous model development, modeling approaches, available information, potential algorithms, and data needs for developing the next-generation oil spill biological effects model are being reviewed. Processes being considered in the biological effects modeling review include (1) oil hydrocarbon exposure (to floating oil, entrained droplets, dissolved hydrocarbons, oil in sediments, and oil on shoreline) for habitats, wildlife, fish and invertebrates, including consideration of behavior (i.e., normal, avoidance and attraction); (2) pathways and rates of uptake of hydrocarbons into biota; (3) lethal and sublethal effects levels for mechanical, smothering, thermal, and/or toxicological effects of (whole) oil on wildlife and aquatic biota; (4) acute and chronic toxicity of hydrocarbons on aquatic biota, including consideration of duration of exposure and long-term effects on development, growth, reproduction, etc.; (5) phototoxicity of polynuclear aromatic hydrocarbons (PAHs) on aquatic biota; and (6) population and ecosystem level effects and recovery rates. This paper focuses on model development to date: that addressing acute toxic effects resulting from short-term exposure. Long-term effects of oil hydrocarbon exposure have not been addressed in oil spill models to date, due to the complexity, site-specificity required, and paucity of quantitative information to develop such a model.

2 Coupled Physical Fates Model

The SIMAP (Spill Impact Model Application Package) physical fates model (French McCay, 2004), to which the SIMAP biological effects model is coupled

(Figure 1), is described briefly here to illustrate the needed capabilities for input to the biological effects model calculations. The SIMAP models were derived from the physical fates and biological effects submodels in the Natural Resource Damage Assessment Model for Coastal and Marine Environments (NRDAM/CME), which were developed for the U.S. Department of the Interior (USDOI) as the basis of Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) Natural Resource Damage Assessment (NRDA) regulations for Type A assessments (French et al., 1996). The SIMAP physical fates model is described in detail in French McCay (2004).

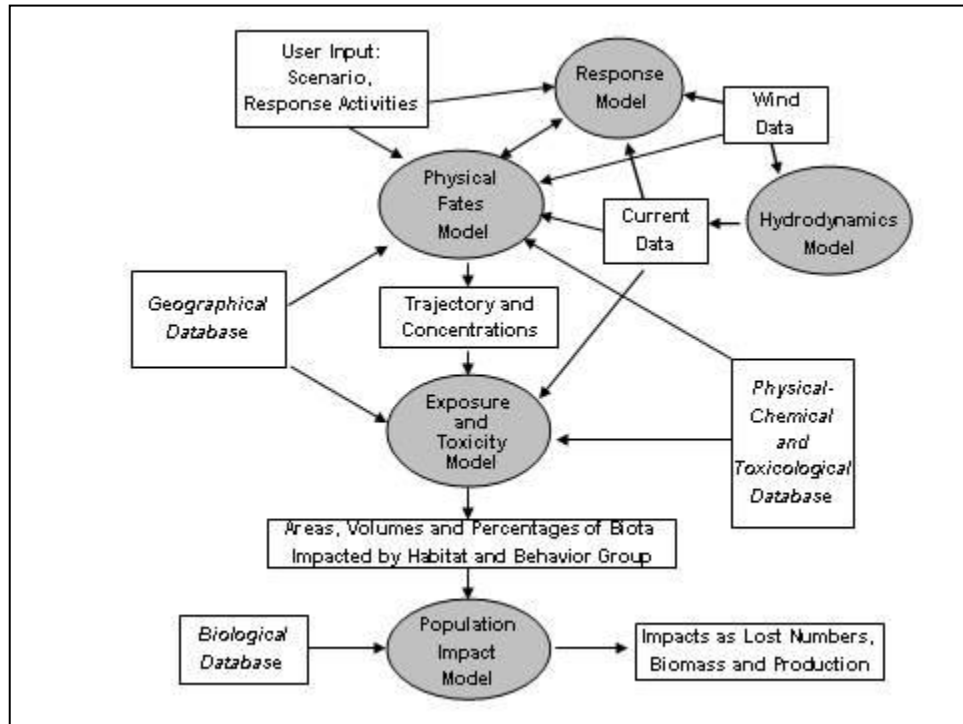


Figure 1. Diagram of the SIMAP Model System

The transport model in SIMAP (French McCay, 2003, 2004, also the earlier version in French et al., 1996) and other oil spill models (Mackay et al., 1982; Spaulding et al., 1983; Spaulding, 1988; Lehr et al., 1995, 2000; Galt, 1998; Reed et al., 1999, 2000) utilize similar algorithms for calculating advective movements and turbulent dispersion, i.e., Lagrangian elements (“LES” or “spilletts”) are used to track the oil movements and weathering. The SIMAP physical fates model (French McCay, 2004) uses wind data, current data, and transport and weathering algorithms to calculate the mass of oil components in various environmental compartments (water surface, shoreline, water column, atmosphere, sediments, etc.), oil pathway over time (trajectory), surface oil distribution, and concentrations of the oil components in water and sediments over time. The distribution of oil in space and time is expressed as mass per unit area on the water surface, mass per unit area on shorelines, concentration in the water column, and mass loading per area of the sediments.

Processes simulated in the physical fates model include slick spreading, evaporation of volatiles from surface oil, transport on the water surface and in the water column, randomized dispersion, emulsification, entrainment of oil as droplets into the water column, resurfacing of larger droplets, dissolution of soluble components (i.e., lower molecular weight aromatics), volatilization from the water column, partitioning of semi-soluble components between sediments and the dissolved form, sedimentation, stranding on shorelines, and degradation. Oil mass is tracked separately for lower-molecular-weight aromatics (1 to 3-ring aromatics), which are soluble and so cause most acute toxicity to aquatic organisms (French McCay, 2002, see below), other volatiles, and non-volatiles. The lower molecular weight aromatics dissolve both from the surface oil slick and whole oil droplets in the water column, and they are partitioned in the water column and sediments according to equilibrium partitioning theory (French et al., 1996; French McCay, 2003, 2004).

“Whole” oil (containing non-volatiles and volatile components not yet volatilized or dissolved from the oil) is simulated as floating slicks, emulsions and/or tar balls, or as dispersed oil droplets of varying diameter (some of which may resurface). Sublots of the spilled oil are represented by individual spilletts, each characterized by mass of hydrocarbon components and water content, location, thickness, diameter, density, and viscosity. Spreading (gravitational and by transport processes), emulsification, weathering (volatilization and dissolution loss), entrainment, resurfacing, and transport processes determine the thickness, dimensions, and locations of floating oil over time. The output of the fate model includes the location, dimensions, and physical-chemical characteristics over time of each spillet representing oil (French McCay, 2003, 2004).

Concentrations in the water column are calculated in SIMAP by summing mass (in the spilletts) within each grid cell of three-dimensional grid scaled each time step to just cover the dimensions of the plume. This includes all potential contamination in the water column, while maximizing the resolution of the contour map at each time step to reduce error caused by averaging mass over large cell volumes. Distribution of mass around the particle center is described as Gaussian in three dimensions, with one standard deviation equal to twice the diffusive distance ($2D_x t$ in the horizontal and $2D_z t$ in the vertical, where D_x is the horizontal and D_z is the vertical diffusion coefficient, and t is particle age). The plume grid edges are set at one standard deviation out from the outer-most particle. Concentrations of particulate (oil droplet) and dissolved aromatic concentrations are calculated in each cell and time step and saved to files for later viewing and calculations. These data are used by the biological effects model to evaluate exposure, toxicity, and effects.

In summary, the fates model quantifies, in space and over time:

- The spatial distribution of oil mass and volume on the water surface (including flooded intertidal areas and lands) over time;
- Oil mass, volume and thickness on shorelines of varying types over time;
- Subsurface (in-water) oil droplet concentrations, as total hydrocarbons, in three spatial dimensions over time;
- Dissolved aromatic concentrations in water in three spatial dimensions over time; and
- Total hydrocarbons and aromatics in the sediments over time.

The fates model output at each time step includes:

- Oil thickness (microns or g/m^2) floating on water surfaces,
- Oil loading (g/km and g/m^2) on shorelines,
- Subsurface oil droplet concentrations in water (ppb), as total hydrocarbons,
- Dissolved aromatic concentrations in water (ppb),
- Total hydrocarbon loading on sediments (g/m^2), and
- Dissolved aromatics concentrations in sediment pore water (ppb).

The SIMAP transport model has been validated with more than 20 case histories, including the *Exxon Valdez* and other large spills (French McCay, 2003, 2004; French McCay and Rowe, 2004), as well as test spills designed to verify the model's transport algorithms (French et al., 1997; French McCay et al., 2007). It has been used in many NRDA cases in the US, as well as in ecological risk assessments for potential spills world wide.

3 Biological Effects Model

The biological exposure model estimates the area or volume where organisms are adversely affected by surface oil, concentrations of oil components in the water, and/or sediment contamination. The area or volume impacted may be multiplied by organism density to calculate an impact or evaluated as a portion of a stock or population affected. The biological effects model (Figure 2) estimates losses resulting from acute exposure after a spill (i.e., losses at the time of the spill and while floating oil and acutely toxic concentrations remain in the environment) in terms of direct mortality and lost production because of direct exposure or the loss of food resources from the food web. The model first estimates percentage losses in discrete habitat areas or volumes by behavior group (e.g., aerial seabirds in areas, pelagic fish in volumes), translating these to equivalent areas and volumes of 100% loss (i.e., by summing the percent loss times area or volume affected). These equivalent areas and volumes are then multiplied by biological density data to estimate direct losses of species or species groups of fish, invertebrates (i.e., shellfish and non-fished species) and wildlife (birds, mammals, reptiles). Lost production of aquatic plants (microalgae and macrophytes) and lower trophic levels of animals are also estimated in the direct loss model calculations. Lost production of prey species are translated to losses higher in the food web using a food web model. Future losses are calculated using a population model, accounting for lost future growth as well as natural and harvest mortalities for each life stage and annual age class.

The area potentially affected by the spill is represented by a rectangular grid with each grid cell coded as to habitat type. The same habitat grid is also used by the physical fates model to define the shoreline location and type, as well as habitat and sediment type. A habitat is an area of essentially uniform physical and biological characteristics that is occupied by a group of organisms that are distributed throughout that area. A contiguous grouping of habitat grid cells with the same habitat code represents an ecosystem in the biological model. Pre-spill densities of fish, invertebrates, and wildlife (birds, mammals, reptiles, and amphibians) are assumed evenly distributed across each habitat type defined in the application of the model. Habitat types may be defined to resolve areas of differing density for each species, and the impact in each habitat type is then separately computed. While

biological distributions are known to be highly variable in time and space, data are generally not sufficient to characterize this patchiness. Oil is also patchy in distribution. The patchiness is assumed to be on the same scale so that the intersection of the oil and biota is equivalent to overlays of spatial mean distributions.

Mobile fish, invertebrates and wildlife are assumed to move at random within each ecosystem during the simulation period, a reasonable assumption for the few weeks following oil release that are modeled. Aquatic organisms are modeled using Lagrangian particles representing schools or groups of individuals. Benthic organisms remain stationary on or in the bottom. Planktonic stages, such as pelagic fish eggs and larvae are transported by the currents (input to the model).

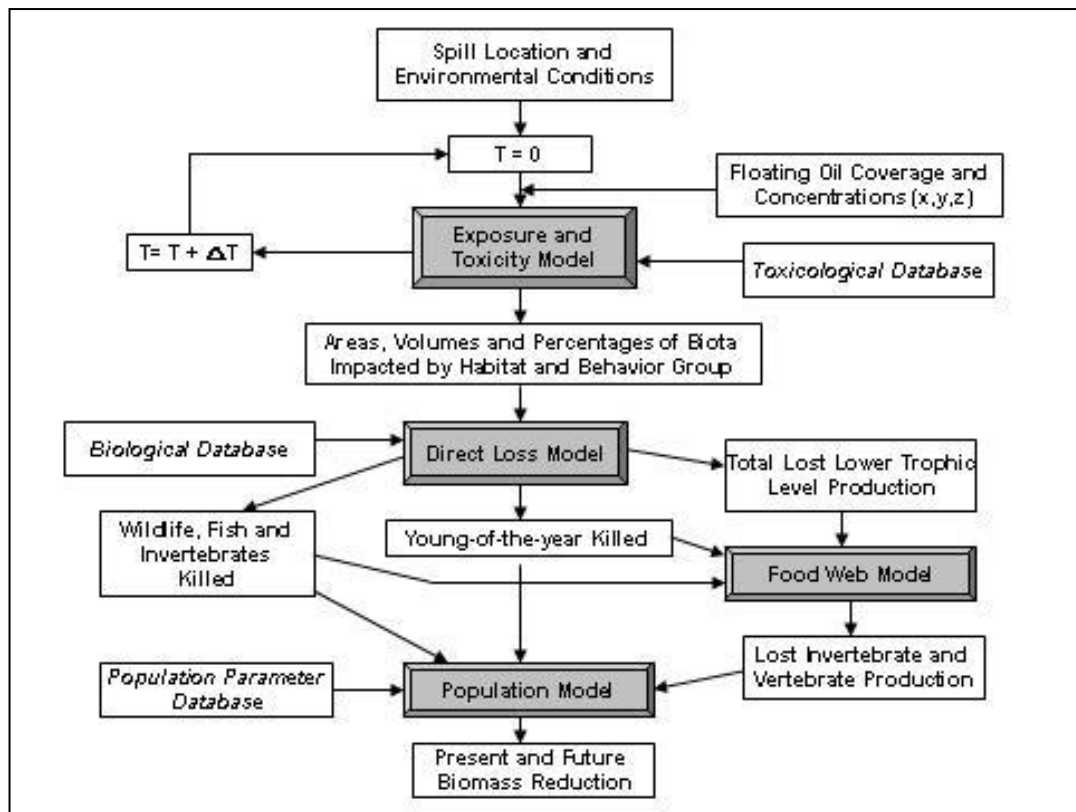


Figure 2. Diagram of the Biological Effects Model

Habitats include open water, invertebrate reef, wetland (marsh, swamp), seagrass, macroalgal bed, and shoreline environments. Habitat types are defined by depth, salinity regime, proximity to shoreline(s), bottom/shore type, dominant vegetation type, and the presence of invertebrate reefs. With respect to salinity and proximity to shoreline(s), habitats are designated as landward or seaward. This designation allows different biological abundances to be simulated in landward and seaward zones of the same habitat type (e.g., open water with sand bottom). Thus, the landward-seaward designation is operational and tailored to the needs for a particular assessment in an oil-affected area. The SIMAP model has been applied to marine, estuarine and freshwater environments, with habitats defined accordingly.

