IMPACTS OF OIL SPILLS ON ARCTIC MARINE SPECIES: A RISK ASSESSMENT PERSPECTIVE

by

© Faisal Fahd B.Tech., M.Eng.

A Thesis submitted to the

School of Graduate Studies

in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

Faculty of Engineering and Applied Science

Memorial University of Newfoundland

August 2021

St. John's Newfoundland

Abstract

The Arctic region is characterized by an ubiquitous presence of sea ice, harsh weather conditions and inhabitation of some unique marine animals. The changing climatic conditions have resulted in receding of the sea ice in the regions along the Arctic boundaries. The receding sea ice has opened the possibilities of tapping into hitherto unexplored reserves of natural resources in the Arctic. Shipping operations along the Northern Sea Route have received an impetus because of the changed sea ice conditions in the region. However, the growing anthropogenic activities also increase the risk of environmental pollution in the region. The Arctic is a home to many unique marine species, such as the polar bear, beluga whales, seals and polar cod. The Arctic marine food chain is non-complex, with limited prey options forming a large portion of the diet of the marine species. Thus, adverse impact to the populations of a species may also impact the well-being of its predators in the Arctic food chain. The knowledge gap in the exposure and toxicological modeling of Arctic marine species were identified to be the presence of limited availability of the toxicity data and dose response relationship.

The research questions addressed in this study are as follows:

1. Is the toxicity and impacts in Arctic fish different from their temperate counterparts?
2. What is the risk of mortality to lower tropic sentinel species in the event of an oil spill?
3. What is the risk to apex marine species in the event of an oil spill?

The components of Environmental Risk Assessment (ERA) are hazard identification, exposure modeling, toxicological modeling and risk characterization. The thesis followed the steps laid down for ERA and identified the polar cod as the sentinel species for the Arctic food chain. The study also identified apex marine predators, polar bear and beluga whales, as species of interest along with polar cod. Polycyclic Aromatic Hydrocarbons are major constituent of the crude oil that can cause deleterious effects in the marine species. The spill scenarios considered for risk assessment to polar cod, polar bear and whale are as follows: Spill over thick sea ice; Spill over thin sea ice; Spill under thick ice.

A review of current exposure and toxicological models used for marine species was conducted and a novel toxicological model where the effects of the toxicant exposure were quantified based on the probability of cellular damage and metabolites interactions was proposed. This toxicodynamic approach in conjunction with physiology based toxicokinetic approach was proposed as the best suited approach for modeling and estimating toxicity in the Arctic marine species.

This research studied physiological causal dependencies leading to toxicity and mortality in polar cod from PAH exposure. Toxicity is also affected by environmental factors, such as sea ice and feeding behaviors. Presence of sea ice, could mitigate or aggravate the exposure to crude oil, thereby affecting the toxicity of the fish. The polar cod could biotransform some of the ingested PAH using a two-step process, namely, phase I and phase II processes. In phase one, the cytochrome P 4501A (CYP1A) enzymes react with the lipophilic xenobiotic, such as PAH, and convert it to water soluble metabolites for elimination. Phase II reactions further enhance the water solubility of the metabolites produced during the phase I step. The phase II conjugation reactions with glutathione are facilitated by glutathione-S-transferase (GST). The biotransformation toxicity

is a result of cell death when the toxic metabolites resulting from the phase I process exceeds the conjugating capacity of the organism via the phase II process. The other pathways of toxicity in polar cod are lipid peroxidation and cell damage. Although the pathways of PAH toxicity and the impact of sea ice on PAH ingestion for the fish had been studied previously, an effort to combine these factors as causal dependencies to estimate mortality in polar cod was never made. A novel Bayesian Network (BN) based model was developed as a part of this research, combining the physiological and environmental factors affecting the PAH exposure and toxicity in polar cod.

To estimate the risk to apex marine predators, an Arctic food chain was considered with the sentinel species, polar cod, at its bottom and apex species at its top. The risk to the apex species can be from exposure and susceptibility due to oil spill and additional risk from reduced food availability owing to decreased prey populations from the spill. Another BN based model was developed based on the food chain and spill conditions. The average daily food consumption and baseline population density of seals, polar bears and whales was collated from literature. The changes in populations of the polar cod, seals polar bear and whales due to oil spill were input in the BN model and probabilities of apex species survival are estimated.

The BN based risk models developed in this study were demonstrated for a hypothetical spill scenario in a geographic region around the Svalbard Island and Fram Strait. The oil spill scenarios considered in this research are spill over and under thick sea ice and spill on thin sea ice. Three spill sizes considered in this study were 15000 tonnes, 18000 tonnes and 40000 tonnes for low, medium and high states. The PAH weight percentage of 3.9% is assumed in the crude oil, along with a uniform dissolution in the water column.

The results of the BN model developed for polar cod and sensitivity analysis of the results suggested that physiological factors followed by sea ice played important role in risk mitigation.

The presence of thick sea ice in winter decreased the risk of mortality in polar cod by 16%. The ability of polar cod target organs to eliminate the xenobiotics, evident by various biomarkers activity, decreased the risk of mortality by 25% for worst case scenario. The spill scenario causing highest risk for polar cod population (29% mortality in population) was spill over thin ice in Autumn. The results from the apex species BN risk model predicted a polar cod recruitment collapse for the spill scenarios considered in this study, causing a higher risk of mortality of polar bears, beluga whales, and Narwhals in the Arctic region. Whales (adult and calves) were predicted to be at higher risk when the spill was under thick ice, while adult polar bears were at higher risk when the spill occurred on thin ice. A spill over the thick ice caused the least risk to whale and adult polar bears. The spill's timing and location had a significant impact on the marine animals in the Arctic region due to its unique sea ice dynamics, simple food web, and short periods of food abundance. In summary, this study identifies key marine species in the region and conducts an ecological risk assessment for the species based on the Arctic food chain. Four peer reviewed journal papers were published in the Marine Pollution Bulletin journal as the outcome of this research/thesis.

Acknowledgement

I would like to express my sincerest gratitude to my supervisors, Drs. Faisal Khan, Brian Veitch and Ming Yang for their guidance, support, and motivation throughout my doctoral program.

I am thankful to Dr. Faisal Khan and Dr. Brian Veitch, for accepted me in the LRF research group and provided me an opportunity to work in this research project. It is their dynamic supervision and thought-provoking feedback that helped me to reach to my milestones in this programme. I would also extend my sincere thanks to Dr. Ming Yang and Dr. Mawuli Afenyo, Dr. Douglas Smith for the helpful discussions in the LRF graduate office. It was a great pleasure to work with all the LRF team. I also extend my thanks to the staff of the Faculty of Engineering and Applied Science and School of Graduate Studies, Memorial University of Newfoundland for facilitating administrative work necessary throughout the programme.

The financial support of the Lloyd's Register Foundation is acknowledged with gratitude. Lloyd's Register Foundation helps to protect life and property by supporting engineering-related education, public engagement, and the application of research.

My stay in St. Johns forged great lifelong friendships. I extend my thanks to all my friends in St. John's, especially Ibrahim, Shariq, Shams and Zayed for their friendships, and making my stay enjoyable. My two daughters (Maryam and Arafah) had also forged great friendships here. I would also like to extend my thanks to Dr. Faisal Khan for weaving and holding on the social fabric in the community which many international students have drawn great support from, including me and my wife (who was also his student). I am profoundly grateful to Ayesha, who is always by my side in good and bad times and for her endless care, motivation, and patience. Thanks to my other family members for their continuous support and encouragement.

Table of Contents

List of Tables

List of Figures

Nomenclature

ADME - Absorption, disposition, metabolism, and excretion.

AOP - Adverse Outcome Pathways.

BBN – Bayesian Belief Network.

BN – Bayesian Network.

CBR – Critical Body Residue.

Chl a - Chlorophyll a.

CPT – Conditional Probability Table.

DAM – Damage Assessment Model.

EPA – Environmental Protection Agency.

ERA - Environmental Risk Assessment.

