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ABSTRACT

In order to explore the potential impacts of petroleum-associated chemicals, including polycyclic aromatic hydrocarbons (PAHs), on cetacean populations, we modified the IWC Pollution 2020 individual based model (SPoC) to incorporate additional effects of petroleum-associated chemical exposure following an oil spill on a simulated population of bottlenose dolphins (*Tursiops truncatus*). The existing model framework allows for the exploration of impacts due to maternal polychlorinated biphenyl (PCB) exposure on calf survival and immunity. This is achieved using concentration-response functions, whereby survival probabilities are modified depending on exposure. Potential population growth rates are estimated using a stochastic model that incorporates a degree of parameter uncertainty in the simulations. However, using concentration-response or dose-response functions was not possible for petroleum-associated chemicals, as estimating exposure for these compounds in cetaceans is fraught with difficulties due to the complexities of chemical analyses, the mixture of compounds, the different routes of exposure and variations in metabolism. Instead a broader approach was taken. Reductions in survival probability and fecundity in a population of bottlenose dolphins were estimated following the *Deepwater Horizon* (DWH) oil spill in the Gulf of Mexico (*Deepwater Horizon* Natural Resource Damage Assessment Trustees 2016). The magnitude and duration of these vital rate changes were then incorporated into the Pollution 2020 individual based model to investigate the effect on potential population growth rates (λ) over a 100 year period. This simulated the impact of an oil spill at least the size of the DWH spill occurring in year 40. The advantage of this approach is that it allows for the cumulative effects of PCB and petroleum-associated chemical exposure to be considered. Three example scenarios were run, demonstrating that chemical exposure as a result of large oil spills can have a major effect on small cetacean abundance, particularly when combined with the effect of other pollutants and stressors.

INTRODUCTION

Petroleum-associated chemicals include polycyclic aromatic hydrocarbons (PAHs), which are hydrocarbon compounds that include, for example, naphthalene, phenanthrene, chrysene and benzo(a)pyrene. The PAHs compounds are among the most toxic components of oil, both crude oil and refined petroleum products (Ball & Truskewycz 2013) and many are classed as either carcinogenic or probably carcinogenic to humans by the WHO International Agency for Research on Cancer (IARC, 2010). Whilst petroleum-associated chemical toxicity studies on cetaceans are rare (Englehardt 1983), a few laboratory studies have shown that both hepatotoxic and dermal toxicity effects can occur following exposure (Geraci et al. 1988). Additionally, studies in pinnipeds reported effects on pulmonary and renal function, as well as a reduction in survival (Englehardt 1982, Englehardt 1983). Surrogate laboratory animal models, such as mink, suggest that petroleum-associated compounds are also likely to affect adrenal and haematological function and exposure will reduce fecundity and survival (Mazet et al. 2000, Mazet et al. 2001).

A number of large scale oil spills have highlighted the health risks to cetaceans following exposure (Carson & Walsh 2008, Neuparth et al. 2012), including the *Exxon Valdez* spill in Alaska. Mortality among resident and transient killer whales (*Orca orcinus*) that were present in Prince William Sound during the spill was estimated to have increased by 30-40% over an 18 month follow up period (Matkin et al. 2008). In addition, other marine mammal species suffered increased mortality rates including sea otters (*Enhydra lutris*), and harbour seals (*Phoca vitulina*), (Garrott et al. 1993, Frost et al. 1999). But by far the most comprehensive studies on the effects of petroleum-associated chemical exposure (and therefore PAH exposure) to cetaceans, particularly bottlenose dolphins (*Tursiops truncatus*), were carried out following the *Deepwater Horizon* spill in the Gulf of Mexico (Schwacke et al. 2014, *Deepwater Horizon* Natural Resource Damage Assessment Trustees 2016).

Thus, whilst the NOAA *Deepwater Horizon* Oil Spill Final Programmatic Damage Assessment and Restoration Plan (PDARP) determined that many cetacean species occurring in the Gulf were affected, the detailed survival and health effect studies on the bottlenose dolphins occurring in the bays, sounds and estuaries along the coast where oiling occurred, provide the most useful sources for investigating the potential population impacts (Schwacke et al. 2014, Lane et al. 2015, Venn-Watson et al. 2015). In particular, the results of these studies allowed us to modify the IWC Pollution 2020 SPoC (Effect**S** of **P**ollutants **O**n **C**etacean populations, (Hall et al. 2011, Hall et al. 2013) model to investigate the impacts of petroleum-associated chemicalexposure at a level at least as severe as that which occurred following the *Deepwater Horizon* spill.