Wildlife (i.e., air-breathing vertebrates) individuals that move through the area swept by floating oil are assumed to be oiled based on probability of encounter. Those oiled above a threshold dose are then assumed to die.

Fish, shellfish, their eggs and larvae, and other plankton are affected by dissolved concentrations of hydrocarbons in the water or sediment. Mortality is calculated using laboratory acute toxicity test data (LC50, concentration lethal to 50% of test individuals) corrected for temperature and time of exposure, and assuming a log normal relationship between percent mortality and dissolved concentration. The model accounts for the uptake, accumulation and additive effects of the mixture of hydrocarbons to which the organisms are exposed. Organisms killed are integrated over space and time and by habitat type to calculate a total short term kill. This total short term kill occurs from the time of the spill until the time the contaminants are dispersed to the point where concentrations are below toxic levels.

Lower trophic level biota are generally not evaluated by species or species group, and impacts are quantified as lost production in each habitat affected. Lost primary (plant) and secondary (herbivore) production due to sublethal concentrations of dissolved hydrocarbons is estimated using the EC50, the concentration where growth rate is 50% of the clean control, correcting it for temperature and assuming a log normal relationship between percent of uninhibited growth rate and concentration. Primary and secondary production losses are integrated over space and time and by habitat to calculate the total biomass not produced during the spill. For areas affected by sublethal concentrations, the rates of production are assumed to return to normal immediately following the dispersion of the contamination to non-toxic concentrations.

For microalgae (phytoplankton and benthic microflora), concentrations which reduce production to near zero are essentially lethal. The above calculation procedure covers these losses. Reseeding of these groups into affected habitats which are no longer toxic is so rapid that recovery may be assumed immediate following dispersion of toxic concentrations. However, for macrophytes, recovery following exposure to lethal concentrations is not immediate. In habitats dominated by macrophytes (e.g., marshes, swamps, kelp beds, seagrass beds), complete mortality of plants is assumed if a lethal threshold is reached (in water concentrations or in oil coverage). Losses from the direct kill and over the period of natural recovery after the toxicity is gone are included in the tabulation for macrophytes and for animals (wildlife, fish, shellfish, eggs, larvae and benthos) dependant on the affected habitat for food or development.

Biomass which is not produced as a result of a loss of food resources is estimated using a simple food web model. The portion of the lost primary production which would have produced primary consumer (secondary producer or herbivore) biomass is estimated based on observations of ecological efficiency made on representative ecosystems. The fractions of the lost secondary production which would have been consumed by each of their predators are assumed to be proportional to the biomass of that predator relative to the sum of its competitors. The output of this part of the model is lost production of the various fish, shellfish, and wildlife species as a result of the spill. This is added to the total direct kill to yield a total loss of biomass by habitat as a short term result of the spill.

In addition to the direct kill and food web losses of eggs, larvae and juveniles, these young-of-the-year may be lost via habitat disruption. This is included in the model for macrophyte-dominated habitats affected by lethal oiling. Losses are assumed proportional to the macrophyte loss. Thus, recovery of spawning and nursery habitat in wetlands, kelp beds, and seagrass beds follows recovery of macrophyte biomass and production. Similar assumptions are made for coral reefs based on the same arguments.

Potential long term losses (losses realized after the spill has dissipated) which can result from a spill of a toxic substance include: (1) lost recruitment to a fishery of the larvae and juveniles killed at the time of the spill; (2) lost future growth of the adults killed at the time of the spill; (3) changes in food web structure and, therefore, productivity of specific trophic levels and populations; and (4) chronic effects of sublethal levels of contaminants in tissues or the environment, such as reduced growth rate or loss of reproductive potential. The first two effects are typically the most dominant following an acute event such as an oil spill. The latter two effects have not been included in any model to date, both due to the complexity of developing such models, which necessitates that they be site specific, and to the lack of quantitative information required to apply these models (e.g., see Hansen, 1984). Thus, the impacts estimated in the biological effects model are the results of acute toxicity and resulting direct effects on long term productivity and yield: lost recruitment and lost growth. As such, the model does not consider any indirect (chronic) effects or feedbacks after the spill has dissipated, such as changes in mortality due to density dependent effects, changes in food web structure and predator prey relationships, or changes in reproductive potential. The modeled spill impact extends only for the life span of the species considered, and growth and reproduction are assumed to return to normal after the effects of the spill have dissipated.

For fish and invertebrates (juveniles over the age of one year and adults of a species), it is assumed that a constant natural mortality rate (due to natural causes) applies; juveniles (over age one) and adults inhabit the same habitat at the same time; there is a constant fishing mortality rate for animals past the age of recruitment to the fishery; and that growth follows the von Bertalanffy relationship (Ricker, 1975). Using these assumptions and standard steady state fisheries models, the age structure of a population and long term losses in yield as the result of the spill may be estimated (Ricker, 1975). The direct kill, in numbers by age, is calculated from biomass killed, weight as a function of age, and natural and fishing mortality rates. For each year following the spill, the lost catch, calculated from fishing mortality, weight by age and average number alive during that year, is calculated. Thus, only those individuals which would have been caught in present and future years, and not those which would have died naturally, are included in the quantification of the future production and catch losses due to the incident.

The relatively high mortality rates of eggs and larvae are considered in the biological effects model, since a high number killed at the time of the spill would have died regardless of the spill. The young-of-the-year (eggs, larvae, and juveniles less than one year old) of each fishery species category are tracked as percents of the age one population. Young-of-the-year and older age classes are not assumed to inhabit the same environment concurrently, and their losses are calculated separately.

For waterfowl and mammals, losses to hunting are calculated assuming constant natural and hunting mortality rates over time after the spill. For all wildlife, population losses are calculated as the number not left alive in present and future years as the result of the spill, after constant natural mortality (and hunting mortality) is subtracted.

The biological effects model is designed to be generally applicable, while restricting the data and parameter requirements to information which is generally available. As part of the development of the NRDAM/CME (French et al., 1996), biological data were compiled for a series of habitat types within each of 77 regions of U.S. coastal and marine waters, as opposed to site specific information for every possible geographic location. A similar approach has been taken when applying the model to other locations outside US waters. In addition, freshwater databases have been developed for the Great lakes and other inland waters. Site- and event-specific databases have been developed for specific case investigations or risk assessments.

Required biological data for applying the model to obtain biomass and numerical losses by species are: estimates of fish and invertebrate biomass per area ($\text{kg wet weight}/\text{km}^2$), numbers of wildlife (birds, mammals, reptiles) per unit area ($\#/ \text{km}^2$), and rates of production for lower trophic levels (plants and invertebrates, $\text{g C m}^{-2} \text{ day}^{-1}$). These data may be specific for the time of a spill or, if a biological database is developed, seasonal or monthly means in each habitat type.

Parameters required for the population model include estimates of natural and fishing (hunting) mortality rates, available from the fisheries and wildlife literature; age specific growth rates, available from length or weight at age catch data; and age at recruitment and life span. The spawning areas and times for fish and invertebrate species and development information for the young of all species groups are also compiled when evaluating these losses. All of these estimates are specific to habitat within each geographic region.

3.1 Wildlife: Air-Breathing Vertebrates

3.1.1 Model Algorithm

For wildlife (air-breathing vertebrates: birds, mammals, and reptiles, and adult amphibians), the number or fraction of a population suffering oil-induced effects is proportional to the water-surface area swept by oil (slicks, emulsions, or other floating forms such as tar balls) of sufficient quantity to provide a lethal dose to an exposed animal. Wildlife populations are assumed to be in equal density across each ecosystem (each grouping of like habitat grid cells) and to remix within each ecosystem each day. For each day of the simulation, those oiled above a threshold dose are assumed to die, and the remainder may be oiled in subsequent days if oil is still present on the water surface.

Wildlife individuals are assumed to move at random within the ecosystem for the period of the simulation of the spill. Studies have shown that while birds and mammals may sometimes try to avoid oil once they have experienced it, for the most part animals respond to overriding desires of obtaining food or other behaviors such that avoidance is negligible (Varoujean et al., 1983; Geraci and St. Aubin, 1988, 1990). While the majority of fulmars near an experimental spill were observed by Lorentsen and Anker-Nilssen (1993) to avoid oil, 4% entered oil sheen because they were attracted to food remains thrown overboard from the research vessel.

Avoidance (or attraction) is simulated in the model by adjusting the probability of intersecting oil (see below).

For each of a series of surface spilllets, the physical fates model has estimated the location and size (radius of circular spreading spilllet) as a function of time. This information is input to the biological exposure model where the area swept by a surface spilllet in a given time step is calculated as the quadrilateral area defined by the path swept by the spilllet diameter. The areas swept by all spilllets are summed over all (physical fates model) time steps in a given day, and separately for each habitat type where the oil passes. Spilllets sweeping the same area of water surface at the same time are superimposed. The total area swept by oil greater than a threshold thickness in each habitat type is multiplied by the probability that a species uses that habitat (0 or 1, depending upon its behavior) and a combined probability of oiling and mortality. This calculation is made for each surface-floating spilllet and each habitat for the duration of the model simulation. The calculations are summarized as follows:

$$N' = P_w \sum_{t=0}^{t=\infty} A_s N_t \Delta t \quad (1)$$

where N' is the total number killed of a wildlife species in a given ecosystem, P_w is the probability of oiling and dying given that a surface slick is encountered for the wildlife behavior group of the species, A_s is the portion of the ecosystem area swept by oil (greater than a threshold thickness or g/m^2 such that animal would obtain a lethal dose) over the time interval $\Delta t = 1$ day, and N_t is the number remaining alive at time t (of the species and ecosystem of concern).

3.1.2 Threshold Thickness for Lethal Dose

The threshold thickness of oil that would impart a lethal dose to an intersecting wildlife individual is 10 microns ($\sim 10 \text{ g/m}^2$), based on the following review. See Engelhardt (1983), Clark (1984), Geraci and St. Aubin (1988), and Jenssen (1994) for reviews of the literature on oil effects on aquatic birds and marine mammals.

Varoujean et al. (1983) cite that, when confined to oil, 1 g/m^2 is 100% lethal to birds oiled by such a slick, while 0.1 g/m^2 is not enough to cause acute mortality. Peakall et al. (1985) state that blue sheen (which is $< 1 \mu\text{m}$ thick, National Research Council, 1985) is not harmful to seabirds. Jenssen and Ekker (1991a,b) studied the effects of exposure of eiders to oil of varying doses. Greater than 20 ml of (crude) oil was the required dose for an effect on metabolism. However, their review of the literature revealed that an order of magnitude more oil is the required dose for significant and potentially lethal effects.

Birds incubating eggs can transfer oil to the egg from their plumage (Albers and Szaro, 1978; King and Lefever, 1979; Albers, 1980). Clutches of common eider eggs treated with 20 μl of fuel oil had significantly greater embryonic mortality than control clutches (Albers and Szaro, 1978). Hatching success was significantly reduced for mallards with plumage exposed to 100 ml/m^2 (0.1 mm) of Prudhoe Bay crude oil for 48 hours while incubating eggs (which were oiled by transfer from the

adult plumage), whereas the reduction in hatching success was not significant at 5 ml/m² of oil exposure. However, survival rates of newly hatched ducklings and adults exposed to up to 100 ml/m² oil were not significantly lowered (Albers, 1980). Mortality of mallard (*Anas platyrhynchos*) eggs treated with 1 and 5 µl South Louisiana crude oil was 35% and 91%, respectively. For chicken (*Gallus gallus*) eggs, mortality was 38%, 80% and 98% with applications of 1, 5 and 10 µl of oil, respectively (Hoffman, 1978).