EROD - Ethoxyresorufin-O-deethylase.

GPx – Glutathione peroxidase.

GST - Glutathione S-transferase.

GUTS - General Unified Threshold Model of Survival.

ICE- Interspecies Correlation Estimation.

ITD - Individual Tolerance Distribution.

JIP – Joint Industry Programme.

MDA – Malonaldehyde.

MO- Modes of Action.

NOEC - No observed effect concentrations.

NOEL – No Observed Effects Level.

OH - Hydroxyl radical.

PAH – Polycyclic Aromatic Hydrocarbon.

PBTK – Physiology based toxicokinetics.

PNEC - Predicted no effect concentration.

QSAAR - Quantitative Structure Activity-Activity Relationships.

QSAR- Quantitative Structure-Activity Relationships.

RA- Read Across.

ROO – Peroxyl radical.

SD - Stochastic Death.

SER - smooth endoplasmic reticulum.

SOD - Superoxide dismutase.

SSD- Species Sensitivity Distribution.

TD – Toxicodynamics

THM - Threshold Hazard Model.

TK – Toxicokinetics

TKTD – Toxicokinetic and Toxicodynamic.

TOSC - Total Oxygen Scavenging Capacity.

UV – Ultraviolet.

VC - Verhaar Classification.

Chapter 1: Introduction

1.1 Introduction

1.1.1 The Arctic marine region and its major marine species

The Arctic region is defined as the region above the Arctic Circle (66^0 32ˆN) and extends across northern areas of North America, Europe, and Russia (AMAP, 1998). The Arctic marine area includes the Arctic Ocean, adjacent shelf areas (Beaufort, Barents, Chukchi, Kara Seas), the Nordic Seas (Greenland, Norwegian, Iceland Seas), the Labrador Sea, Baffin Bay, Hudson Bay, the Canadian Arctic Archipelago and the Bering Sea (AMAP, 1998). The Arctic region can also be defined using the climate and treeline boundaries (AMAP, 1998). Based on temperature, Arctic is defined as the area north of 10^0C July isotherm. Terrestrial Arctic region is delimited by the treeline boundary, which is defined as the boundary above which trees do not grow. Based on the oceanographic characteristics, the Arctic region is situated along the convergence of less saline and colder waters from the Arctic Ocean and saltier and warmer waters to its south. A representation of Arctic circle according to 66^0 32ˆN latitude and the region of interest for this thesis is shown in Figure 1.

The Arctic region is characterized by the presence of sea ice, extreme weather, periods of prolonged sunlight and extended darkness, long winters with thick sea ice and short summers with periods of open or broken sea ice (Berge et al. 2015; De Vries et al., 2021). Many Arctic marine organisms have adapted to the extreme conditions and thrived in this region (Christiansen et al., 1996). The food chain in this region is relatively non-complex (Hoondert et al., 2020; Kaiser at

al., 2011; Chapman and Riddle 2005). Phytoplankton and zooplankton (amphipods and copepods) are at the bottom of the Arctic marine food web. Among the fishes, Boreogadus saida (Polar cod) has presence throughout the region and largest in stock compared to other fishes (Jonsson et al., 2010; De Vries et al., 2021). Polar cod feed on the amphipods and copepods under the sea ice and in the water column (Jonsson et al., 2010). Many marine birds and different species of seals feed on the Polar cod. Seals constitute major part of the diet of polar bears, while some marine birds, land animals and whale carcass also are a part of its diet (Jagielski et al., 2021; Pagano et al., 2018; Dyck and Kebreab, 2009; Hilderband et al., 1999). Small fishes such as the polar cod form the bulk of the diet of whales in the Arctic region (Kastelein et al., 1994). Six species of seals live in the Arctic, namely, Pusa hispida (ringed seal), Erignathus barbatus (bearded seal), Phoca largha (spotted seal), Cystophora cristata (hooded seal), Pagophilus groenlandicus (harp seal), and Histriophoca fasciata (ribbon seal) (Ryg and Ortisland 1991; Laidre et al., 2015; De la Vega et al., 2020). Three whale species, Delphinapterus leucas (Beluga whale), Monodon Monoceros (Narwhal), and Balaena mysticetus (bowhead whale), are endemic to the Arctic region all year (Kastelein et al. 1994; Laidre et al. 2015). The food chain considered in this research is shown in Figure 2.

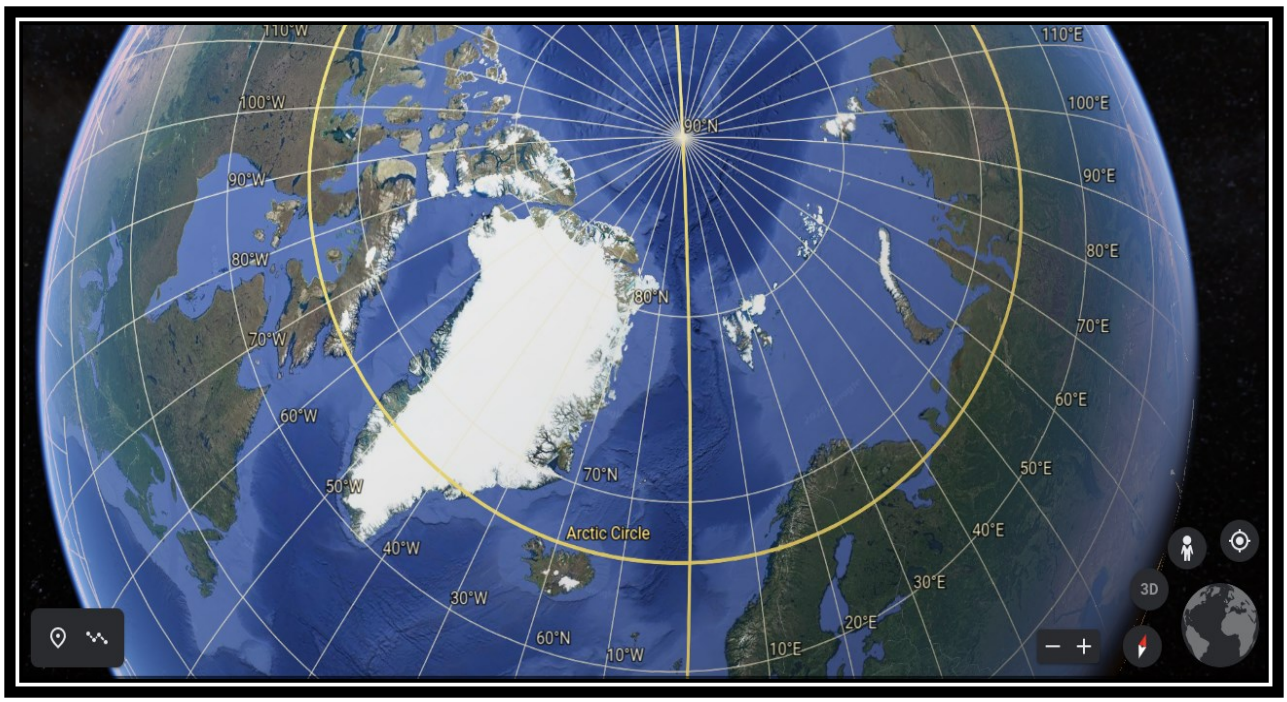


Figure 1: Arctic Circle-Region of interest for current thesis (Source: Google Earth).