Live capture health assessments of bottlenose dolphins from Barataria Bay, Louisiana following the spill concluded that petroleum-associated chemical exposure had caused a significant increase in lung and adrenal disease (hypoadrenocorticism) (Schwacke et al. 2014, Lane et al. 2015, Venn-Watson et al. 2015). Due to the short half-life of petroleum-associated chemicalsin the system and the difficulties associated with measuring PAHs in marine mammal tissues, it was not possible to construct a concentration-response function. However, annual mark-recapture photo-id studies of the population estimated that survivorship amongst the exposed animals declined by approximately 10% per year for the three years following the spill (Deepwater Horizon Natural Resource Damage Assessment Trustees 2016).

During the health assessments in Barataria Bay, reproductive success was also found to be very low (Lane et al. 2015). Of the females studied (n=20), between 2010 and 2014, 10 were pregnant but only two (20%) gave birth to a viable calf. In comparison, the success rate for the Sarasota Florida population was 83% (Wells 2014). The injury quantification of the PDARP (Deepwater Horizon Natural Resource Damage Assessment Trustees 2016) therefore estimated the excess failed pregnancies to be 46%. This enabled us to investigate the additional effect of reduced fecundity on the potential population growth rate and the rate of recovery.

METHODS

SPoC model for Bottlenose dolphins

Details of the SPoC model framework are given in (Hall et al. 2006, Hall et al. 2011). The model has been constructed using the statistical and modelling package R (R Development Core Team, 2015) and it simulates the fate of individual females using published fecundity and survival data to construct an initial, appropriately sized, population of animals with a stable age structure. The model then simulates the accumulation of polychlorinated biphenyls (PCBs) through transplacental transfer, suckling and prey ingestion and loss of PCBs from the mature females' blubber. Maternal blubber PCB concentrations then affect the first-year calf survival probability in a dose-dependent matter. In addition the effects of PCBs on immunity have been included (Hall et al. 2012) where the survival of all age classes is modified in relation to the concentration of PCBs in their blubber and how this can affect survival probability following immunosuppression and exposure to a pathogen. Concentration response functions (relating female blubber PCB concentrations to survival of their calves or overall survival relating to effects of PCBs on immune function) use published data from laboratory animal models (Luster et al. 1993, Fuchsman et al. 2008) and include the uncertainty in these relationships.

The model is stochastic so that each of the birth and survival outcomes are determined by whether a random number (generated from a uniform distribution) was less than or equal to the probability associated with that event.

Model Structure

Each animal is assigned a state variable of 1, alive or 0, dead, an age and blubber PCB concentration (mg/kg lipid weight). The model is a post-breeding census and age class 1 is equivalent to newborn calves. Each model simulation spans a period of 100 years and a starting abundance is chosen based on the specific populations being simulated. For any given set of fecundity or survivorship values the stable age structure is calculated by multiplying an arbitrary seed age structure by the appropriate Leslie matrix 100 times. The stable age structure is then used as the underlying population structure of the initial population of *n* females. At first each animal is assigned zero PCB level and after the first year, animals are then allocated an appropriate blubber PCB concentration depending on their age class and reproductive status. After approximately the 40th simulation year, the relationship between PCB concentrations and age stabilises. From the population trajectories after the first 40 years the mean potential growth rate of the population is calculated and the 2.5 and 97.5 percentile growth rates estimated from the ranked individual growth rates. The variation in potential population growth rate with varying annual PCB accumulation rates can then be investigated, incorporating uncertainty into the concentration-response relationship. This is achieved by each 100-year simulation the model choosing random concentration-response model coefficients from a set of coefficients generated by data resampling. Effects of PCBs on immunity are similarly modelled, using relationships between blubber PCBs, immune function assays and decreased host resistance (again using resampling to incorporate uncertainty). The level of reduced survival at the population level is then determined by randomly assigning a chosen proportion of the population to be exposed to a novel virulent pathogen.