Wolfe and Esher (1981) exposed rice rats (*Oryzomys palustris*) to 200 ml/m² (~200 g/m²) and 20 ml/m² (~20 g/m²) of crude oil on the water surface in laboratory test chambers with 1 m² water and two islands. In both exposures, willingness to enter the water and swim was reduced, whereas survival 24 hours later was significantly lowered in the higher exposure treatment. Survival rate was not measured beyond 24 hrs after exposure. These results suggest that mortality would occur for other semi-aquatic mammals, such as muskrat (*Ondatra zibethicus*), nutria (*Myocastor coypus*), mink (*Mustela vison*), and otter (*Lutra canadensis*) that swim through oil. River otters were observed to be killed by *Exxon Valdez* oil (Spies et al., 1996).

Little research is available to quantify oil exposure effects on sea turtles. Much of what is available is synthesized by Vargo et al. (1986). In addition to direct mechanical and toxic effects, impacts include reduced hatching rates and developmental deformities (Milton et al. 2003). For turtles of all ages, ingestion of tarballs is a major issue because turtles eat anything that appears to be the same size as their preferred prey. Ingestion can result in starvation from gut blockage, decreased absorption efficiency, absorption of toxins, buoyancy problems from buildup of fermentation gasses, and other effects (Milton et al. 2003). Inhalation of vapor is of concern for turtles since when they prepare for a dive they inhale a large volume of air before submerging. They thus have prolonged exposures to any inhaled hydrocarbons. Sea turtles have not been shown to exhibit avoidance behavior when surrounded by petroleum fumes (Milton et al. 2003).

The model utilizes an estimate of the minimum (external) dose of oil that is lethal. While there is one observation of a 70 ml dose causing a significant change in metabolic rate, 200-500 ml has been observed as a lethal dose when applied to the plumage of ducks (Jenssen, 1994). In the model, 350 ml is assumed to be the lethal dose for all wildlife. Assuming swimming bird has a width of 15 cm, it would need to swim through 23 m of oil of 100 µm thickness, 230 m of oil of 10 µm thickness, or 2300 m of oil of 1 µm thickness, to obtain a dose of 350 ml. This distance spent in oil need not be in a straight line. If an animal swims 10 m/min., 23 m would be covered in about 2 minutes; 230 m in 23 min; and 2300 m in 230 min (3.8 hrs).

To determine a dose obtained by a wildlife individual swimming through oil, the area and thickness of the oil intersected need to be estimated. The SIMAP physical fates model provides an estimate of slick size (radius of a "spillet" treated as a circle) and thickness at any given time and location. If the volume of the spillet is less than 20 ml, no effects are assumed. Spillets with greater than 20 ml of oil are assumed to oil birds sufficiently to affect hatching success (if within the nesting season for the species). If the diameter of the spillet is less than 230 m, a thickness of 100 µm is assumed as a threshold thickness for oiling mortality of wildlife. If the

spillet is larger than 230 m in diameter, 10 μm is assumed as a threshold thickness for oiling mortality.

3.1.3 Probability of Encounter with Oil and Mortality

The behavior of species influences the likelihood of their being oiled. Characteristics which make certain bird species more susceptible to oiling include: spending large periods of time on the water, weak flying capability such that they dive often, having flightless feather-molting stages, diving foraging behavior, and roosting at night on water (Speich et al., 1991). Birds that roost on land (e.g., gulls, cormorants) would have a lower probability of oiling (integrated over a daily time step). Thus, the probability of encounter with the slick is related to the percentage of the time an animal spends on the water or shoreline surface, including any diel or oil avoidance behavior.

Birds, mammals, reptiles, and amphibians (wildlife) are categorized by behavior patterns, i.e.,:

- Dabbling waterfowl and surface seabirds: surface divers (ducks, geese, swans, coots, murre) spend most of their time on the surface of the water and fly from place to place only occasionally.
- Aerial nearshore divers: birds that fly over the habitat most of the day time and dive for food (e.g., gulls, terns, osprey).
- Aerial seabirds: birds that fly over the habitat most of the time and dive occasionally (e.g., albatross) or have demonstrated oil avoidance behavior (e.g., fulmar).
- Wetland wildlife: wading birds, shorebirds, muskrats, and other wildlife typical of wetlands and shallow water habitats that walk, wade or swim in shallow water, wetlands, and intertidal or shoreline habitats.
- Terrestrial wildlife: Animals which typically walk and forage in shoreline or wetland habitats, and do so only a small percentage of their time.
- Marine wildlife: Marine mammals and sea turtles swim on the surface or live under it some percentage of the time.

Once oiled, it is generally agreed that birds have a very low survival rate, even when rescue and cleaning is attempted (Bourne et al., 1967; Holmes and Cronshaw, 1977; Croxall, 1977; Ohlendorf et al., 1978; Chapman, 1981; Ford et al., 1982; Samuels and Lanfear, 1982; Varoujean et al., 1983; Ford, 1985; Evans and Nettleship, 1985; Fry, 1987; Seip et al., 1991; Anderson et al., 2000). Death may be due to loss of body heat, toxicity through the skin, and/or ingestion of toxins via grooming. Also, death may not be immediate. Samuels and Lanfear (1982) estimated that 95% of oiled seabirds die while most of the other authors cited above estimate the probability of dying near 100%. Thus, the probability of mortality once oiled is assumed 100% for birds and fur-covered mammals (assuming they are not successfully treated) and much lower for other wildlife.

The estimated products of the two probabilities for various wildlife behavior groups are in Table 1. Estimates for the probabilities are derived from information on behavior and field observations of mortality after spills. Table 2 contains generic category estimates, also calculated in the model.

Table 1. Combined probability of oil encounter and mortality once oiled assumed for species groups, if present in the area swept by oil exceeding a threshold thickness. Area swept is calculated for the habitats occupied.

| Wildlife Group | Probability | Habitats Occupied |
|---|--------------------|--|
| Surface divers: dabbling waterfowl | 99% | Intertidal, wetland, near-shore waters, bays, lakes, ponds, rivers/streams |
| Surface divers: seabirds | 99% | All intertidal and waters |
| Nearshore aerial divers | 35% | Intertidal, wetland, near-shore waters, bays, lakes, ponds, rivers/streams |
| Aerial seabirds | 5% | All intertidal and waters |
| Wetland birds (waders, shorebirds) | 35% | All wetlands, shorelines, seagrass beds |
| Terrestrial mammals in wetlands and on shorelines | 0.1% | All wetlands, shorelines |
| Raptors (other than eagles and osprey) | 0.1% | Intertidal, wetland, near-shore waters, bays, lakes, ponds, rivers/streams |
| Cetaceans | 0.1% | All subtidal marine waters |
| Furbearing marine mammals | 75% | All intertidal and marine waters |
| Furbearing aquatic mammals | 75% | All shorelines and nearshore waters (freshwater and estuarine systems) |
| Non-fur-bearing pinnipeds, manatee | 1% | All marine intertidal and waters |
| Sea turtles (juvenile, adult) | 5% | All subtidal marine waters |
| Sea turtles (hatchlings) | 50% | All subtidal marine waters* |
| Terrestrial reptiles and amphibians | 75% | All shorelines and waters (typically freshwater systems) |

* Oiling on nesting beaches is evaluated geographically, based on mapped information.

Table 2. Combined probability of encounter with oil and mortality once oiled for generic behavior categories, if present in the habitats listed and area swept by oil exceeding a threshold thickness.

| Wildlife Group | Probability | Habitats* |
|--|--------------------|-------------------------------------|
| Surface birds in seaward habitats only | 99% | All seaward intertidal and subtidal |
| Surface diving birds in seaward habitats only | 35% | All seaward intertidal and subtidal |
| Aerial divers in seaward habitats only | 5% | All seaward intertidal and subtidal |
| Surface birds in landward habitats only | 99% | All landward intertidal and waters |
| Surface diving birds in landward habitats only | 35% | All landward intertidal and waters |
| Aerial divers in landward habitats only | 5% | All landward intertidal and waters |

| Wildlife Group | Probability | Habitats* |
|---|--------------------|------------------|
| Surface diving birds in water habitats only | 35% | All waters |
| Aerial divers in water only | 5% | All waters |

* Intertidal includes all between-tide or terrestrial areas flooded by tides or by storm surges; seaward and landward designations are operationally defined for the area modeled, e.g., marine = seaward and estuarine = landward or estuarine = seaward and freshwater = landward.

Documentation of the probability of oiling and the mortality of wildlife which have been oiled is not readily available due to difficulties in obtaining estimates. Accurate beach counts of dead animals are not enough, since many dead individuals sink or are consumed before being washed ashore (National Research Council, 1985). In studies of the *Puerto Rican* and *Apex Houston* spills (Point Reyes Bird Observatory, 1985; Page and Carter, 1986; Page et al., 1990; Carter et al., 2003), 20 to 52 percent of oiled birds recovered from beaches were dead or died following recovery. In order to estimate the total mortality of birds attributable to each of the spills, the Point Reyes Bird Observatory scientists estimated how many birds died but were not observed. From observations of the trajectory and path width of each spill, the density of birds in the area and estimates of daily carcass deposition and survival rates for beached oiled birds, they estimated the number of birds killed by the spill for seabird species groups. The estimated mortalities averaged 90% in the *Puerto Rican* spill and 68% in the *Apex Houston* spill. These rates would be overestimates to the extent that some oiled birds survived oiling but were not observed. However, they are underestimates to the extent that oiled birds flew off to other areas before dying.

This approach (termed the Beached Bird Model, Page et al., 1990; Carter et al., 2003) of estimating total mortality from counts of oiled animals has been utilized in several large oil spill cases in the US (e.g., *Puerto Rican*, *Apex Houston*, *Exxon Valdez*, *Kure*, *New Carissa*, *Stuyvesant*, *Luckenback*). The calculations include corrections for losses at sea, losses on shorelines after beaching, background non-spill-related beaching rates, and observational effort and search success (Carter and Page, 1989; Ford, 1987; Ford et al., 1996). Results of these and similar detailed studies provide data for estimating probability of oiling if a bird is present in the area swept by oil.

Studies of the *Exxon Valdez* incident provide such estimates. Gundlach et al. (1991) give an estimate of 30,000 km² of water surface swept by oil slicks and sheen. Within this area Piatt et al. (1990) estimated that 283,000 – 370,000 seabirds were present in April 1989 when mortalities occurred. Piatt et al. (1990) estimate 100,000 – 300,000 seabirds were killed or 61% percent of those present using the midpoints. U.S.A. (1991) estimated bird kills at 260,000 – 580,000, which is 78% using the midpoints. Later reports were that about 250,000 seabirds were oiled (Spies et al., 1996; Ford et al., 1996), inferring 77% of those birds present in the area were oiled. Of these, 74% were murre.

Wilhelm et al. (2007) observed 50% of murre and 8% of dovekeys on the sea surface at any given time, as opposed to flying, in the area of a crude oil spill off Newfoundland. Their mean estimate of birds oiled assumed 50% of the flying birds and all the birds on the water would intersect the oil, i.e., 75% of the murre and 54% of dovekeys. However, for the flying birds Wilhelm et al. (2007) simply picked the

mid-point of a range of up to 100% that would likely be oiled.

The above estimates are for seabirds of a variety of species, some of which are surface swimmers and would have a high probability of contacting surface slicks (e.g., murre), while other species are aerial divers and would have a lower probability of oiling (Holmes and Cronshaw, 1977; King and Sanger, 1979; Varoujean et al., 1983; Ford et al., 1982; Samuels and Ladino, 1984; Holmes, 1984; Ford, 1985; Evans and Nettleship, 1985; Seip et al., 1991). Ford (1985) estimates the probabilities of slick encounter and subsequent mortality for several bird species. His estimates average 90% for surface swimmers and 35% for aerial divers, assuming no avoidance of slicks. These values are consistent with estimates derived from spill observations cited above. Values assumed for P_w in the present model are in Table 1. For surface swimming birds, 99% is used because the majority of evidence shows that oiled birds do not survive.

Aerial seabirds spend much of their time flying above the water surface. They are not oiled in large numbers in oil spills. A probability of 5% is reasonable (Table 1). This group would also include species known to avoid oil, such as fulmars (Lorentsen and Anker-Nilssen, 1993).

For waders and shorebirds, little information is available for estimation of probability of oiling and mortality given a slick's presence. Chapman (1981) observed shorebirds along the South Texas coast as the *Ixtoc* blowout oil came ashore there. He observed up to 10% of shorebirds present were oiled on average. At the time of maximum shoreline oiling, 40% of royal terns were observed oiled, this being the most vulnerable species. However, the oil was present as mousse and tar balls over 2 months old, in scattered patches. Chapman (1981) did observe avoidance behavior, but the availability of clean beach may have facilitated this. Thus, 10% or even 40% would be too low an estimate for probability of oiling on a beach covered with fresh oil. Since these species do contact the surface much like surface swimming birds, and oil on feet was observed by Chapman (1981) to be transferred to plumage and so be ingested, probabilities of oiling and dying are most likely near the high end of this range for waders and shorebirds. A value of 35% is assumed in the model (Table 1) and applied when oil on shorelines exceeds 100 g/m^2 . At this oil thickness, a bird would need to move along a path 35 m long and 10 cm wide to obtain a lethal dose of 350 ml. Thus, the assumed threshold thickness is reasonable, as more scattered oil on a shoreline would require proportionately longer distances where birds would be in contact with oil.