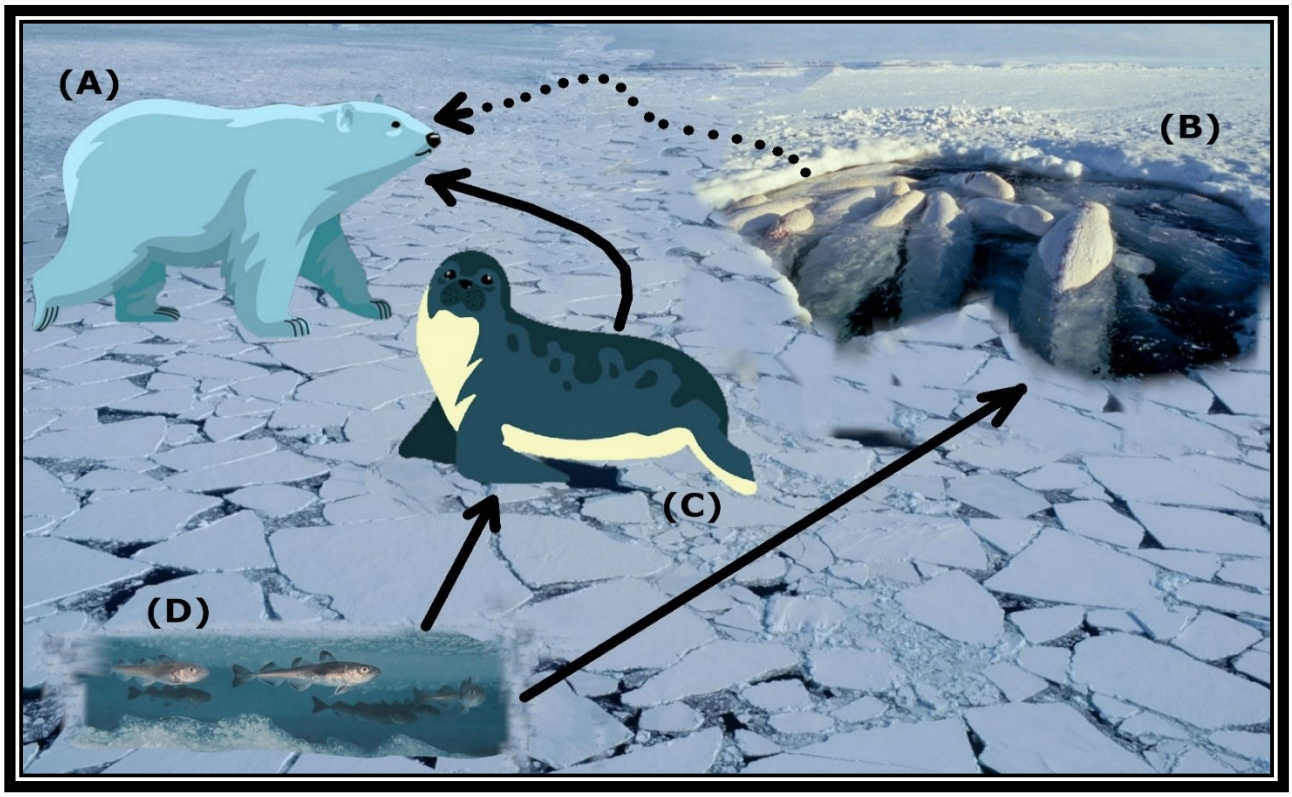


Figure 2: Arctic food web considered in the thesis. (A) Polar bear (B) Whale (C) Seal (D) Polar cod.

Following months of prolonged dark nights in winters, the summers in Arctic is a time of high biological productivity among the marine organisms (David et al. 2016). The increased biological productivity is a result of openings in sea ice and increased sunlight presence in the water column. The resulting algal bloom sets in motion biological productivity across the Arctic food chain (Werner 2006, Fahd et al., 2019). The Arctic sea ice plays a major role in the various life stages of the marine animals in this region. Many phytoplankton and zooplankton thrive in the leads and crevices in the sea ice. They also thrive on the rough underside of the sea ice (Jonsson et al. 2010). The rough under side of the sea ice is also a habitat for juvenile polar cod (up to one year) while the adult polar cod preys in the water column (Jonsson et al. 2010). The seals and polar bears use the sea ice to rear their offspring (NSIDC, 2020). The seals also use the ice for resting periods and polar bears use ice as platforms for hunting the seals. Thus, any changes to the sea ice concentrations or a toxicant spill over/under the ice may lead to significant impact of these marine organisms.

1.2 Problem statement

The Arctic region is rapidly changing to a new state, driven by increasing temperatures and greenhouse gas concentrations in the Earth's atmosphere (IPCC, 2014). Climate change resulted in receding sea ice in summers over the recent years. The seasonal openings in ice opened the possibility of reduced ship travel time along the Northern Sea Route (NSR) propelling interest in shipping activities in the Arctic region. Increased shipping activities in this ocean corridor also substantially increased the risk of shipping accidents. The Genesis of the environmental issues in the Arctic region pertaining to the shipping operations results from the probability of ship accidents resulting in environmental pollution. The Arctic region is an environmentally sensitive region,

with implications lasting over longer periods of time and large uncertainties associated with the region as well (Chapman and Riddle, 2003 & 2005). Arctic animals are over-dependent on one animal as a major source of energy. For example, whale diet comprises of small fish, such as polar cod and capelin, while the seals are significant source of energy requirement of polar bears. Therefore, a significant impact to one species could result in ripple effect of impacts in the Arctic ecosystem. Fortunately, there had not occurred an oil spill in the Arctic region from shipping activities, however, such a unique situation also increases the uncertainties in assessing the impacts to the marine species exposed to a hypothetical or potential spill.

The open ice season is when the Arctic marine species engage in reproduction and the open ice season also provides ample food opportunities for the marine species. This short growth period is very important for these marine species and any disruption, man-made or ortherwise, in the natural and biological processes of these organisms will have a lasting effect on their populations (AMAP, 2010). Changes in the population of the lower trophic animals can lead to cascading effects on the populations of the apex animals in the food chain. Thus, conducting risk assessment of various marine species and predicting change in populations becomes imperative for planning spill remediation measures.

The general steps in environmental risk assessment (ERA) are Hazard identification, Exposure modeling, Toxicological modeling and Risk characterization (EPA, 1992). Hazard identification deals with identifying the toxicant of interest and species of interest. A significant hazard of shipping operations in the Arctic region is the possibility of crude oil spill. The toxicant of interest, Polycyclic Aromatic Hydrocarbon (PAH), is a significant constituent of crude oil causing deleterious effects to the marine organisms. The food web considered for this thesis is shown in Figure 2. The energy flow of the apex marine predators is primarily controlled by polar cod.

Therefore, polar cod is selected as species of interest. The zooplanktons, on which polar cod feed, are not considered in this study.

Risk to marine species is assessed based on the following

Risk = Probability (Exposure) X Probability (Susceptibility)

The goal of the study was to develop a risk assessment model tailored to the environmental conditions of Arctic region and incorporate marine species behavioral factors. The model further assesses the mortality risk to polar cod populations and subsequently assesses risk to higher trophic Arctic marine species. The risk to the higher trophic marine animals could be two-fold, i.e., direct risk from spilled oil and Increased risk from prey scarcity.

1.3 Knowledge gaps

Despite the availability of large database for various aquatic species toxicity data, there is a paucity of toxicity data for Arctic marine species. This is due to limited experimental work on the ecotoxicological effects that have been conducted on the Arctic aquatic species (Song et al., 2019; King and Riddle, 2001; Olsen et al., 2011; Jensen, 2011). Isomorphic animals in temperate regions and Arctic regions differ significantly at various stages of their life cycle (Sorhus et al., 2021; Riget et al., 2020; Gewurtz et al., (2006), Jensen (2011), Hallanger et al., 2011; Veltman et al., 2014), thus, using temperate marine species toxicity data as a representation of the Arctic species is not apt. The way forward for obtaining toxicity data of Arctic marine species is to resort to developing novel in-silico ecotoxicological methods to generate missing toxicity data for the sentinel species selected i.e., polar cod. Polar cod is selected as the sentinel species because it is the main source

of energy flow in the Arctic food web. Seals, whales and many marine birds are highly dependent on the polar cod as diet (Riget et al., 2020).