Model parameters

The vital rates (fecundity and survival) and other explicit model parameters such as initial population size and maximum age class used in the model simulations are given Table 1. Where possible, species and population specific parameters were used. Those used in the following simulations are taken for bottlenose dolphins based on the Sarasota Bay, Florida population and are the same as the parameters used in the initial simulations reported in Hall et al. (2006). In this study data from various sources is used to estimate the proportion of PCBs transferred form the female to the calf *in utero* (0.6) and during lactation (0.77) (Tanabe et al. 1982, Cockcroft et al. 1989, Salata et al. 1995). Where the calf (of either sex) dies within its first year we assume death occurs at 6 months and the depuration for that year is halved to 0.38. Subsequently the birth of male calves is ignored by the model.

Table 1. Model parameters used to simulate effect of maternal PCB concentrations on first calf survival and population growth rate for bottlenose dolphin and humpback whale.

Parameter	Bottlenose Dolphin	Reference
No. females	300	
Maximum age	40 years	(Wells and Scott 1990)
1st year calf survival	0.811	(Wells and Scott 1990)
Adult survival	0.962	(Wells and Scott 1990)
Fecundity	0.177	(Wells and Scott 1990)
Calf sex ratio	1:1	(Wells and Scott 1990)
Length of lactation	2 years	Oftedal 1997

Modifications to investigate the impact of exposure to PAHs

The model was then modified to include the impact of an oil spill and therefore exposure of the population to petroleum-associated compounds, on potential population growth rate using a simulated population of bottlenose dolphins as a case study. The vital rates from Table 1 thus gave a baseline population growth (λ) of 1.014 (i.e. 1.4% annual growth). We have termed the evaluated population outcome 'potential' population growth as the effects of density dependence are not accounted for.

In agreement with the findings of the PDARP (Deepwater Horizon Natural Resource Damage Assessment Trustees 2016), we used the following scenarios to investigate the population consequences of an oil spill at least as large as the DWH spill.

1. A 10% reduction in survival the year of the spill and the two following years with a linear increase in survival back to baseline over the next five years, including the impact of PCBs on first calf survival at a PCB exposure annual increment level of 1 mg/kg with no effects on immunity.
2. A 10% reduction in survival the year of the spill and the two following years with a linear increase in survival back to baseline over the next five years, including the impact of PCBs on first calf survival at a PCB exposure annual increment level of 1 mg/kg with effects on immunity and 5% of the population exposed a novel pathogen.

3. A 10% reduction in survival the year of the spill and the two following years with a linear increase in survival back to baseline over the next five years, including the impact of PCBs on calf survival at a PCB exposure annual increment level of 1 mg/kg with effects on immunity and 5% of the population exposed a novel pathogen, plus the additional effect of petroleum-associated compounds on fecundity, decreasing by 46% for the five years following the spill.

RESULTS

The potential population growth for a population of bottlenose dolphins with an annual accumulation 1 mg/kg lipid weight blubber total PCBs and only accounting for the effect of PCBs on calf survival for comparison with the first scenario is shown in Fig. 1.

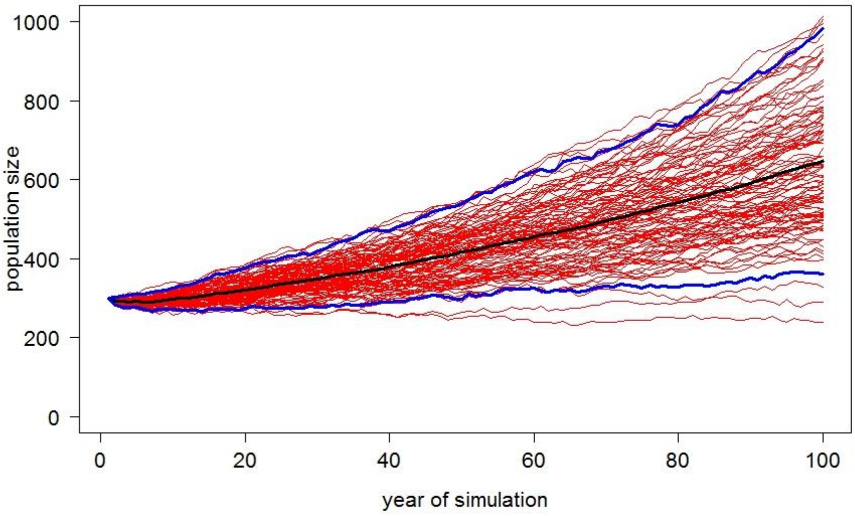

Figure 1. Potential population growth for a 100 simulated populations of bottlenose dolphins, over 100 years, with an annual accumulation of 1 mg/kg blubber total PCBs accounting for the effects of maternal PCBs on calf survival.