For mammals, the evidence is that oiling most often causes mortality for semi-aquatic furbearers (e.g., muskrat, otter, mink, beaver) and for those marine mammals which have a fur pelage used for retaining body heat (e.g., sea otters and fur seals). This was born out in the *Exxon Valdez* incident where sea otters were the mammal species most heavily impacted (U.S.A., 1991). It was estimated that 3500 – 5500 sea otters were killed by *Exxon Valdez* oil (Spies et al., 1996). The cause of mortality for fur bearing mammals is both due to loss of body heat (exposure) and to ingestion of toxins via the frequent grooming of the pelage (Wragg, 1954; McEvan et al., 1974; Geraci and Smith, 1976; Engelhardt, 1983, 1987; Geraci and St. Aubin, 1988). It is reasonable to assume that marine and semi-aquatic furbearers will have similar sensitivity to oil (Engelhardt, 1983; and by comparison of the results of Wragg, 1954, McEvan et al., 1974, and Wolfe and Esher, 1981, for muskrats to those of the other

citations above). Ford (1985) has estimated a probability of an oiling encounter plus subsequent mortality for furbearing mammals (fur seals) at 75%. This value is assumed for all swimming furbearers in the model.

It was estimated that 200 harbor seals were killed by *Exxon Valdez* oil (Spies et al., 1996). Other non-fur-bearing marine mammals could not be definitively documented as having been killed by the *Exxon Valdez* oil, although inferences from circumstantial evidence were made (Loughlin et al., 1996). Similarly, in an earlier spill in the Santa Barbara Channel, sea lions did not appear to suffer high mortality rates from oiling (Simpson and Gilmartin, 1970; Brownell and LeBoeuf, 1971; LeBoeuf, 1971). For non-furbearing pinnipeds, a low probability of oiling and mortality of 1% is assumed; while for cetaceans 0.1% is assumed (Table 1).

No estimates are available for raptors. Since bald eagles and osprey behave as aerial divers, diving for fish in the near-shore area, the estimated probability of dying of oiling is assumed to be 35%, as for other aerial diving birds. For other raptors (hawks, owls, etc.) the probability of oil mortality would be much lower because of lower encounter frequency. An estimate of 0.1% is assumed (Table 1).

Sea turtle behavior and likelihood of exposure to oil are reviewed by Vargo et al. (1986) and Shigenaka (2003, see especially Milton et al., 2003). A summary of the major spills where impacts to turtles were observed is available in Yender and Mearns (2003). Turtles do not exhibit avoidance behavior when encountering an oil slick (Milton et al. 2003). Hatchlings are the most vulnerable stage because only a small amount of oil is needed to completely coat them. Also, since smaller turtles have more limited motility, they are often caught in the same currents as oil slicks and can end up in convergence zones more frequently than juveniles or adults. Finally, hatchlings spend more time on the surface than older turtles, thus increasing the potential for contact with oil slicks (Milton et al. 2003). Once oiled, hatchlings may not be able to swim as well, thereby increasing their predation risk.

The oiling probabilities for sea turtles were derived from the following. Data from small, attached time-depth-location data logger devices, and miniature video-cameras, indicate that adult and juvenile sea turtles at sea spend only 1 to 10 % of their time at the surface, and each dive duration is generally between 30-70 minutes (Blair Witherington, Fish and Wildlife Conservation Commission, Florida Marine Research Institute, Sea Turtle Research Station, Melbourne, FL, and David Bernhart – NOAA/NMFS Southeast Region Office of Protected Resources, St. Petersburg, FL, personal communication, September 2001). Regarding the effects of oil, literature indicates a moderate to high short-term survival rate if oiling occurs (Vargo et al., 1986). There are few definitive data regarding the long term effects of oil on any reptile. The value of 5% for this combined factor is reasonable for adult and juvenile sea turtles, based on the available information.

Field observations of hatchling sea turtle behavior at sea were described by B. Witherington (pers. comm., September, 2001). Hatchlings are very buoyant and have little ability to dive (estimated maximum dive depths are 6-7 meters and less than 1 minute duration), and may dive only to avoid predation by birds. Hatchlings spend most (~99%) of their in-water time at the surface. Their size and anatomy that would make them susceptible to passing oil and potentially dying from suffocation as a result of this exposure. Hatchlings are small (41-78 mm SCL, 18-50 g, mostly less than 25 g), can only lift their head 1 or 2 cm above the surface, and have very tiny

nares, about 0.5 mm diameter. The literature indicates hatchlings are injured by both fresh and weathered oil, but can survive in the short term (several weeks) after acute (2 day) exposure to weathered oil (Vargo et al., 1986). The likely range of probability for oiling and dying is 10-100%, with 50% as a best estimate.

Other aquatic reptiles are modeled using information for sea turtles, as these are the only reptiles where such information related to behavior and vulnerability to oil is available. Impacts to terrestrially-based turtles and snakes present in oiled habitats would likely occur at oil doses similar to those for sea turtles. However, probability of oiling of these animals (when oil is present in the habitat occupied) is likely higher than for sea turtles that spend much of their time underwater. Thus, the probability of oiling for terrestrially-based reptiles present in oiled habitats is assumed 75%, similar to fur-bearing mammals. The probability of oiling for terrestrial stages of amphibians (e.g., frogs, toads) present in oiled habitats is also assumed 75%.

In addition, any reptile eggs laid in an oiled habitat would likely be lost, as evidence shows that sea turtle eggs are killed by small doses of oil (Vargo et al., 1986). The evidence from studies on bird eggs (reviewed above) are supportive that reptile eggs should be vulnerable at low oil doses as well.

In the model, area swept is calculated for the habitats occupied by each of the behavior groups of wildlife listed in Table 1. A species or species group is assigned to a behavior group to evaluate its loss, which is calculated as the area swept multiplied by the combined probability (Table 1) and the density at the time of the spill.

The wildlife mortality model applied to birds and sea otters was evaluated with more than 20 case histories, including the *Exxon Valdez* and other large spills, verifying that these values are reasonable (French and Rines, 1997; French McCay 2003, 2004; French McCay and Rowe, 2004). Insufficient data are available to evaluate the model algorithms for other wildlife groups.

Wildlife mortality is directly proportional to abundance per unit area and the percent mortalities in Table 1. Note that the abundance of a species should be specific to the affected area at the time of the spill. For example, in the case of severe storm events such as the hurricanes, bird abundance may be lower than normal due to avoidance of the storm. Thus, the density data used for impact estimations should reflect this behavior if it is known or assumed to occur. Model uncertainty is decreased when field data are used to estimate bird density and to calibrate the probability of an at-risk bird becoming oiled.

If densities are unknown, area affected by sufficient oil to cause a lethal dose can be used as an index of potential for impacts to any wildlife present in the areas oiled. The exposure index we have used for seabirds and other offshore wildlife is the water area swept by more than 10- μ m thick ($> 10 \text{ g/m}^2$) oil, which is sufficient to provide a lethal dose, as discussed above. The probability of exposure is related to behavior: i.e., the habitats used and percentage of the time spent in those habitats on the surface of the water. For shorebirds and other wildlife on or along the shore, an exposure index is length of shoreline oiled by $> 100 \text{ g/m}^2$. Areas of exposure above these thresholds have been used in environmental risk assessment studies (French McCay et al., 2003a, 2004, 2005a,b,c).

3.2 Aquatic Biota: Fish, Invertebrates, and In-Water Stages of Amphibians

The most acutely toxic components of oil to water column and benthic organisms are low molecular weight compounds, which are both volatile and soluble in water, especially the aromatic compounds (Neff et al., 1976; Rice et al., 1977; Neff and Anderson, 1981; Malins and Hodgins, 1981; National Research Council, 1985, 2002; Anderson, 1985; McAuliffe, 1987; and French McCay, 2002). This is because organisms must be exposed to hydrocarbons in order for uptake to occur and aquatic biota are exposed primarily to hydrocarbons (primarily aromatics) dissolved in water. Thus, exposure and potential effects to water column and bottom-dwelling aquatic organisms are related to concentrations of dissolved aromatics in the water. Exposure to microscopic oil droplets may also impact aquatic biota either mechanically (especially filter feeders) or as a conduit for exposure to semi-soluble hydrocarbons (which might be taken up via the gills or digestive tract).

The effects of the uptake and accumulation of the dissolved hydrocarbon components in tissues are additive. Thus, an additive acute toxicity model and available LC50 data for individual compounds (under known temperature and duration of exposure conditions) may be used to estimate the LC50 (lethal concentration to 50% of exposed organisms) of the mixture of monoaromatic hydrocarbons (MAHs) and PAHs in oil to which aquatic organisms are exposed. Note that the LC50 is a proxy measurement for the concentration that leads to sufficient uptake into tissues for lethal effects to occur for 50% of individuals exposed. (See French McCay (2002) for a detailed explanation of the derivation of the LC50-based model commonly used in aquatic toxicology, which is briefly summarized below). Oil toxicity is a function of MAH and PAH content and composition in the oil. The toxicity of oils and refined products has been estimated and verified with available bioassay data. The verified oil toxicity model may be used to estimate toxicity of untested oils under varying environmental conditions (French-McCay, 2002).

Hydrocarbons accumulate in lipids (such as in the cell membranes) and disrupt cellular and tissue function. The more hydrophobic is the compound, the more accumulation in the tissues and the more severe the impact. However, the more hydrophobic the compound, the less soluble it is in water, and so the less available it is to aquatic organisms. Thus, impact is the result of a balance between bioavailability (dissolved-component exposure) and toxicity once exposed (see review in DiToro et al., 2000).

PAHs are more hydrophobic than MAHs, and so are more toxic. There is a continuum from the most soluble and least toxic benzene (simplest MAH) through the naphthalenes (2-ring PAHs) to the 3- and 4-ring PAHs. The more complex 4-ring PAHs are so insoluble that they are not dissolved or (acutely) bioavailable to a significant extent. This functional relationship can be described by a regression model using available data on a variety of compounds and species (French-McCay, 2002). A similar approach has been used to develop US Environmental Protection Agency (USEPA) water and sediment quality criteria for PAHs (DiToro et al., 2000; DiToro and McGrath, 2000).

Because of the relative solubility and volatility of various MAHs and PAHs, and the relative concentrations of the various compounds in oil, most of the acute toxicity is caused by the PAHs, and specifically the substituted naphthalenes (C2- and

C3-naphthalenes). However, all the compounds in the mixture contribute to toxicity (French-McCay, 2002).

Mortality is a function of duration of exposure – the longer the duration of exposure, the lower the effects concentration (see review in French McCay, 2002). This is due to the accumulation of toxicant over time up to a critical tissue concentration that causes mortality. The accumulation is slower for more hydrophobic compounds. The accumulation is also slower at colder temperature. Thus, for brief exposures at low temperature, toxic effects require a higher concentration than would be necessary at higher temperature or for instances where exposure times are longer (see Figure 3). At a given concentration after a certain period of time, all individuals that will die have done so. The incipient LC50 (LC50_∞) is the asymptotic LC50 reached after infinite exposure time (or long enough that the asymptotic level is approached, Figure 3). At a given exposure duration, percent mortality is a log-normal function of concentration, with the LC50 the center of the distribution.

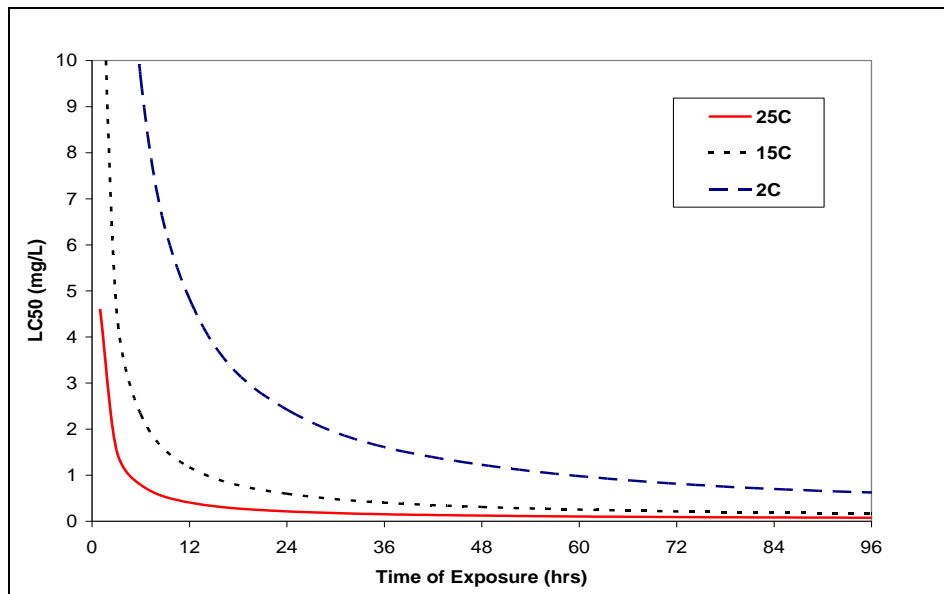


Figure 3. LC50 of dissolved PAH mixtures from oil, as a function of exposure duration and temperature (French McCay, 2002).