Huntington (2009) identified the major areas of anthropogenic activity posing a threat to the Arctic environment and reviewed various factors contributing to quantification of these threats. The various fields of human influence included offshore oil and gas activities, shipping, hunting and commercial fishing. Helle et al., (2020), Nevalainen et al., (2011), Nevalainen et al., (2018) had studied the risk due to oil spill in polar bear and whales in the Arctic region. Gallaway et al., (2017), Carroll (2018) had simulated the oil spill scenario in for polar cod and Northeast Arctic cod respectively. The studies predicted the risk of mortality to these fish populations in the Artic and Barents Sea regions. However, no study investigated the cascading and synergistic risk in polar bear and whales due to impact to lower trophic species in the food web from an oil spill (Bender et al., 2021). To achieve this goal of risk assessment of marine species, first the risk to sentinel species, polar cod, was assessed. Subsequently risk to apex marine animals was assessed due to food scarcity on top of the risk from oil spill.

1.4 Research Questions

Keeping in view the need for this research and knowledge gaps in this research, the following research questions were answered by this thesis.

I. Are PAH or crude oil toxicity end points in Arctic species different from their counterpart temperate species?
II. What is the risk to a lower tropic sentinel species in Arctic food web due to oil spill?
III. What is the risk to apex marine predators, such as, polar bear and whale due to a spill?

1.5 Objectives and research tasks

The objectives of the research probes and follows the direction of the research questions.

The current research is planned with following objectives:

I. To investigate the knowledge gaps in the current toxicity assessment model and further investigate their applicability for the Arctic marine species.
II. To investigate the mechanism of mortality in populations of the sentinel species (polar cod)
III. To develop the mortality in the apex marine predators (Polar bear and whale):

The objectives of the research study were further pursued as research tasks. Below are details of research task which led to the publication in peer reviewed journals.

Task 1: - Following the research objective 1, a review of the current ecotoxicological models used to predict the elicited impact/fatality of target organisms due to toxicant exposure was conducted. Further, the applicability of these ecotoxicological models for the Arctic marine animals is investigated.

Task 2: Following the research objective 2, a study of the mechanism of PAH toxicity in polar cod was conducted and environmental and physiological factors that can mitigate or aggravate the toxicity in the polar cod was also investigated. Combine the toxicity mechanism with the environmental and physiological factors and graphically represent them using a Bayesian Belief Network (BBN) model and estimate mortality in polar cod population for given spill scenarios.

Task 3: A study of the recruitment in polar cod stock in the region of interest is conducted and polar cod biomass surviving upon exposure to various spill scenarios is modeled.

Task 4: Following the research objective 3, the mortality in apex marine predators can be two pronged. Apart from the risk from exposure and susceptibility due to oil spill, Arctic region could face an acute challenge, such as mortality in adult and calf, from food scarcity and food web imbalance due to the oil spill in the region.

The research outcomes are listed in Table *1*, while

Figure *3* depicts the research outcomes with respect to the scope and outline of this thesis.

Table 1:Research outcome- Journal papers published as part of the thesis.

Journal paper	**Title**	**Authors**	**Journal, issue, year, link**
JP 1	Aquatic ecotoxicological models and their applicability in Arctic regions	Faisal Fahd, Faisal Khan, Brian Veitch, and Ming Yang.	Marine Pollution Bulletin, 120,2017.
JP 2	Arctic marine fish 'biotransformation toxicity' model for ecological risk assessment	Faisal Fahd, Faisal Khan, and Brian Veitch.	Marine Pollution Bulletin, 142, 2019.
JP 3	Risk assessment of Arctic aquatic species using ecotoxicological biomarkers and Bayesian network.	Faisal Fahd, Faisal Khan, and Brian Veitch.	Marine Pollution Bulletin, 156, 2020.
JP 4	A food chain based ecological risk assessment model for oil spills in the Arctic environment.	Faisal Fahd, Ming Yang, Faisal Khan, and Brian Veitch.	Marine Pollution Bulletin, submitted

1.6 Outline of the thesis:

The chapters of this thesis are arranged such that each of the objectives is described in each chapter.

Refer to

Figure *3* for the overview of the thesis.

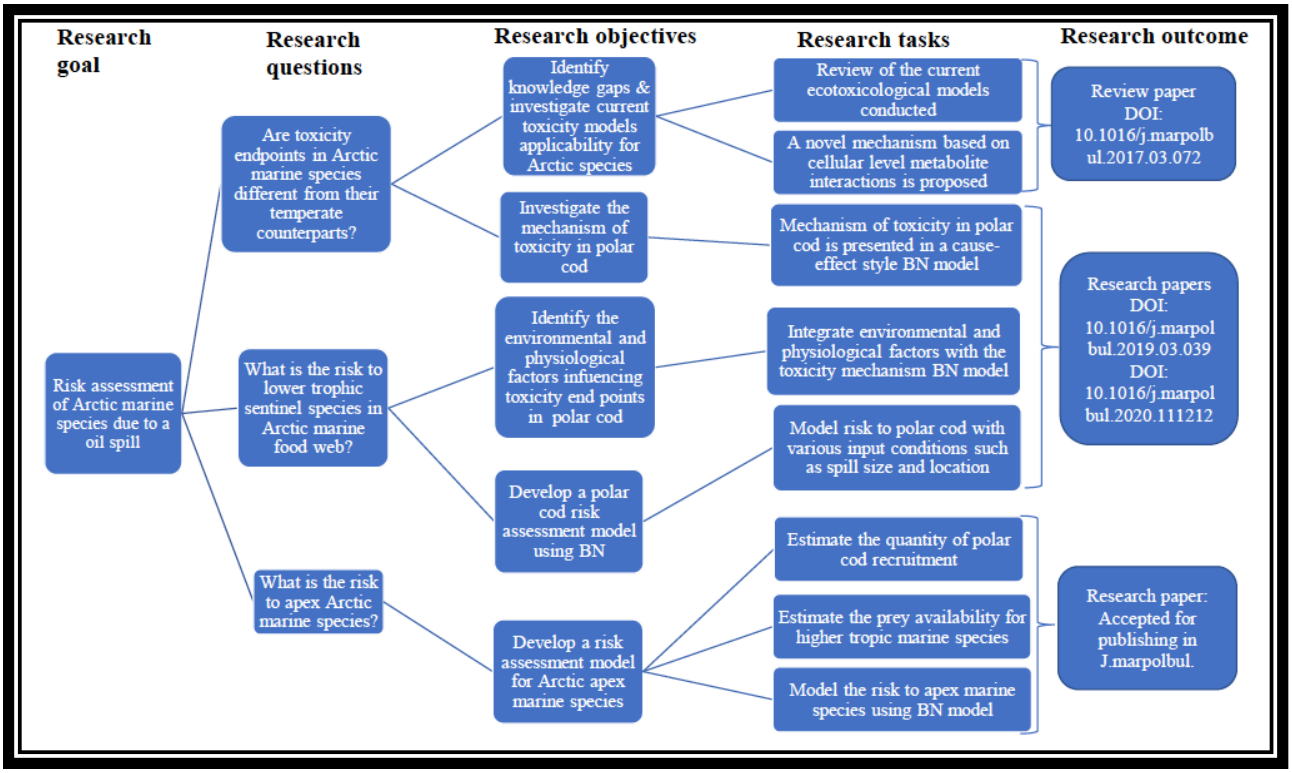


Figure 3: Overview of the thesis (A) Goal, Research question, objectives, and research tasks in the thesis (B) Papers published.

1.6.1 Chapter 2

The absence of dose-response relationships and data on No Observed Effect Concentration (NOEC) is a key knowledge gap for conducting risk assessment of the Arctic marine species. A review of in-silico methods currently used to estimate the dose-response relationships in target organisms and applicability of these methods to assess arctic marine species was conducted and published as Fahd et al. (2017) as a part of this thesis (JP1, Table 1). Species Dose-response relationship help to establish the lethal (LC 50) and sub lethal concentrations for a given exposure

duration. The mechanism based TKTD models take into consideration the concept of biotransformation and detoxification. However, the biotransformation, cellular damage and detoxification constants in the mathematical and empirical equations are based on data of the species toxicity experiments. The toxicity experiments are conducted by exposing the target species (vertebrate and invertebrate freshwater and marine water species) to field like conditions. After an exposure over a given duration, the target species are examined to determine the percentage mortality and recovery. Fahd et al. (2017;2019) proposed to use enzyme activity as proxy for cellular damage and repair in polar cod, which subsequently can be linked to the risk assessment model of polar cod. Current dose-response methodologies and proposed methodology is described in Chapter 2.