The estimated potential annual growth rate (λ) was 1.0093 (95% CI 1.0011, 1.0157), indicating a moderate impact of PCBs on the population (unexposed baseline population (λ = 1.014).

Scenario 1. Effect of PCBs on calf survival and impact of oil spill

In this scenario, an oil spill occurs in year 40 of the simulation runs which impacts the population for a period of 8 years, three years where all-age survival is reduced by 10% and five years where the survival linearly recovers to pre-spill levels. The results of 100 simulations for 100 years are shown in Fig. 2. The potential population growth over the pre-spill years (0-39) was 1.0037 (95% CI 0.9976, 1.0095) and on average, over the years affected by the spill (40-47) the population was declining with an annual growth rate of only 0.9541 (95% CI 0.9398, 0.9661), a decline of approximately 5% per annum. Thus while in the comparison population the abundance of dolphins more than doubles in size (Fig. 1), it declines, it only just reaches the pre-spill abundance by year 100, 60 years after the spill (Fig 2).

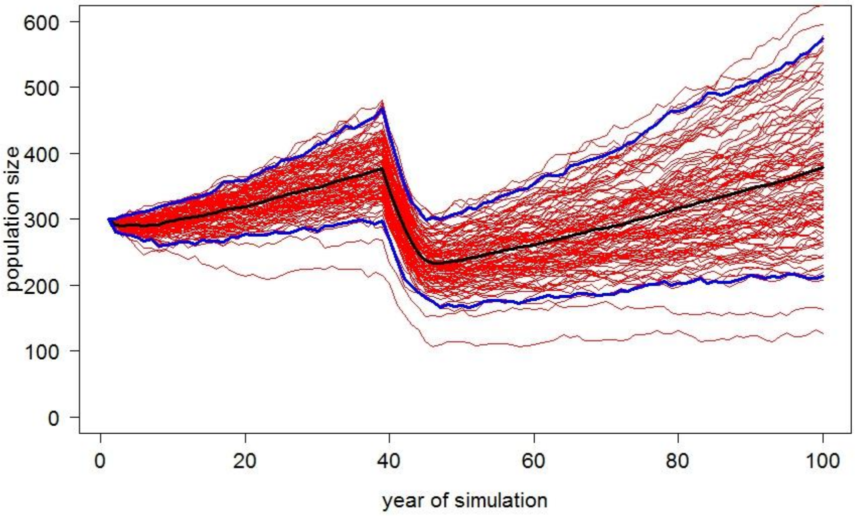

Figure 2. Potential population growth for a 100 simulated populations of bottlenose dolphins, over 100 years, with an annual accumulation of 1 mg/kg blubber total PCBs accounting for the effects of maternal PCBs on calf survival and including an oil spill (scenario 1) occurring at year 40.

Scenario 2. Effect of PCBs on calf survival, immunity and impact of oil spill

Again for comparative purposes, the model is first run with effects of PCBs on calf survival and immunity (with 5% of the population exposed to a low virulence pathogen) without an oil spill. The results of these simulations are shown in Fig. 3 with an estimated potential population growth rate of 1.0067 (95% CI 0.996, 1.0142).

This is a scenario similar to the first set of model runs, except in this second set we include estimating the additional effect of PCB uptake on immunity. The results of a further 100 simulated populations is shown in Fig. 4, where the oil spill again occurs in year 40 of the simulation runs. The potential population growth over the pre-spill years (0-39) was 1.0012 (95% CI 0.995, 1.0073) and on average, over the years affected by the spill (40-47) the population was declining with an annual growth rate of only 0.9532 (95% CI 0.9383, 0.9698), again a decline of approximately 5% per annum. After the five years of reduced survival due to PAH exposure, the population starts to slowly recover with a potential growth rate of 1.0067 (95% CI 0.9995, 1.0131) but at this rate, abundance does not return to pre-spill levels until around year 100 (mean abundance from 100 simulations n=291). The difference between the population trajectories before and after the spill are likely due to the fact that the first 40 years are required for the age related PCB concentrations to stabilise in the population.