For most fuel and crude oils (where PAHs cause most of the toxicity), the value of LC50_∞ ranges from 5-400 µg/L for 95% of species (Figure 4) exposed to dissolved PAH mixtures for over 96 hrs (which is sufficient time to approach the asymptote, as seen in Figure 3; French McCay, 2002). The LC50_∞ for the average species is about 40-50 µg/L (ppb) of dissolved PAH (varying slightly among oils and fuels by percent composition of the PAH mixture). These LC50_∞ values have been validated with oil bioassay data (French McCay, 2002), as well as in an application of SIMAP to the *North Cape* oil spill where field and model estimates of lobster impacts were within 10% of each other (French McCay, 2003).

Mortality of fish, invertebrates, and their eggs and larvae is computed as a function of temperature, concentration, and time of exposure. Percent mortality is estimated for each of a large number of Lagrangian particles representing organisms

of a particular behavior class, i.e., planktonic, demersal (on the bottom), and benthic (in bottom sediments), or fish (or invertebrate nekton) that are classed as small pelagic (slower swimming), large pelagic (faster swimming), or demersal (near bottom) and occupying specific habitats (open water, wetland or reef; varying by seaward and landward habitats, as defined above). For each Lagrangian particle, the model evaluates exposure duration to dissolved hydrocarbons, and corrects the $LC50_{\infty}$ for time of exposure and temperature (Figure 3; see French McCay (2002) for equations). Percent mortality is then calculated from the mean exposure concentration, C , and the corrected $LC50_t$ using a log-normal function (with 50% mortality at $C = LC50_t$, 1% mortality at $C = LC50_t/100$, and 99% mortality at $C = 100 \times LC50_t$). The percent mortalities are summed, weighed by the area represented by each Lagrangian particle to estimate a total equivalent volume for 100% mortality. In this way, mortality is estimated on a volume basis, rather than necessitating estimates of species densities to evaluate potential impacts. In addition to the mortality estimates, the volume exceeding $1 \mu\text{g/L}$ total dissolved aromatics may be used as an index for exposure for fish, invertebrates, and plankton. The algorithms for these calculations are described in French McCay (2002, 2003, 2004).

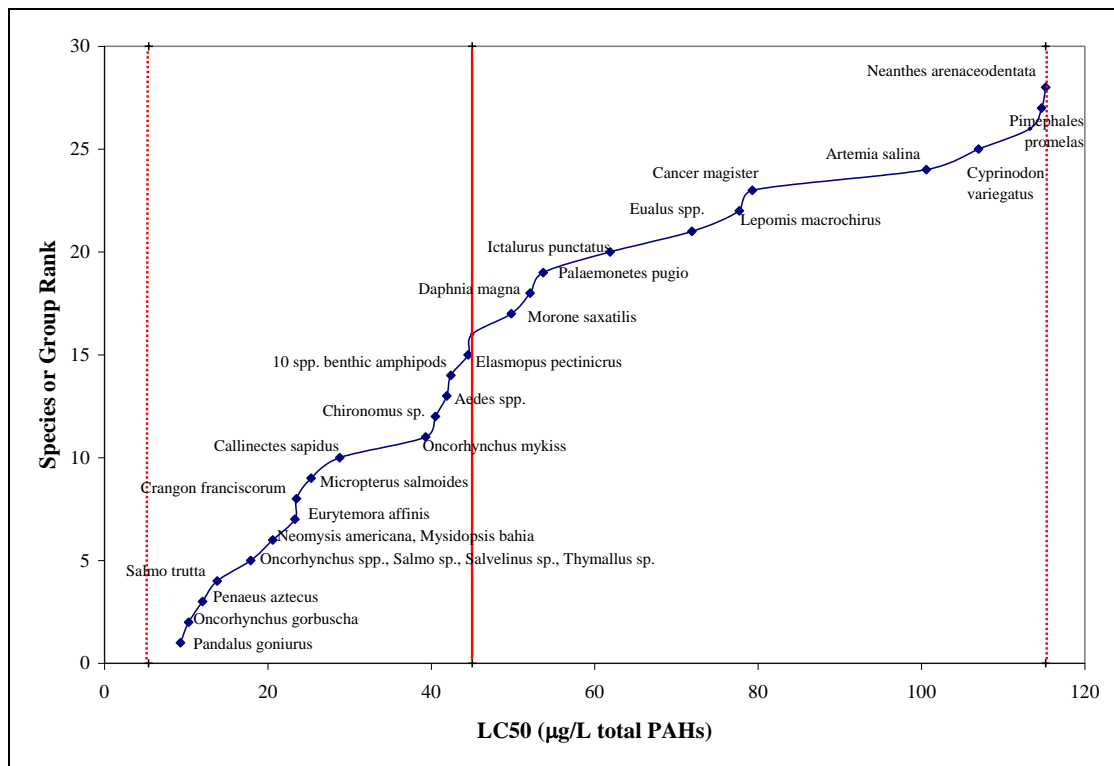


Figure 4. Variation in $LC50_{\infty}$ for dissolved PAH mixtures from a typical oil, by species in rank order of sensitivity (French McCay, 2002).

3.3 Sublethal Effects on Primary and Secondary Producers

Three primary producer categories are designated in the model: phytoplankton, benthic microalgae and macrophytes (macroalgae and/or angiosperms). Secondary and tertiary producers (primary and secondary consumers) which are not evaluated

except through the food chain are divided into eight trophic categories: zooplankton, benthos, the air-breathing stages of insects, planktivorous forage fish, herbivorous forage fish, benthic forage fish, insectivores (e.g., moles, shrews) and small herbivorous mammals (e.g., rodents). Early life stages of insects which are aquatic are considered part of the benthos or zooplankton.

Estimates of primary production and secondary production are included in the biological database input to the model for each habitat type. Since organic carbon accumulation in sediments is less than 10% of supply in most environments, all primary production by phytoplankton and benthic microalgae is assumed to be consumed by the water column organisms and benthos (Hargrave, 1973).

Phytoplankton are assumed to inhabit the upper water column (or entire water column in shallow water or if it is mixed to the bottom) and to be uniformly distributed with depth. Thus, their exposure is to the concentration field in the surface water layer. Zooplankton are assumed to be distributed throughout the water column and are exposed to contaminant concentrations at all depths. Macrophytes, present only in shallow-water habitats, are assumed to be exposed to the average concentration over the entire water column. Benthic microalgae and the benthos are assumed to be exposed to sediment pore water concentrations.

For each time step and for each of the concentration grid cells output by the physical fates model, lost primary, zooplankton and benthic production (P_L) is calculated as follows:

$$P_L = (1 - F_k) P_i V \Delta t \quad (2)$$

where F_k is the fraction of the uninhibited rate of production which is realized at the contaminant concentration, P_i is the rate of production (g dry weight m^{-3}/day^{-1}), V is volume contaminated (m^3), and Δt is the number of days contaminated. [Appropriate conversion factors for biomass and production are 12.5 from g C to g wet weight, and 40-45% of dry weight is carbon (Odum, 1971).] This calculation is performed for each habitat grid cell and vertical section of the water column affected by toxic concentrations, at each time step (Δt). Total production loss is summed over time and space.

The value of F_k is calculated from the EC50 for growth, corrected for temperature, using the log-normal toxicity model (Section 3.2) relating cumulative response (in this case, percent growth rate, F_k) to concentration. However, unlike for LC50's, the EC50 is not corrected for exposure time, i.e., reduction in growth rate is assumed to be uniform over time. Since P_i is a rate per time, multiplication by Δt accounts for exposure time.

Concentrations from the spill are assumed to either be toxic (if the concentrations are high enough) or to have no effect on productivity. Although stimulation of phytoplankton primary production has been noted in a few studies, in the vast majority of studies, negative effects have been observed (National Research Council, 1985).

Biomass of upper trophic level biota which is not produced as a result of a loss of food resources from affected lower trophic levels (i.e., P_L) is estimated using a simple food web model. The portion of the lost primary production which would

have produced primary consumer (secondary producer or herbivore) biomass is estimated based on observations of ecological efficiency made on representative ecosystems. Angiosperm biomass passes up the food web primarily via detritivores consuming the plant material and (more importantly) the attached microbial communities (Teal, 1962; Odum and de la Cruz, 1967; Thayer et al., 1984; Howes et al., 1985; Newell and Porter, 2000). The detritivores are then prey for larger animals (e.g. in marshes: decapods, such as grass shrimp, *Palaemonetes pugio*, and small fish, such as the mummichog, *Fundulus heteroclitus*, and other killifishes) and ultimately support production of recreationally and commercially important finfish, shellfish, waterfowl and wading birds (Teal, 1962). The ecological efficiency of detritivore production per unit primary producer production is low because a high percentage of biomass produced by the plant is broken down by microorganisms (primarily fungi at a transfer efficiency of 55%: Newell and Porter, 2000) before it can be assimilated by detritivores. Benthic meiofauna and macrofauna also directly consume benthic and epiphytic microalgae directly. French McCay and Rowe (2003) estimated the transfer efficiencies from plants to detritivores as 3.4% in (*Spartina*-dominated) saltmarsh and 7.2% in seagrass beds. Transfer efficiencies for woody vegetation would be lower than these values. As a general value, 4% is assumed in the model.

Values for production of predator per unit production of prey (i.e. ecological efficiency) for invertebrate and fish consumers of animal prey have been estimated to be 10-30% in both freshwater and marine environments by a number of authors (e.g. Slobodkin, 1960; Odum, 1971; Steele, 1974; Cohen et al., 1982; Pimm, 1982; Pauly and Christensen, 1995; Jennings et al., 2002). In the model, the transfer efficiency of fish and invertebrates consuming animal prey is assumed 20%.

For birds and mammals (which as homeotherms are less efficient), ecological efficiency is much lower, with estimates ranging from 1-5% (McNeill and Lawton, 1970; Steele, 1974; Whittaker, 1975; Grodzinski and Wunder, 1975; Pimm, 1982). In the model, the ecological efficiency of birds and mammals feeding on fish or invertebrate prey is assumed to be 2%.

Figure 5 shows the food web compartments used in the model. The fractions of the lost secondary production which would have been consumed by each of their predators are assumed to be proportional to the biomass of that predator relative to the sum of its competitors. The fraction of prey compartment j 's production which is consumed by predator i , a_{ij} is:

$$a_{ij} = (\rho_i B_i^{3/4}) / \sum_k (\rho_k B_k^{3/4}) \quad (3)$$

where B_i is biomass per unit area of predator i , B_k is the biomass per unit area of predator k which preys on j , where k represents all predators of prey j (including i), and ρ_i or ρ_k represents the ratio of consumption to biomass of the predator (i or k) relative to consumption to biomass of fish.

The correction factor ρ is based on the fact that homeotherms (birds and mammals) have higher metabolic rates at a given body size than poikilotherms (fish and invertebrates) and so have a higher consumption to biomass ratio (Zeuthen, 1953; Hemmingsen, 1960; Fenchel, 1974). Respiration rate has been shown to be proportional to body weight (W) to the 3/4 power (Kleiber, 1947; Zeuthen, 1953;

Hemmingsen, 1960; Fenchel, 1974). Fenchel (1974) showed that the ratio of respiration rate to $W^{3/4}$ (where W = body weight) between homeotherms and poikilotherms is 28 (on average). Assuming food consumption is proportional to respiration rate, wildlife consumption per $W^{3/4}$ is 28 times fish consumption per $W^{3/4}$. In the biological submodel, all fish and invertebrates are assumed to have the same consumption to biomass ratio. Birds and mammals each are assumed to have 28 times higher consumption to biomass ratios at a given body size.

The total production of compartment i (P_i) is then equal to the sum of its consumption of prey production times the ecological efficiency, ε_i for the predator i .

$$P_i^{pred} = \varepsilon_i \sum_j a_{ij} P_j^{prey} \quad (4)$$

where P_j is the production for prey compartment j . If the predator has no competitors, it consumes 100% of that prey's production.

Production rate estimates of primary producers (angiosperms and algae) and benthos are input to the model. Production rates of zooplankton, forage fishes, insects and secondary consumers not evaluated by individual species are calculated from their prey production rates and the trophic transfer efficiencies described above. For upper trophic level fish, invertebrate and wildlife species, biomass estimates are input to the model. Thus, in order to estimate production loss via a prey compartment where only biomass is known, i.e., for all upper trophic level losses resulting from lost primary and secondary production, the ratio of annual production to biomass (P/B) is assumed to be as derived by Banse and Mosher (1980), as follows: P/B = $0.65 M_k^{0.37}$ for invertebrates, P/B = $2.75 M_k^{0.26}$ for fish; and P/B = $12.88 M_k^{0.33}$ for birds and mammals; where M_k is body mass in kcal (1 kcal/g wet weight for invertebrates, 1.3 kcal/g wet weight for fish, and 1.5 kcal/g wet weight for birds and mammals).