1.6.2 Chapter 3

This chapter deals with the developing of the BN model to estimate polar cod mortality due to PAH exposure. However, only one type of toxicity mechanism, i.e., toxicity from biotransformation (metabolism) of the PAH, is discussed in this chapter. The impacts of sea ice, physiological characteristics of polar cod, and extreme light regime and their effects on xenobiotic distribution and metabolism were studied for polar cod. A BN was developed to incorporate all the physiological, geophysical, and environmental factors influencing the ecotoxicity of polar cod. Mechanism of defense and toxicity in polar cod was studied by Varnasi (1989) and is represented as causal effects in the BN model along with physiological, geophysical and environmental factors (Fahd et al., 2019;2020). This chapter is published as JP2 (see Table 1 for details).

1.6.3 Chapter 4

The chapter 4 develops the ecotoxicity model for the mechanisms of lipid peroxidation and biotransformation in polar cod. The ecotoxicity model is a BN based model with inputs from Arctic

environmental and marine species physiological factors as well. The content of this chapter is published as JP3 (See Table 1 for details).

1.6.4 Chapter 5

Chapter 5 describes the risk to apex marine predators. This chapter describes the linkage between the acute impacts of the apex marine mammals with the responses of the polar cod to an oil spill, and the ability for the fish stock to recruit. The BN investigates the indirect effects of n oil spill, such as decreasing stock of polar cod and its cascading effect on the survival and reproduction of polar bears and whales. The content of this chapter is published as JP4 (See Table 1 for details).

1.6.5 Chapter 6

The field of Arctic marine ecotoxicity assessment is an emerging field of research with greater emphasis on the consequences in the ecosystem due to proposed anthropogenic activities in that region. Chapter 6 describes the discussion and conclusions from this research. The chapter also details future recommendations for this research.

1.7 Novelty and contributions

Current ecotoxicological methods use a combination of experimental and mechanism based mathematical equations to model toxicity in marine aquatic species. The concept of damage and recovery in the animals tested in laboratories was incorporated in the modeling equations using various mathematical constants. This research identifies the knowledge gaps in current ecotoxicological models and proposes to develop toxicity modeling that can circumvent the use of toxicity assays for Arctic marine species. This research developed a novel BN based causal effect model to estimate mortality in polar cod due to oil spill exposure. This study digressed from using damage and recovery constants derived from toxicity assays, rather used conceptual cause-effect model wherein each node represents variable in the system. The variables in the BN model include

factors such as, the sea ice conditions, polar cod lipid content, feeding activity, baseline metabolism in polar cod, increased liver activity (biotransformation), and cell damage and recovering ability evident by pertinent biomarker activity.

This novel methodology uses BN, a tool for complex causal dependencies among processes and mechanism. The advantage of a BN model is that the conditional probabilities defining each of the variable or dependencies between different variables can be updated when new knowledge is available.

Another Novel aspect of this research is developing a food chain based ecological risk assessment model for oil spill in the Arctic region. Earlier studies such as, Nevalainen et al. (2017) and Helle et al. (2020), have investigated the impacts/mortality for apex marine species from exposure to oil spills. Modeling studies, such as Carroll et al. (2018) and Gallaway et al. (2017), simulated impacts on polar cod and northeast Arctic cod fisheries. However, no study has made an effort study the synergistic impacts of both direct from oil spill exposure and indirect from changing prey availability in the Arctic food web.

1.8 Co-authorship statement

The contribution of Faisal Fahd, Dr. Faisal Khan, and Dr. Brian Veitch as mentioned here are for all the four manuscripts, while Dr. Ming Yang contributed towards first and last manuscripts, i.e., JP1 and JP4 as shown in Table 1.

Faisal Fahd: Conceptualization and idea formulation, development of design of methodology, software (BN model) development, development of BN model algorithm, performing data analysis and testing of the model. Writing original draft of the manuscript along with all supporting documents for submission to journals. Reviewing and editing the manuscripts based on feedback from co-authors and journal reviewers.

Faisal Khan: Idea formulation of research, development of design of methodology, development of BN model algorithm, guidance in data analysis, and re-organizing and review of the manuscripts.

Brian Veitch: Idea formulation of research, re-organizing, and review of the manuscripts.

Ming Yang: Guidance in development of BN model and in writing of the original draft of JP4 (See Table 1 for details). Organizing and review of the manuscripts JP1 and JP4 (Details in Table 1).

Chapter 2: Literature Review

2.1 Introduction

Arctic regions are of great interest to the petroleum industry due to depleting energy resources in other regions (Camus et al., 2003; Hoop et al., 2011; Carroll et al., 2018; Suprenand et al., 2020). Receding seasonal sea ice has increased access to remote areas in the Arctic region, along with associated human activity, such as hydrocarbon exploration, shipping, and tourism (Chapman and Riddle, 2003; Gardiner et al., 2013; Hoop et al., 2011; Wade et al., 2021). The Arctic ecosystem is a fragile ecosystem (Pecuchet et al., 2020; Hansen et al., 2013; Chapman and Riddle, 2005), vulnerable to impacts from anthropogenic activities and climate change (Hansen et al., 2014). Therefore, the need to understand the impacts to the aquatic animals in case of oil spills, and the capability to conduct environmental risk assessment (ERA) of the aquatic animals are imperative. Two of the steps in the ERA framework involve i) determining the concentration exposed and ii) obtaining the toxicity data (dose-response curves) for the species of concern and, subsequently, for measurement endpoints, such as No Observed Effects Level (NOEL), to determine the species' sensitivity to the exposure (Fahd et al., 2014). The results from these steps in ERA are used to determine the survivability in populations of organisms and their recolonization potential. Such exposure concentrations and toxicity data are obtained by developing the ecotoxicological modeling or conducting toxicity experiments with 'toxicant of concern' and target organisms. To experimentally define a toxicity value for new chemicals and the large number of Arctic aquatic animals is costly and involves techniques that raise ethical issues. Instead, the European Chemicals Legislation, Registration, Evaluation and Authorization of Chemicals (REACH) recommends in-silico ecotoxicological methods to be utilized to generate missing toxicity data (Brinkmann et al., 2014; Patlewicz and Fitzpatrick, 2016). Apart from the EU commission, the

National Academy of Science and US EPA have proposed a shift from whole organism toxicology to a pathway perturbation-based paradigm for toxicity testing and subsequent environmental risk assessment studies (Euling, 2013).

Ecotoxicological modeling refers to the study of the chemical interactions in the target tissues of an individual organism and the effects of the toxicant on life expectancy and other reversible and/or irreversible effects in an organism and, subsequently, the ecosystem (Escher, 2001). Ecotoxicology modeling faces two main challenges: i) the large number of species that can come in contact with the target chemical; and ii) the large number and variety of chemicals that can affect a target organism (Verhaar et al., 1997). The latter is further complicated by the presence of multiple chemicals acting at one time. Owing to the descriptive nature (testing and experimenting) of earlier toxicology studies, large data sets of the dose-response for specific chemicals are available. Databases for ecotoxicity information include ECETOC Aquatic Toxicity (EAT) database and ECOTOXicology (ECOTOX) by US EPA, and TOXicology data NETwork (TOXNET) by the US National Library of Medicine. In spite of the availability of large aquatic animal toxicity literature, there is a paucity of toxicity data for Arctic marine species (Jensen, 2011; Chapman and Riddle, 2003). Toxicity studies in the last decade focussed on developing toxicity assays and thus acquired toxicity data for Arctic marine zooplankton species (Barron et al., 2020). However, limited experimental work on ecotoxicological effects (Chapman, 1993; Chapman and McPherson, 1993; Lenihan et al., 1995; Ling et al., 1998; King and Riddle, 2001; Liess et al., 2001; Olsen, 2011; Jensen, 2011; Barron et al., 2020) have been conducted on the Arctic fish species such as polar cod and Arctic charr.