Scenario 3. Effect of PCBs on calf survival, immunity and impact of oil spill on survival and fecundity

In this last scenario we investigate the additional potential impact of oil on fecundity combined with the impacts of PCBs on calf survival and immunity and the effect of oil on survival. Again, 100 simulations were run with an oil spill at year 40. Again the potential population growth over the pre-spill years (0-39) was 1.0004 (95% CI 0.9929, 1.006) and as expected, over the years affected by the spill (40-47) the population was declining at a higher rate than in scenario 2, with an annual growth rate over the 5 year period of 0.9352 (95% CI 0.9185, 0.9529), a reduction of 7% growth per year. After the years of reduced survival and fecundity due to PAH exposure, the potential growth rate returned to 1.0076 (95% CI 0.9974, 1.0146) but this rate is not sufficient to restore the population to pre-spill levels by year 100 (mean abundance from 100 simulations, n = 257, Fig. 5).

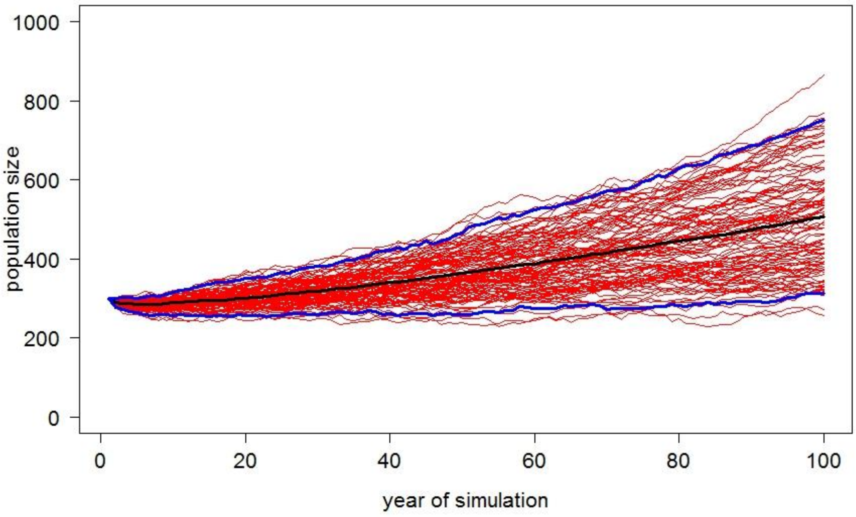

Figure 3. Potential population growth for a 100 simulated populations of bottlenose dolphins, over 100 years, with an annual accumulation of 1 mg/kg blubber total PCBs accounting for the effects of maternal PCBs on calf survival and immunity with 5% of the population exposed to a low virulence pathogen.

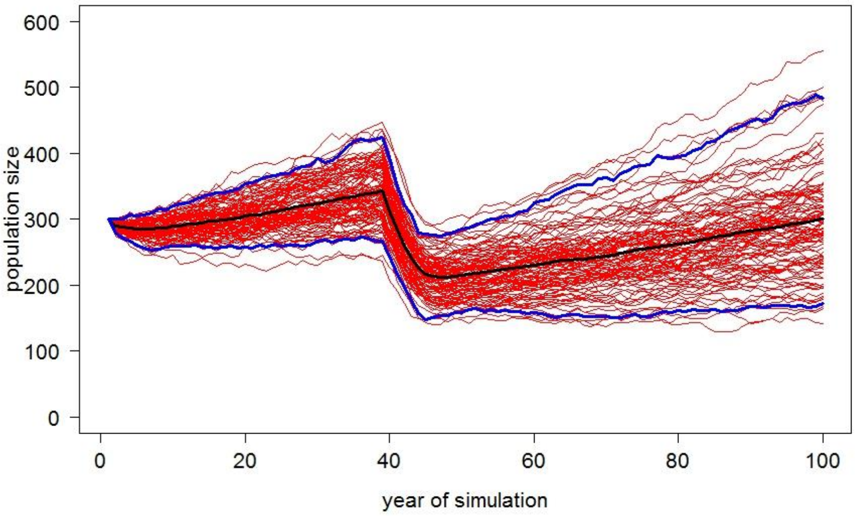

Figure 4. Potential population growth for a 100 simulated populations of bottlenose dolphins, over 100 years, with an annual accumulation of 1 mg/kg blubber total PCBs accounting for the effects of maternal PCBs on calf survival and immunity with 5% of the population exposed to a low virulence pathogen and including an oil spill (scenario 2) occurring at year 40.