In calculating lost production of consumers in the food web, a proportionate loss of lower trophic level production is translated to a proportionate loss higher in the food web. For example, a 30% loss in phytoplankton is translated to a 30% loss in all of the upper trophic level production rates which are dependent ultimately on phytoplankton production. However, in the case where toxicity has reduced zooplankton, benthic or forage fish production to a greater degree than the reduction of primary production, consumers of zooplankton and benthos and forage fish are assumed to suffer proportionate losses to the losses for these food resources. Thus, zooplankton, benthos and forage fish are assumed either food limited or toxicant limited, whichever is greater.

Larvae of fish and invertebrates are assumed to feed entirely on zooplankton and to be food-limited. Feeding studies on fish larvae have shown that survival of larvae is dependent on finding high enough densities of food (Munk and Kiorboe, 1985). Thus, in the model it is assumed that reduced zooplankton production causes a proportionate reduction in larval (young-of-the-year) numbers. This is termed "indirect kill," as opposed to the direct kill via toxicity to larvae.

The output of this part of the model is lost production at lower trophic levels translated into losses of upper trophic level species (fish, invertebrates, and wildlife)

as a result of the spill. This is added to the total direct kill to yield a total loss of biomass by habitat as a short term result of the spill.

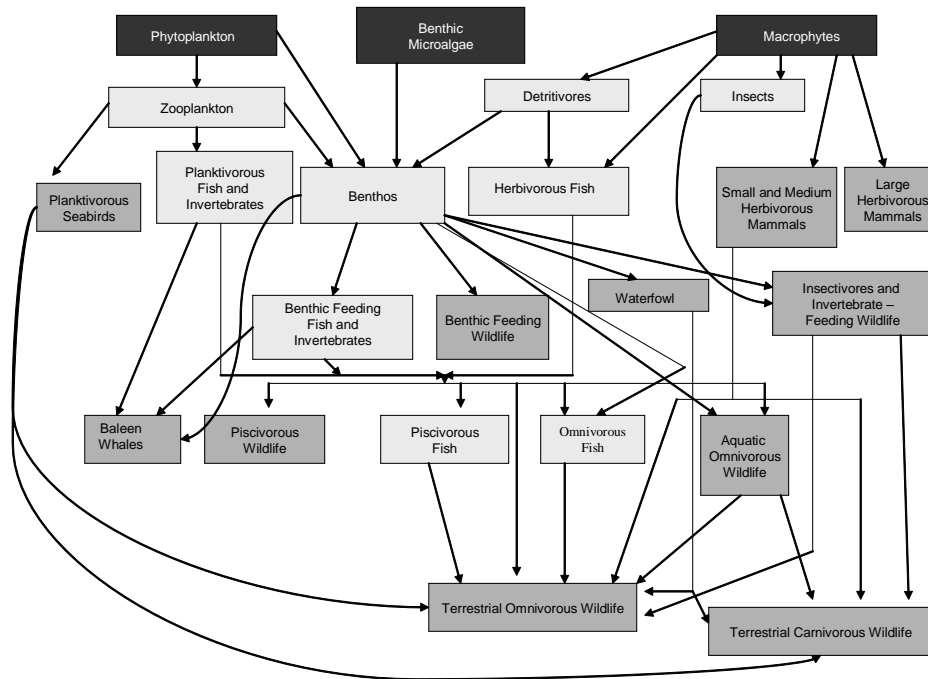


Figure 5. Food web compartments used in the model.

3.4 Intertidal, Wetland, and Terrestrial Plants and Invertebrates

In addition to the quantifying impacts in water habitats, the oiling of intertidal (wetland, rocky shore, gravel and sand beach, and mudflat) and temporarily flooded habitats with enough oil to impact plants and invertebrates may be evaluated. This is calculated as the area oiled above threshold(s) for injury times the habitat- and species-specific density or production rate of biota in that habitat and area.

Alexander and Webb (1985, 1987), Cubit et al. (1987) and Moody (1990) document mortality or other impacts on wetland vegetation by oiling in significant quantities. For submerged macrophyte and seagrass beds, dissolved concentrations of oil hydrocarbons are assumed to affect production, not surface floating oil. Exposure to floating oil is assumed lethal at the thresholds established below if the habitat area is not flooded with water at the time of exposure. No data are available to quantify effects thresholds for sunken oil on macrophytes.

Based on review of the literature performed in the late 1980s, French et al. (1996) assumed a threshold of 14 mm as lethal to wetland vegetation in the Type A model (NRDAM/CME). Alexander and Webb (1985) reported that 0.35 gal./ft² had a detrimental effect on Texas saltmarsh plants (*Spartina* spp), which is equivalent to about 14 mm-thick oil. The review by Moody (1990) supported the 14 mm threshold level. The Ile Grande marsh oiled by 50-200 mm of crude oil after the *Amoco Cadiz* spill was completely killed (Johnson and Pastorok, 1985). However, other data suggests the threshold for effects is lower than 14 mm. Baker (1971) reported that 0.5 mm of fresh Kuwait crude (a light oil) spread over saltmarsh vegetation was lethal

when applied during the growing season (May or August), but if applied in November, growth in the following season was not affected. Baca et al. (1987) measured oil thickness in marshes where vegetation was killed by a heavy fuel spill in the Cape Fear River (NC) in 1982, finding 0.25-0.5 mm of heavy fuel oil coat on leaves of *Spartina* saltmarsh plants to be lethal and that the surface area of the affected marsh was covered with about 10 mm of oil. The reviews by Johnson and Pastorok (1985) and Shigenaka (2002) summarize other literature and case histories, and indicates that coating of the leaves is necessary for plant mortality.

Lin and Mendelssohn (1996) applied south Louisiana crude oil to natural marsh sods at rates of 0, 4, 8, 16 and 24 L/m² (1 L/m² is approximately 1 mm of oil), observing reduction in plant biomass above 4 L/m². The year following oil application, no regrowth of *Spartina patens* and *S. alterniflora* occurred at oil levels above 8 L/m². Lin et al. (2002) performed additional experiments with No. 2 fuel oil (diesel) applied to *S. alterniflora*, finding a significant decrease in total (above- plus below-ground) plant biomass at concentrations above 57 mg/g dry soil (= 2.5 L/m² = 2.5 mm) of oil. A significant decrease in below-ground biomass compared to the control was identified at a dosage as low as 29 mg/g dry soil (= 1.2 L/m² soil = 1.2 mm), but not at 14 mg/g (= 0.6 L/m² soil = 0.6 mm).

Based on these observations, it appears that more than 1 mm of oil during the growing season would be required to impact marsh or mangrove plants significantly. Thus, 1 mm is the assumed lethal threshold for wetland vegetation.

Numerous reports document suppression of intertidal invertebrate densities on visibly oiled shorelines and that invertebrates are more sensitive to oiling than intertidal macrophytes (e.g., Stirling, 1977; Boucher, 1980, 1985; Gilfillan et al., 1981; Cubit et al., 1987; Jackson et al., 1989; McGuinness 1990; Garrity and Levings, 1993; Burns et al., 1993; Clarke and Ward, 1994; Ansari and Ingole, 2002; Yamamoto et al., 2003; Teruhisa et al., 2003; Anderson et al., 2008). However, specific measurements of the amount of oil required to lethally impact invertebrates are lacking. Owens and Sergy (1994) define oil “stain/film” as <0.1mm, oil “coat” as 0.1-1mm, and oil “cover” is 1-10mm. For benthic epifaunal invertebrates living in intertidal habitats on hard substrates, a threshold of 0.1 mm oil thickness would be enough to coat the animal and likely impact its survival and reproductive capacity, while stain (<0.1 mm) would be less likely to have an effect. Thus, 0.1 mm (100 g/m²) of oil is assumed as the lethal threshold for invertebrates on hard substrates (rocky, artificial/man-made, rip-rap, etc.) and sediments (mud, silt, sand, or gravel) in intertidal habitats.

The impact thresholds are 1 kg/m² (1 mm) for vegetation and 100 g/m² (0.1 mm) for invertebrates. Injuries to wetland and intertidal biota oiled at these levels or higher are calculated as the product of the normal production rate (g dry weight m⁻² day⁻¹) and the m²-days of loss realized over a recovery period where production hyperbolically increased back to the pre-spill level over a specified number of years (based on literature documenting such recovery rates). Discounting at 3% per year is included to translate losses in future years (interim loss) to present-day values. The discounting multiplier for translating value n years after the spill to present value (i.e., for the year of the spill) is calculated as $(1+d)^{-n} = 1/(1+d)^n$, where $d=0.03$.

The recovery of plant or invertebrate production in an oiled habitat is assumed to follow a sigmoid function described by:

$$\frac{dP_R}{dt} = a_r P_R (1 - P_r) \quad (5)$$

where P_R is portion recovered, t is time, and a_r is a constant. The sigmoid function was chosen since, at first, recovery is slow while seeding/settlement and early succession takes place. Later recovery speeds up as filled-in vegetation and new settlers grow rapidly, but the final establishment of the mature habitat proceeds at a slower rate. The value of the constant a_r is derived from solution of the equation assuming P_R at $t = 0$ is 0.01 and P_R at $t = t_{rec}$ is 0.99, where upon the above equation may be solved using

$$P_R = 1/(1 + 99 \exp(-a_r t)) \quad (6)$$

What is needed is one data point of P_R at some time t . At $t = t_{rec}$, $a_r = 9.19/t_{rec}$.

In the model, losses are integrated over time using this recovery curve equation. The loss in the time interval t to $t+\Delta t$ (P_L) is calculated as follows:

$$P_L = (1 - P_R) P_i A \Delta t \quad (7)$$

where P_i is the normal (pre-spill) production rate in the habitat, A is the area affected, and Δt is the time step.

Literature regarding the recovery rate of vegetation or other habitat structure after the structural organisms are killed or severely damaged is reviewed below and summarized in Table 3. Assumed values of t_{rec} , the time to 99% recovery, are specific to habitat type and are based on experiences from observations of natural recovery following disturbance (including spills) and from habitat creation projects. Much of the wetlands creation and restoration literature emphasizes that correct hydrology be established on the site before planting, that soils are fertile, and that the site must be properly maintained and monitored (Mancini, 1989). If these conditions are met, recovery of structure and function is much more rapid and successful. As the recovery in the model is for areas which are naturally those habitats, it is assumed that the hydrology and soils remain, and that recovery is as rapid as could be expected.

Time for recovery (to 99% function) for intertidal invertebrates (based on a natural recovery curve) is estimated as 3-5 years (French et al., 1996; Table 3; also see review below). It is assumed that the affected areas are not cleaned in a manner that would slow recovery.

Table 3. Recovery rates for vegetation or other structural organisms in habitats, and for benthic invertebrates where habitat structure is not impacted (based on review below).

| Habitat | Description | Vegetation or Structure: Years to 99% Recovery | Benthic Invertebrates: Years to 99% Recovery |
|-------------------------|---|--|--|
| Rocky Shore | Rocky shore (including rock seawalls) without significant seaweed cover | - | 3 |
| Artificial Shore | Vertical piers, seawalls made of artificial, man-made materials | - | 3 |
| Gravel Beach | Gravel or cobble beach without significant seaweed cover | - | 3 |
| Sand Beach | Coarse- or fine-grained sand beach | - | 3 |
| Mud Flat | Silty-mud intertidal flats | - | 3 |
| Submerged Aquatic Bed | Seagrass or other submerged angiosperm-dominated habitats | 10 | 3 |
| Wetland: Emergent Marsh | Saltmarsh dominated by <i>Spartina</i> spp.; brackish marsh; intermediate marsh | 15 | 5 |
| Wetland: Swamp | Forested wetlands; shrub-scrub wetlands | 20 | 5 |
| Macroalgal Bed | Kelp or other seaweed | 15 | 5 |
| Coral Reef | Reefs dominated by coral | 30 | 30 |
| Mollusk Reef | Oyster or mussel reef | 3 | 2 |

Rocky, Man-made and Artificial Shores

The *Esso Bernicia* spill in 1978 oiled rocky shorelines of the Shetland Islands and recovery was monitored for nine years. Shores which were left untreated had nearly recovered by one year later, whereas those shores which were cleaned and where the biota were obliterated had not recovered after nine years (Rolan and Gallagher, 1991). Houghton et al. (1991) observed that Prince William Sound rocky shores which were cleaned after the *Exxon Valdez* spill would take many years to recover. Broman et al. (1983) also observed that hot water cleaning after an oil spill in the Baltic Sea did more harm than good and slowed recovery dramatically. From Southward and Southward (1978), recolonization and recovery of rocky shores in Cornwall, England after the *Torrey Canyon* spill took 5-8 years if the shores were lightly oiled and received light dispersant treatment. Recovery took 9-10 years or more if the shore received repeated dispersant treatment. No sites were observed (or available) that were left untreated.