The current practice of using temperate marine species toxicity data as a representation of the Arctic marine species toxicity data is much debated (Olsen et al., 2011; Olsen et al., 2007). Studies have shown that isomorphic animals in temperate and Arctic regions differ significantly in physiology at some or all stages of the life cycle (Sobek et al., 2010; Hallanger et al., 2011; Olsen et al., 2007; Olsen et al., 2011; Veltman et al., 2014). The physiological factors, such as lipid content and rate of metabolism, in aquatic animals alter the toxicity effects in the organism (Gewurtz et al., 2006; Ashauer et al., 2011; Gergs et al., 2015). Factors such as Voltinism and fecundity also impact the toxic effects in individual organisms (Galic et al., 2014) and Arctic aquatic species have shorter breeding periods than their temperate counterparts. Environmental and geophysical factors, such as presence of sea ice, sediments, and prolonged exposure to UV light, also affects aquatic species toxicity. The sea ice is intertwined with behavioral and feeding habits of many aquatic species, thus playing a major factor in bioaccumulation of contaminants. Bioaccumulation of xenobiotics leads to bio-distribution, biotransformation and eventually a possible toxic scenario.

Sparsely available toxicity data for Arctic aquatic organisms' risk assessment and the unsuitability of most temperate species data to their Arctic counterparts mandates the development of a novel mechanistic model that circumvents the need for animal testing. The proposed mechanistic model should predict the effects in the animals exposed to toxic pollutants considering their ambient environment, behavior and physiology along with using available temperate and Arctic aquatic animal toxicity data and data of known toxicity mechanisms in surrogate organisms. The chapter is published as a baseline review paper in peer reviewed journal, Marine Pollution Bulletin[1].

[1] Fahd F., Khan F., Veitch B., Yang M. Aquatic ecotoxicological models and their applicability in the Arctic regions. Marine Pollution Bulletin, 2017, 120, 1-2,428-437. DOI: 10.1016/j.marpolbul.2017.03.072.

2.2 Literature review

A review of the current in-silico ecotoxicological methods is presented in this chapter and their applicability to the Arctic aquatic environment is also discussed. The study details different approaches described in the literature for the estimation of aquatic toxicity from chemicals of concern. An approach that best suits the effect assessment in the Arctic aquatic animals is identified as using physiology based toxicokinetic (TK) models and molecular damage based toxicodynamic (TD) models. The field of Toxicokinetics (TK) characterizes the exposure to organism by determining the internal concentration, i.e., concentration in the target tissues (Barron et al. 1990, Ashauer and Escher, 2010; Ashauer et al. 2011, Jager et al. 2011, Chen et al., 2012). The field of Toxicodynamics (TD) characterizes the effects/susceptibility from the exposure of toxicant in the target tissues (Ashauer and Brown, 2008; Ashauer and Escher, 2010; Ashauer et al. 2011, Ducrot et al., 2016). The proposed mechanistic molecular damage-based TD model is based on metabolomics and metabolic pathway network. Metabolomics, a branch in ecotoxicogenomics, is the study of the molecule metabolic intermediates and products from the processes of metabolism and excretion. Metabolic pathway network is the illustrations of the interactions of the exposed toxicant to induced enzymatic activity and possible intermediate metabolites and final soluble metabolites before final excretion. Ecotoxicogenomics is defined as the study of the set of the genes or protein expression in an ecological organism to provide insight into its toxicity (Kim et al., 2015). Molecular mechanism in effects is to be quantified in terms of the enzymatic activity (Haber et al., 2001). To accomplish such a task, a great deal of study is required for each of the target species. However, if such a study is available, a molecular mechanism-based effect assessment modeling will circumvent the need for further experimental work and produce data resulting from a mechanistic understanding, as opposed to a statistical (regression) modeling, thus answering the Arctic challenge posed above.

Toxicology studies were initially restricted to the field of drug effectiveness studies and pharmacology, to cope with the changing/new drugs and their use in humans. The focus of researchers in toxicology then shifted from pharmacology to physiology-based pesticide studies (Raies and Bajic, 2016). The assessment endpoints in pharmacology emphasized on sublethal effects, while the endpoints in ecotoxicology focused on lethal (survival) and, to some extent, sublethal endpoints (larval growth and development, reproduction and recolonization) (Ashauer et al., 2011b). Ecotoxicology in pesticide studies dealt with biocidal actions in target organisms and residual toxic effects in non-target organisms. Ecotoxicological models fall under two categories, namely, experimental models and in-silico (computational) models. Most of the early advances in the science of ecotoxicology have been descriptive in nature (i.e., based on experimental works). This led to accumulation of empirical effects data sets of specific pollutants on selected species. Using in-silico methods, rather than only experimental tests, enables computer-based tools to estimate toxicity endpoints and dose response curves. The in-silico methods can further be classified into two groups: statistical and physiology-based methods.

2.2.1 Quantitative Structure Activity Relationships

The rhetoric of the ecotoxicological models has shifted from experimental methods to in-silico methods, such as the Quantitative Structure-Activity Relationships (QSAR) that could determine the relevant concentration endpoints of various chemicals. Quantitative Structure Activity-Activity Relationship (QSAAR) models were developed to assess chemical toxicity endpoints and to extrapolate species to species toxicity endpoints. Subsequently, mechanism-based methods were developed to determine the toxicity endpoints. The mechanism based ecotoxicological methods are developed in two tiers. The first tier (toxicokinetic step) estimates the internal concentration of the contaminant. The second tier (toxicodynamic step) determines the effects from the exposure. Figure 4 presents an overview of various in-silico methods and statistical methods used in the field

of aquatic toxicology to determine toxicity endpoints. The arrows in the Figure 4 do not show dependency, but rather the general progression of the research in the ecotoxicological field.

One of the first non-testing models to determine the acute aquatic toxicity adopted the QSAR and Read Across (RA) models. The QSARs are computational models used to fill data gaps for chemical endpoints using regression analysis of the known toxicity endpoints of chemicals with similar chemical structure as that of the toxicant (OECD, 2004; Netzeva et al., 2008). Hoff et al. (2010) and Patlewicz and Fitzpatrick (2016) presented a detailed description of the QSAR methodology. The first step in QSAR methodology is grouping of the chemicals based on molecular structure; the understanding is that molecules with similar structure have similar toxicity endpoints. Gathered data is processed to achieve normality and the processed data is divided into a training set and a testing set to evaluate internal and external predictive performance, respectively. Grouping of chemicals is an important step in all the statistical approaches, as will be discussed in latter methods. Studies have also grouped chemicals based on their modes of action (MOA), along with grouping based on common chemical functional group (Nendza et al., 2014; Netzeva et al., 2007). Netzeva et al. (2007) argued that combining the data based on modes of action along with chemical class would give a better understanding of the interaction between the chemical and the target organism. The four MOAs associated with different chemicals are described by Verhaar et al. (1992) and are based on selective reaction of the chemical when exposed to a target organism. The QSARs can be a linear relationship between the toxicity and descriptor, or a quadratic relationship. For detailed understanding and illustration of the QSAR model refer Dimitriv et al. (2000), Austin et al. (2015), Barron et al. (1997) and Furuhama (2015).

A couple of the descriptors used in the QSARs are membrane-water partition coefficient (K_{mw}), and octonal-water partition coefficient (K_{ow}).