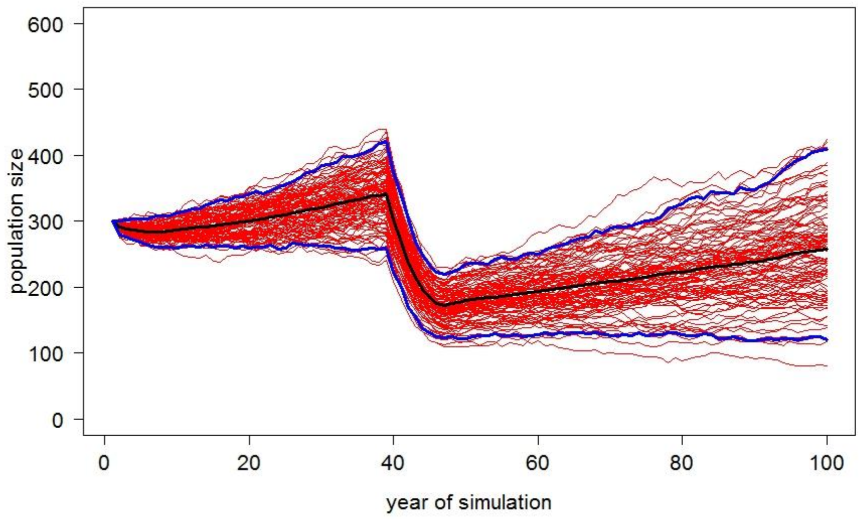

Figure 5. Potential population growth for a 100 simulated populations of bottlenose dolphins, over 100 years, with an annual accumulation of 1 mg/kg blubber total PCBs accounting for the effects of maternal PCBs on calf survival and immunity with 5% of the population exposed to a low virulence pathogen and including an oil spill occurring at year 40. Additional effects of petroleum-associated compounds on fecundity are also included (scenario 3).

CONCLUSIONS

This approach indicates how the relative impact of different contaminants or catastrophic events can be estimated using the SPoC model framework. It also allows for additional (cumulative) effects to be assessed by the addition of various stressors whose effect on survival or fecundity is known or at least can be estimated. Here we show that the oil spill scenarios which mimic the estimated population consequences of a spill at least as large as the DWH spill, can, as expected, have long term implications for potential population growth and abundance. This is clearly particularly important when petroleum-associated chemical exposure occurs together with already existing POP contaminant uptake that may already be having effects on survival, fecundity and immunity. Under the three scenarios tested, it takes 3-4 decades for the populations to recover to pre-spill abundances. These recovery time estimates are in line with those estimated in the DWH PDARP (39 years, 95% CI 24-80 for the Barataria Bay population) using a slightly different population modelling approach (Deepwater Horizon Natural Resource Damage Assessment Trustees 2016).

There are some caveats that should be borne in mind when exploring these results. Firstly not all potential adverse effects of PCB uptake are accounted for (such as effects on fecundity). In these simulations the annual accumulation of PCBs was set at a low level (in line with the PCB concentrations reported for the Barataria Bay dolphins (Schwacke et al. 2014) and to be able to determine the additional effects of petroleum-associated compounds without driving this small population to extinction within the simulation period. The effects of higher PCB uptake rates could be explored in future with larger initial population sizes, noting that this is computationally more time consuming. In addition, recent results from a study of PCB concentrations in female harbour porpoise and their foetuses (Hall et al. 2016) suggests that the proportional transplacental transfer of contaminants is higher than previously estimated. This new study will allow us to incorporate uncertainty into the *in utero* transfer model parameter in future.

Pregnancy success rates were significantly lower in Barataria Bay dolphins compared to those in the reference population of Sarasota, Florida with a 46% reduction. This was used to explore additional effects of petroleum-associated chemical exposure on fecundity over a limited time period, with the assumption that adversely affected females will eventually be lost from the population. This caused the population to decline more rapidly during the spill-affected years, further delaying the recovery of the population but it is unclear as to how long these effects on fecundity are likely to persist in the population.

This study demonstrates that exposure to petroleum-associated chemicals can be incorporated into the SPoC model provided some information about the magnitude of any future spills in comparison to the DWH event can be estimated. But perhaps more useful in a conservation and management context is the ability to compare the effect of different scenarios that incorporate the impact of current pollutant exposures with potential future events. This will allow conservation managers to estimate and predict the likely impact of major events. For example, future simulations could also include the additional impact of a morbillivirus epidemic, similar to the recent outbreak among bottlenose dolphins along the east coast of the US (Morris et al. 2015).

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