Baker et al. (1990) reported that rocky shores in the Baltic Sea had nearly recovered by one year after the *Tsesis* spill of 1977. As cited by Ganning et al. (1984) cleaning slows recovery: recovery from a medium fuel oil spill followed by

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mechanical cleaning in the Baltic Sea took four years, recovery from a Bunker C spill in Nova Scotia took greater than six years, and recovery from a No. 2 fuel oil spill in Baja California took over ten years. In contrast, Keller and Jackson (1991) summarize recovery of intertidal rock reefs in Panama following a medium crude oil spill as complete by one year. Yamamoto et al. (2003) and Teruhisa et al. (2003) documented decreased densities of invertebrates and vegetation in areas heavily oiled by the *Nakhodka* spill in Japan (but not cleaned). The flora and fauna recovered in 3 years. Jones et al. (1998), based on their own observations and data in Sell et al. (1995), estimate 2-5 years for recovery of rocky intertidal communities.

In the model, three years is assumed for 99% recovery (Table 2-2). Artificial shores, and rock and gravel beaches are assumed to have the same recovery rates.

Sand Beaches and Mud Flats

Keller and Jackson (1991) summarized recovery of sand beaches in Panama following oiling as being complete by 1 year, except for certain species. Bodin (1988) observed recovery of three sand beaches in Brittany, France after the *Amoco Cadiz* oil spill over the years 1978 to 1984, stating recovery of the meiofauna was complete by 1983 (5 years). Thomas (1978) observed recovery of invertebrates after 3 years on beaches oiled by the 1970 *Arrow* spill of Bunker C oil. Baker et al. (1990) cite evidence from the Baltic Sea after a 1970 spill of medium and heavy fuel oil with mechanical cleanup where recovery took four years. Judd et al. (1991) observed that Texas dune vegetation took 2-3 years to recover from removal experiments.

Thus, recovery rate is variable, depending on conditions and initial disturbance during the spill response. A median value of three years is assumed in the model (Table 3). Mud flats are assumed to recover at the same rates as sandy beaches.

Seagrass and Submerged Aquatic Beds

If seagrasses are not killed, and only leaves are injured, recovery can be rapid. Jacobs (1980) observed that eelgrass oiled by the *Amoco Cadiz* oil spill had blackened leaves and returned to normal productivity almost immediately. However, there were massive kills of invertebrates. The invertebrate community had mostly recovered by 2 years. In Panama, oil did not induce a total kill of seagrass beds, and they had recovered by 7 months after the spill (Keller and Jackson, 1991). Zieman and Zieman (1989) reviewed recovery rates for Florida seagrass beds. Their estimates are 6 months to 1 year for leaf damage, and 5 years to decades if rhizome damage is severe and no replanting is performed. They estimate it takes 2-5 years for invertebrates to recolonize a *Thalassia* bed.

In the model, if the vegetation is lost, it is assumed that replanting is performed, eliminating the recolonization lag. Recovery of the seagrass bed is assumed to take 10 years. Recovery of freshwater wetlands dominated by grasses or submerged angiosperms is assumed to take 10 years as well. If seagrass or other vegetation is not lost, invertebrates are assumed to recover in 3 years. Three years is assumed to be the recovery time for benthos in other benthic habitats.

Saltmarsh and Other Emergent Wetlands

Getter et al. (1984) state that recovery of saltmarshes after oiling takes 5-20 years, assuming no cleanup or restoration actions are performed. With cleanup and

restoration (replanting), they estimate three years to vegetative cover. Baca et al. (1987) observed that marshes oiled by the *Amoco Cadiz* spill, but not cleaned or treated, had mostly recovered by 8 years.

Joy Zedler and colleagues (Pacific Estuarine Research Laboratory, PERL) followed recovery of experimentally created saltmarsh in Southern California for 7 years. In PERL (1990), they reported 60% recovery by 5 years. Two years later, recovery has not progressed significantly further (Joy Zedler, personal communication). Using the recovery model above, $t_{rec} = 13$ years if 60% recovery is in 5 years and $t_{rec} = 18$ years if 60% recovery is in 7 years.

The 130 acre Muzzi marsh in San Francisco Bay was observed for 10 years of natural recolonization. *Spartina* was dense in the channels by 5 years. In the marsh plain, it was still developing after 10 years. Species composition changed over time (Faber, 1991).

Blair (1991) reported that a saltmarsh created in Chesapeake Bay in 1984 was successful. He stated that it was nearly 100% recovered by 7 years to "almost" like a natural marsh.

Broome et al. (1986) reported on a created marsh along an eroding shoreline in North Carolina. *Spartina alterniflora* was transplanted and monitored for 10 years. The vegetation was equal to a natural marsh by 4 years. Other saltmarsh biota were not monitored.

Dunn and Best (1983) reported natural reestablishment of emergent freshwater marshes in Florida in 4-5 years. D'Avanzo et al. (1989) reports variable rates for freshwater marshes with recovery taking 2-5 years in some cases, but 15-30 years in others where soils used had no initial organic matter content.

Given the variability of recovery rates and of criteria used to estimate them, the literature does not provide a consensus of recovery time estimates. Where saltmarsh structure is lost, 15 years is assumed required for full recovery of all parts of the ecosystem (not just the vegetation). If the vegetation is not lost, 5 years is assumed for recovery of other functions, including invertebrate populations.

Forested and Shrub Wetlands (Swamps)

In examining a 6 ha mangrove forest killed after a JP-5 jet fuel spill, recovery was estimated as requiring 10 years after replanting (Ballou and Lewis, 1989). Lewis (1983) reviewed case histories of oil spills in mangroves. For one example, after a crude oil spill on St. Croix, U.S. Virgin Islands, there was no recolonization after 7 years. Recovery was estimated to require 10-50 years. Getter et al. (1984) estimated recovery after oil spill at 25-30 years if restoration were performed. Research Planning, Inc. (RPI, 1987) estimated 20 years for recovery of an oiled mangrove forest. Keller and Jackson (1991) speculated that for mangroves oiled and killed in Panama "full recovery may require many decades". Mature trees are 50-70 years old. Cubit et al. (1987) summarizes mangrove mortality and the more severe mortality of associated animals in these Panama habitats. Recovery was not complete after 5 years of monitoring. Additional review of literature on recovery of mangroves after oil spills is available in Hoff (2002).

In the model, it is assumed that replanting is performed (Section 5), eliminating the recolonization lag. Recovery of the mangrove swamp is assumed to take 20 years.

Macroalgal Beds

The most important macroalgal bed habitat is the kelp bed of the Pacific coast (*Macrocystis* spp.). Moody (1990) estimated that oiled kelp beds had 90% recovered in 3-4 years. However, after 14 years they had not fully recovered to pre-spill diversity. Foster and Schiel (1985) reviewed a 1957 oil spill in Baja California. There was massive mortality of invertebrates, but vegetation damage was less obvious. Vegetation increased rapidly (due to reduce grazing) by one year later. By four years later, most animal populations had recovered, but a few had not by six years. In the model, recovery time for the vegetation is assumed 15 years and for invertebrates where vegetation is unaffected, 5 years.

Coral Reefs

It is generally agreed that recovery of coral reefs after total destruction of the reef is very slow, but small scale impacts may take much less time for recovery. Brock et al. (1979) performed defaunation experiments on patch reefs in Hawaii. With all (and only) fish removed from small reefs, recovery took one to two years. Fucik et al. (1984) estimates that recovery after small scale localized destruction requires less than ten years. After heavy destruction, recovery takes 10-20 years. Severe impacts require several decades for recovery.

Keller and Jackson (1991) summarized observations on oiled coral reefs in Panama. At 0.5-3.0 m depth, 76% of coral was killed. At 3-6 m depth, 56% of coral was killed. Recovery was speculated to require more than a decade. There was no recovery after two years of observations. Additional review of literature regarding impacts and recovery of coral reefs after oil spills is available in Shigenaka (2001).

In the model, it is assumed that recovery following mortality of a reef requires 30 years (to 99% recovery).

Oyster Reef

Lenihan et al. (2001) found that the fish community compositions and species abundances on oyster reefs restored 6 years before sampling were largely indistinguishable from those on natural oyster reefs. Peterson et al. (2003) evaluated changes in abundance of fish and large mobile crustaceans on oyster reefs over time, finding that for reefs constructed in summer, development of fish and mobile crustacean abundance is virtually complete by the next spring-summer season. Densities do not increase in successive years (Grabowski, 2002). Thus, recovery of reef invertebrates, given structure already present, requires 1-2 years, depending on the timing of the spill impacts. Two years is assumed in the model.

However, if the reef structure is destroyed (e.g, during storms, Livingston et al., 1999), recovery of oyster reefs would take more time. Three years is assumed in the model.

3.5 Quantification of Fish and Invertebrate Impact as Lost Production

3.5.1 Approach

The biomass (kg) of fish and invertebrates killed represents biomass produced before the spill. In addition to this impact, if the spill had not occurred, the killed organisms would have continued to grow until they died naturally or to fishing. This lost future (somatic) production is estimated and added to the direct kill to calculate

the total production foregone. The loss is expressed in “present day” (i.e., year of the spill) values using a 3% annual discount rate for future losses.

In a natural resource damage assessment (NRDA, based on US regulations and practice), restoration should compensate for this loss. The scale of restoration needed is equivalent to production lost when both are expressed in values indexed to the same year, i.e., the injury (impact) inflated to the year restoration occurs or the restoration discounted back to the year of the spill.

Interim losses are sustained in future years (pending recovery to baseline abundance) resulting from the direct kill at the time of the spill. Interim losses potentially include:

- Lost future uses (ecological and human services) of the killed organisms themselves;
- Lost future (somatic) growth of the killed organisms (i.e., production foregone, which provides additional services);
- Lost future reproduction, which would otherwise recruit to the next generation.

The approach used here is that the total loss includes the direct kill and its future services, plus the lost somatic growth of the killed organisms, which would have provided additional services. Because the impact on each species, while locally significant, is relatively small compared to the scale of the total population in the area, it is assumed that density-dependent changes in survival rate are negligible, i.e., changes in natural and fishing mortality of surviving animals do not compensate for the killed animals during the natural life span of the animals killed.

It is also assumed that the impacts were not large enough to significantly affect future reproduction and recruitment in the long term. It is assumed that sufficient eggs will be produced to replace the lost animals in the next generation. The numbers of organisms affected, while potentially locally significant, are relatively small portions of the total reproductive stock. Given the reproductive strategy of the species involved to produce large numbers of eggs, of which only a few survive, it is assumed that density-dependent compensation for lost reproduction occurs naturally.

The services provided by the injured organisms are measured in terms of production, i.e., biomass (kg wet weight) directly lost or not produced. Among other factors, services of biological systems are related to the productivity of the resources, i.e., to the amount of food produced, the usage of other resources (as food and nutrients), the production and recycling of wastes, etc. Particularly in aquatic ecosystems, the rate of turnover (production) is a better measure of ecological services than standing biomass (Odum, 1971). Thus, the sum of the standing stock killed (which resulted from production previous to the spill) plus lost future production is a more appropriate scaler, as opposed to standing stock alone (as number or kg), for measuring lost ecological services.

This injury estimation approach was developed and used previously in the injury quantification for the *North Cape* spill of January 1996 (French McCay et al., 2003b, French McCay and Rowe, 2003) and many other spill cases (e.g., French McCay et al., 2003c). The method makes use of the population model in SIMAP. Injuries are calculated in three steps:

1. The direct kill is quantified by age class using a standard population model used by fisheries scientists.
2. The net (somatic) growth normally to be expected of the killed organisms is computed and summed over the remainder of their life spans (termed production foregone).
3. Future interim losses are calculated in “present day” (year of the spill) values using discounting at a 3% annual rate.

The normal (natural in local waters) survival rates per year and length-weight by age relationships are used to construct a life table of numbers and kg for each annual age class. Production forgone is then estimated using the model of Jensen et al. (1988), which is commonly used in fisheries science (see below).

It should be noted that compensation would be needed for lost production of each of the individual species injured, and that losses are additive. Restoration for a prey species killed will compensate for that prey killed and all the services that prey would have provided in the future to its predators and other resources. The predators that would eat that prey but were directly killed were produced before the spill from *different* prey individuals as food. Thus, the predator’s production loss must be compensated in addition to the prey animals directly killed. This may be accomplished by providing additional prey production to compensate for the direct predator loss.

3.5.2 Equations

The production foregone population model as described by the U.S. Environmental Protection Agency in its 316(b) rule (USEPA, 2004) is used. This approach is recommended by fisheries scientists and the models are those typically used for entrainment and impingement fisheries impact evaluations (EPRI, 2004). The equations are based on fisheries model development described in Ricker (1975).