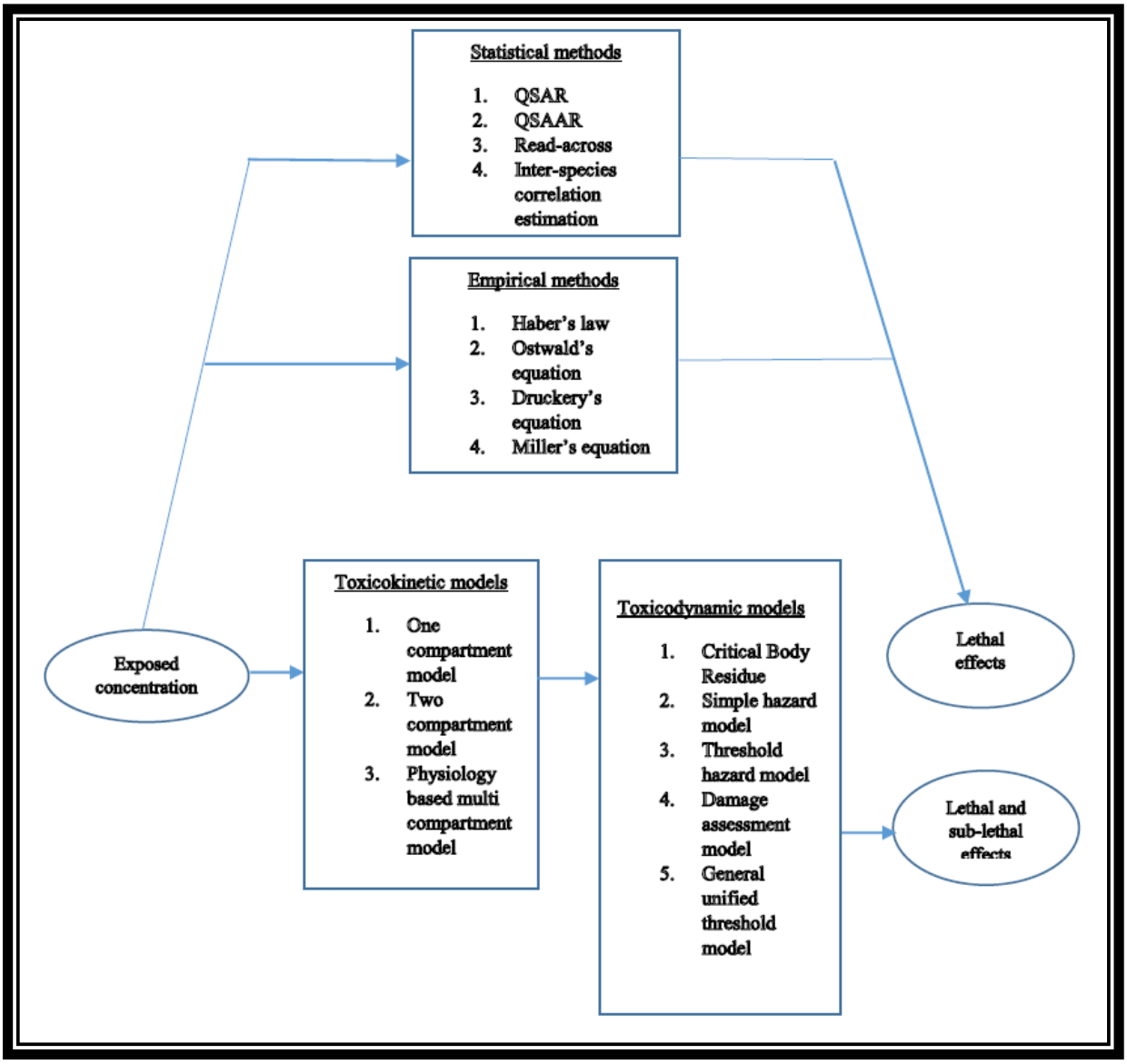


Figure 4: An overview of the current ecotoxicological models

(Statistical models: Netzeva et al. (2007), Kahn et al. (2007), Raies and Bajic (2016), Raimondo et al. (2015). Empirical models: Miller et al. (2000), Doull and Rozman (2000a). Toxicokinetic models: Giulio and Hinton (2008). Toxicodynamic models: Ashauer et al. (2013), Lee et al. (2002)).

The QSAR method is used to predict the toxicity end point of a chemical to any target organism when the chemical adheres to the assumed toxicity MOA. However, QSARs methodology is modified to predict interspecies toxicity data by introducing additional descriptors and thus the methodology is termed quantitative structure activity-activity relationship (QSAAR). Variables, such as molecular weight, certain indicator descriptors, log K_{ow}, and pH, can be taken into account to enhance goodness of fit (Furuhama et al., 2015). An example of QSAR is the relationship between octanol water partition coefficient of a non-polar narcosis causing chemical and acute toxicity of Pimephales promelas as follows (Gramatica, 2007):

$$Log\ (LC_{50}) = -0.846 \log K_{ow} - 1.39$$

Interspecies QSAARs are used to estimate species acute toxicity using measured toxicity of surrogate species and to develop toxicant sensitivity ratios among species. For example, Kahn et al. (2007) studied the relative toxic effects to a fish using ciliate toxicity as a surrogate and developed a relationship between them in their toxic effects using three sensitive structural molecular descriptors: electron distribution, hydrogen bonding ability, and relative nitrogen content. QSAAR developed by Furuhama et al. (2015) is presented here to explain further. The study presented interspecies QSAAR to estimate the relationship between Oryzias latipes (a fish) and daphnia magna acute toxicity of aromatic amines and phenols. The relationship is as follows:

$$Log\ (1/LC_{50}) = 0.975\ da - 0.248$$

The acute toxicity of Daphnia, log ($1/EC_{50}$) is represented by the term '*da*'. The parameters in the QSAAR model, like other statistical methods, have only statistical relationship and no physiological, biological, or mechanistic dependence. Good interspecies coefficient correlation indicates that the studied toxicants may have a similar kind of MOA. Conversely, poor correlation does not explicitly mean that MOA differs between toxicants. Inclusion of log K_{ow} (hydrophobic

compounds) and E_{LUMO} (electrophilic) can improve relationship equations significantly (Zhang et al., 2010).

2.2.2 Interspecies Correlation Estimation

ICE is a web application developed by US EPA. ICE has two modules: the first is species to species extrapolation; the second module is developing SSD. It estimates acute toxicities to aquatic and terrestrial organisms for use in ecological risk assessment (Raimondo et al., 2015). ICE models estimate the acute toxicity of a chemical to a species (predicted species) with no test data from the known toxicity of the chemical, to a species with test data (surrogate species), thereby addressing the data gaps in toxicity for the majority of species. ICE uses log-linear least squares taxon-chemical regressions based on acute toxicity values to predict the relationship between surrogate and predicted species. The ICE models also develop species sensitivity distributions (SSDs) from the multiple surrogate and predicted species to estimate hazard levels protective of most species. SSDs are statistical distributions depicting the variation amongst the species toxicity of a particular chemical. SSDs are used to set the aquatic quality criteria using the 5^{th} percentile concentration (Dyer et al., 2006; Fahd et al., 2014). ICE also compiled more than 2084 species to species regression models based on LC50/EC50 values from 120 fish and invertebrate species. The ICE model could be input with a single toxicity value and estimate acute toxicity values to diverse set of aquatic species. To demonstrate the working of ICE, consider Atlantic salmon (Salmo salar) as surrogate species to predict acute toxicity of amphipod (Gammarus lacustris). Assuming 2 µg/L toxicity value for surrogate species predicts a toxicity value of 0.593 µg/L with a cross validation of 100% and R^2 of 0.89 (https://www3.epa.gov/ceampubl/fchain/webice/). A SSD for the aquatic species can also be developed from webICE, by following the web link https://www3.epa.gov/ceampubl/fchain/webice/.