The production foregone population model makes use of survival rates from one stage to the next. For eggs, survival to age one (S_{e1}) is calculated as:

$$S_{e1} = 2 S_e e^{-\ln(1+S_e)} S_L S_j \quad (8)$$

where S_e , S_L , and S_j are the survival rates for each stage: egg, larvae, and juvenile. For larvae, survival to age one (S_{L1}) is calculated as:

$$S_{L1} = 2 S_L e^{-\ln(1+S_L)} S_j \quad (9)$$

Natural and fishing mortality rates for annual age classes are used to estimate numbers that would remain alive by each age class. The number remaining alive at age t (years), N_t , is:

$$N_t = N_1 e^{(-Z_a)(t-1)} \quad (10)$$

$$Z_a = M_a + F_a \quad (11)$$

where N_1 is the number at age one, Z_a is annual instantaneous total mortality, M_a is annual instantaneous natural mortality, and F_a is annual instantaneous fishing mortality, for age class a . The annual survival rate for age t (S_t) is thus:

$$S_t = e^{(-Z_t)} \quad (12)$$

The fraction dying in a year is $1-S_t$.

Yield foregone (Y_k) (i.e., equivalent yield, or lost catch) may be calculated using the Thompson-Bell equilibrium yield model (Ricker, 1975) where the harvest at each age class is calculated from number starting the class multiplied by fishing mortality rate, $(F_a/Z_a)(1-e^{-Z_a})$:

$$Y_k = \sum_j \sum_a L_{jk} S_{ja} W_a (F_a/Z_a)(1-e^{-Z_a}) \quad (13)$$

where:

Y_k = foregone yield (kg) in year k

L_{jk} = losses of individual fish of stage j in the year k

S_{ja} = cumulative survival fraction from stage j to age a

W_a = average weight (kg) of fish at age a

F_a = instantaneous annual fishing mortality rate for fish of age a

Z_a = instantaneous annual total mortality rate for fish of age a

Total natural mortality (TM_k) is calculated using an analogous model:

$$TM_k = \sum_j \sum_a L_{jk} S_{ja} W_a (M_a/Z_a)(1-e^{-Z_a}) \quad (14)$$

where M_a is the instantaneous annual natural mortality rate for fish of age a .

For this analysis, the losses are for eggs and larvae translated to 1 year of age, i.e., one stage where $j=1$.

Production foregone (USEPA, 2004, Chapter A-5; based on Rago, 1984 and Jensen et al., 1988) which includes yield (harvest) and the production consumed in the food web, is estimated using:

$$Y_k = \sum_j \sum_a [G_a L_{jk} W_a (e^{G_a - Z_a} - 1)] / [G_a - Z_a] \quad (15)$$

where G_a is the instantaneous growth rate for individuals of age a .

Length and weight at age are estimated using the von Bertalanffy equation and a power curve of weight versus length following methods in Ricker (1975). The equations used are as follows. For length (mm) at age t (years):

$$L_t = L_\infty [1 - e^{(-K(t-t_0))}] \quad (16)$$

where L_t is length (mm) at age t (years), L_∞ is the asymptotic maximum length (mm), K is the Brody growth coefficient, and t_0 is a constant. Weight as a function of length (mm) is:

$$W_t = \alpha L_t^\beta \quad (17)$$

where W_t is wet weight (g) at age t years and α and β are constants.

Discounting at 3% per year (NOAA, 1997) is included to translate losses in future years (interim loss) to present-day values. The discounting multiplier for translating value n years after the spill to present value (i.e., for the year of the spill) is calculated as $(1+d)^{-n} = 1/(1+d)^n$, where $d=0.03$. Thus, the losses in future years have a discounted value at the time of the spill. In this analysis, all discounting will be calculated based on the number of years from the year of the spill. Thus, additional discounting is needed to translate all the injuries to compensatory equivalents for the year the restoration is performed. The multiplier for this calculation is $(1+d)^m$, where m is the number of years after the spill when restoration is accomplished.

4 Discussion

Validation

The model has been validated using simulations of over 20 spill events where data are available for comparison (French and Rines, 1997; French McCay, 2003, 2004; French and Rowe, 2004). In most cases (French and Rines, 1997; French McCay, 2004; French and Rowe, 2004), only the wildlife impacts could be verified because of limitations of the available observational data. However, in the *North Cape* spill simulations, both wildlife and water column impacts (lobsters) could be verified. Field and model estimates of lobster impacts were within 10% of each other (French McCay, 2003).

Hindcasts and NRDA

The model has proved useful in numerous hindcast studies as part of NRDA's. The most accurate approach for using a model in an NRDA is to obtain sufficient field data to provide input to the model and calibrate the results.

In the event of an oil spill, in order to fully characterize the impact by field sampling, water and sediment samples would be needed at frequent time intervals over the first few weeks after the release (and especially in the first 24-48 hours), and with enough spatial coverage to characterize the extent of contamination. In addition, comprehensive sampling of each of the species affected is needed in the exposed and unaffected areas. Because marine organisms are so patchy in their distribution, large numbers of stations and samples within stations are needed to accurately map abundance. Such extensive sampling of all (or even selected) species affected is often not feasible, given the rapidity at which the evidence disappears (by scavenging of killed organisms and by migration of animals into the impacted area). Thus, in practice, the needed sampling would require a considerable effort, which is usually both infeasible and too costly to be justifiable by the expected impact of the spill. In spite of these obstacles, historically, attempts at quantification have primarily been made by collecting field data. A more practical and realistic approach is to combine field sampling with modeling.

Biological sampling should be designed to establish pre-spill baseline (by number and weight for each species and life stage and by size classes, as appropriate) and what types of organisms were exposed. Biological effects modeling may then be used to quantify impacts. If it is feasible, field data collections could be focused on exposed species of particular concern, such that enough data might be collected to

indicate and possibly quantify the impacts. If this is possible, such data may be used to verify the modeling results.

Modeling may be used to estimate the range of potential injuries, given the range in species sensitivity and acute toxicity values that have been observed in laboratory-based bioassays performed and reported previously (French McCay, 2002). Given the large variation in sensitivity of various species and life stages (Figure 4), and that many important species have not been tested, acute toxicity bioassays should be performed on exposed organisms of concern to provide more accurate estimates of impact. To simplify this and avoid artifacts of whole-oil bioassays, the toxicity tests can be performed for single hydrocarbon exposures and the toxicity of the dissolved hydrocarbon mixture resulting from oil exposure can be calculated from the bioassay results using the modeling approach described in French McCay (2002).

Risk Assessment Consequence Analyses

In environmental risk assessment studies (e.g., French McCay et al., 2003a, 2004, 2005a,b,c) the objective is to assess potential consequences if a spill were to occur. Hence, multiple model runs and conditions need to be evaluated to develop an expectation of risk of oil impacting each resource of concern. To evaluate the distribution of potential impacts resulting from variation in environmental conditions, the model is run many times, randomizing the start date and time. In addition or alternatively, various other model inputs may be varied within specified ranges or according to probability distributions, such as the spill volume, location, release depth, release duration, density of biota of concern, toxicity values, and assumed parameters for model algorithms. The multiple model runs provide a frequency distribution of model results, for which statistics (e.g., mean, standard deviation, maximum) are calculated and plotted, such as for probabilities of exceeding effects endpoints, areas impacted above these thresholds, or numbers of animals killed. Response strategies (i.e., removal from clean up, booming) may be incorporated into the model and considered in the assessment.

The potential spill conditions causing the median or the worst-case impacts to specific resources may be identified using the probabilistic model results. For example, a worst case for sensitive resources along the coast may be those wind and current conditions which would maximize exposure to those resources. The individual worst case scenario may then be examined in more detail, forecasting it using the 3-dimensional fates and biological effects models, quantifying the worst possible exposure for that resource of concern. Sensitivity analysis provides measures of uncertainty for these predictions. Other worst case scenarios may be identified for additional resources of concern, such as seabirds, marine mammal, sea turtles, fish, etc; and examined in detail with fates and biological effects model analyses. Alternatively, the range of potential impacts may be identified.

Research Needs

The above-described model represents the state-of-the-art for oil spill biological effects modeling. Research needs for informing model parameterization and developing new algorithms are outlined below.

- **Wildlife oiling probabilities:** For wildlife the probabilities of exposure to oil, given presence in the area swept by oil, would be less uncertain with additional quantitative observational data from spills. For example, counts by species in an area oiled could be followed up by counts of oiled birds recovered (with necessary corrections for losses and search effort).
- **Avoidance or attraction behavior:** Wildlife may avoid oil or learn to avoid oil. Alternatively, wildlife may be attracted to biota impacted by oil and become oiled. While many anecdotal observations have been reported, quantitative information to include such behavior in a model is lacking. Alternatively, the issue can be addressed by altering densities of animals exposed.
- **Fish and invertebrate behavior:** Behavioral detail for fish and invertebrates, both in general and in the presence of oil (e.g., avoidance, attraction), could potentially improve accuracy of the modeled movements. Data needed are the details of vertical distribution on a diel basis and overall migration speed of these organisms (as opposed to localized or temporary swimming speed).
- **Effects of suspended oil droplets:** To date, modeling has only quantified acute effects of dissolved hydrocarbon components. While anecdotal information exists to indicate that suspended oil droplets impact aquatic biota, data quantifying the dose-response relationship are lacking. In SIMAP, the fates and concentrations of suspended oil droplets are simulated, such that concentration estimates are available for such evaluations.
- **Impacts on neuston:** The sea surface environment provides an important habitat for many organisms commonly referred to as neuston. Fish eggs, fish larvae and crustacean larvae, including several commercially important species, have been found to encompass significant portions of neustonic communities (Grant, 1986). Besides the permanent invertebrate inhabitants of the surface layer, commonly referred to as euneuston, the larvae of several macroinvertebrates including various families of corals, crabs and spiny lobster have been observed to be concentrated in the surface layer. Larval fish of some species appear to actively seek the surface layer by adjusting their swim bladder to become buoyant (e.g., sardines, Santos et al., 2006). Certain species of fish are noted as having larval stages which are completely neustonic or surface dwelling (i.e. gurnards, (Dactylopteridae)) (Cowen, 2002). Floating fish eggs are particularly vulnerable to the impacts of oil (Longwell, 1977; Longwell and Hughes, 1980). These neustonic assemblages are subject to surface contamination, such as floating oil and oil entrained by breaking waves in the surface wave-mixed layer. Ignoring the presence of neuston has led to underestimation of oil spill impacts in the past (Grant, 1986). Observational and experimental information is needed to support model algorithm development and parameterization.
- **Impacts on surfacing fish:** In addition to larvae and zooplankton, there are several species of adult fish which utilize the sea surface habitat, e.g., flying fish, halfbeaks and needlefish spend a significant amount of time swimming and breaking the sea surface. Many fish species come to the surface to fill their swim bladders. Oil spills which sweep pelagic areas where these and

other surface-dwelling species are found likely impact these species. Injury due to physical interaction between adult fish at the sea surface and floating oil/tar has not been assessed to date for lack of information with which to address the issue.

- **Acute toxicity for short-term (several hour) exposures:** Research and the literature have well-documented that the uptake of semi-soluble organics such as hydrocarbons in oil continues for days to weeks until the tissue concentrations reach effects levels (see reviews in DiToro et al., 200; French McCay, 2002). Indeed, standard acute toxicity tests are performed for 96 hours or longer on this basis (Sprague, 1969; Swartz et al., 1995). The relationship of effects level (LC50) versus duration of exposure is based on uptake modeling and what data is available for short-duration (i.e., hours) exposures. Additional bioassay data for exposures of a few hours are needed to calibrate and verify these relationships.
- **Phototoxicity:** It is well understood that UV light induces phototoxic effects for some PAHs in oil (Barron et. al., 2003, 2004; Lee, 2003; Kirby et al., 2007). UV light intensity decreases with increasing latitude and depth into the water column. However, organisms in shallow water and in the neuston could be significantly affected by phototoxicity of PAHs accumulated in their tissues if they lack pigmentation for protection from these effects (Lee, 2003; Barron et al., 2005). The magnitude of this effect should be evaluated and phototoxicity included in modeling assessments if significant.
- **Chronic effects of oil:** Long-term effects of PAHs from oil are well documented in the literature. Such effects have not been formally included in biological effects models to date. However, the physical fates modeling to support such inclusion would need to be very comprehensive and accurate for such inclusion to be reasonably accurate. Alternatively, and more accurately, long-term biological effects modeling could be driven by observational data of sediment and shoreline concentrations of PAHs. This latter approach is recommended, given the duration of exposure and feasibility of sampling sediments. Concentrations in the water column are of short duration and, thus, do not result in chronic exposures in that habitat.
- **Population and ecosystem level impacts** result from acute and chronic effects on individuals. Modeling of such effects can rely on the extensive literature related to effects of disturbance and recovery. Such modeling has not been attempted for impacts of oil spills to date, due to the magnitude of the task and uncertainties involved.

Finally, it should be noted that the level of detail required in a model varies by the circumstances and needs of the assessment. For example, if water column contamination from a spill results in minimal or negligible toxicity, the accuracies of input density data, algorithms governing water column organism behavior, and population level modeling for fish and invertebrates are inconsequential to results. Also, if an ecological risk assessment is being performed, such that the analysis involves comparisons among alternatives, use of organism density data may not be required; rather comparisons of areas or water volumes impacted can be used. Sensitivity analysis, varying inputs within the range of uncertainty, is used to quantify

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and convey the uncertainties of model results. This elucidates important assumptions and data inputs for a particular application of the model. Calibration may be used to improve accuracy for specific hindcasts.

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