2.2.3 Read Across method

Read across (RA) is one of the early non-testing qualitative approaches used for filling data gaps in chemical toxicity, physiochemical properties, and eco-toxicity data. The basis of the RA technique is that the endpoint information for one chemical is used to predict the same end point for another chemical within the same 'category'. As in other statistical methods, segregation of the chemical in accordance with their category/group is the most significant step in the RA framework (Raies and Bajic, 2016). The definition of category in RA is more robust than previously discussed methods in that it has many more classifiers apart from structure, functional group, and MOA. A chemical category or group in RA is selected based on common functional group, common constituents, and likelihood of common breakdown products via biotransformation. The reliability of property prediction through RA depends on how well the chemical was grouped. The RA technique is interpolation of a property of a chemical to a similar property of another chemical within a category. The framework of RA is illustrated in ECHA (2015) and Vink et al. (2010). The understanding of grouping such chemicals together also stems from the thought that presence of similar constituents and structure would result in similar chemical and bio-transformational outcomes. The assessment of the substances is fit into various hypothesis scenarios (ECHA, 2015), which are based on:

- Similar functional groups of chemicals;
- Similar functional groups biotransformation to common compounds;
- Similar functional groups but different biotransformed derivatives having the similar pattern in effects;
- Different functional groups biotransformation to common compounds and same type of effects is observed for different source substances. The emphasis is laid on forming a

regular pattern in the effects while there could be some substances in the category that include the absence of such effects; and

- Different functional groups with similar effects evoked in animals but with different biotransformed derivatives.

RA is a hypothesis-based method. Once a scenario is selected, a pre-defined set of the assessment queries is checked. After this, possible outcomes are established. The assessment queries determine the weight of supporting evidence, strong experimental data, or other theoretical basis. The assessment outcomes (AOs) are as follows: i) not acceptable; not acceptable in current form; ii) acceptable with sufficient confidence; iii) acceptable with medium confidence; and iv) acceptable with high confidence. The assessment is said to be with sufficient confidence if only a few data points are available, but the trend is acceptable on theoretical grounds. The contrary (i.e., strong experimental data with non-established theoretical grounds) may also lead to assessment with sufficient confidence. A demonstration of read across method to predict toxicity endpoint is presented by Schultz et al. (2017). The study used 2-ethyl-1-hexanol and 2-propyl-1-heptanol as surrogates to read across toxicity endpoint to untested 2-alkyl-1-alkanols in the C5 to C13 groups. The chemicals were categorized based on common biotransformation compounds.

2.2.4 Empirical Dose-response models

Dose-response models are basic empirical models to describe the relationship between exposed concentration and effects without the need for understanding the toxicokinetics involved in them (see Figure 1). The earliest dose-response model was described by Haber's law (Raies and Bajic, 2016). Most of the subsequent dose-response models are variants of Haber's law, such as Ostwald's toxicity equation and Probit and Logit models (Brown and Foureman, 2005; Ashauer,

2007). The Haber's law is based on the assumption that with the same product of concentration and time, the effects remain the same (Gaylor, 2000). However, this assumption is not always the case, as some chemical toxicity could be more dependent on concentration than time, or vice versa (Miller et al., 2000; Doull and Rozman 2000a). Haber's law leaves no scope for representation of effects of physiology, damage, and recovery after effects in an organism. It is simply a mathematical representation of the toxicity data points from concentration-time experiments. Haber's law is represented by Equation 1 when both α and β are 1 (Miller et al., 2000). This expression facilitates the calculation of toxicity end point (such as LC 50) for various exposure durations (Suter, 2007). Refer to Ashauer (2007), Gaylor (2000) and Miller et al. (2000) for a detailed review of such empirical models used in environmental risk assessment.

Equation 1: Haber's law

$$A_x = (C^{\alpha})\,(t^{\beta})$$

where, A_x is the species-specific constant for x% mortality, and C is the exposed aqueous concentration. Ostwald's equation is also based on a similar assumption, except that a power term is added to concentration to better fit the data. Ostwald's equation assumes β as 1 and α as any number that makes the curve fit the given data. Druckery's model emphasizes time over concentration, i.e., α is 1 and β is any number that fits the curve better. Miller's model emphasizes both the concentration and time (Brown and Foureman, 2005). Another frequently used model to describe the time-concentration-effect relationship is the Probit model. The assumption of the Probit model is that the concentration to cause mortality to an individual organism is normally distributed in a population. Probit model transforms the time-concentration-effect into linear

relationships. Methodology of the Probit models is described in Brown and Foureman (2005), Suter (2007) and Raies and Bajic (2016).

Most experimental/empirical work on toxicity evaluation focuses on the relationship between the aqueous concentrations and the effects in the target species. Quantification of the chemical at the target site in an organism, rather than the source, helps to better understand the effects in the organism. Physiology based models account for what the target site is, the concentration at the target site, and the mode of action, unlike the statistical ecotoxicity models that look for effects at the organism level (Escher and Hermens, 2004). There are two tiers in the physiology-based models: the first tier is toxicokinetics (TK) and the second tier is toxicodynamics (TD). TK deals with the uptake, distribution, biotransformation, and depuration of the chemical and is an important tool in toxicity assessment. TD models link the exposed concentration with damage and survival in the organisms. TK models when combined with the TD models can predict the toxic effects to the organisms (Ashauer and Escher, 2010; Stadnicka et al., 2012). As they are mechanism-based models, they can be applied to a wide range of chemicals and also for extrapolation between different species and chemicals (Ducrot et al., 2016).

2.2.5 Physiology-based models

2.2.5.1 Toxicokinetic models

The TK models are generally one or multi-compartment models. In a one-compartment model, the chemical concentration in the organism is assumed to be uniform and homogenously distributed in the animal, whereas the multi-compartment model assumes that the concentration of the chemical differs in various organs and tissues (Stadnicka et al., 2012). The earliest application of TK models was in the field of pharmokinetics. The TK approach was later extended for the aquatic

toxicology (Barron et al. 1990). One-compartment models are more easily developed as they require fewer physiological parameters to estimate the chemical concentration in the whole body of the organism (Barron et al. 1990; Stadnicka et al. 2012). The simplest one-compartment model assumes that the animal body behaves like a well-mixed single compartment. This could be mathematically presented as follows (Stadnicka et al. 2012):

Equation 2: One compartment TK model

$$\frac{d}{dt} C_{int}(t) = K_{in} * C_{source}(t) - K_{out} * C_{int}(t)$$

where $C_{int}(t)$ is the internal chemical concentration, $C_{source}(t)$ is the chemical concentration at the source, K_{in} is the uptake rate constant and K_{out} is the elimination/depuration rate constant.

The TK models are a mixture of theoretical and empirical approaches that use one, two, or more compartments and the parameters describing the processes are based on fitting a model to the experimental data on the time course of chemical concentration in a target organ/compartment. Giulio and Hinton (2008) discussed the theory and mathematical representation of one, two, and multi-compartment TK models. A multi-compartment TK model increases the complexity in assessment by identifying and partitioning the contaminant in each possible target tissue and organs. An example of the multi-compartment TK model is the physiology based toxicokinetic (PBTK) models. The framework of PBTK models for fish, rat, and bird are illustrated in Giulio and Hinton (2008). Developing a PBTK models requires the knowledge of the presence and abundance of the target tissues. Further, the biological defense mechanism is of great relevance to understand the species selectivity and sensitivity (Escher and Hermens, 2004). PBTK model is based on the physiology and biochemistry and gives a better understanding of the uptake and disposition of a chemical. The numbers of compartments in the model depend on the inclusion of

each target organ as a separate compartment (Devillers, 2009). However, combining multiple target organs as one is frequently done if the metabolic and accumulation characteristics of these target organs are not significantly different. PBTK model platforms were developed for various organisms such as rat, fish, and birds. The fish PBTK model employs a set of mass balance differential equations determining the toxicant concentration with respect to time in each of the five tissue compartments: liver, kidney, fat, and richly perfused, and poorly perfused tissue (Nichols et al., 1990; Giulio and Hinton, 2008). The differential equations in the PBTK model describe the absorption, disposition, metabolism, and excretion (ADME) processes. Two categories of parameters are required to simulate the toxicokinetics in PBTK model: i) physiological parameters that are chemical independent; and ii) chemical dependent parameters determined by in-vitro methods, or QSAR (in-silico methods) (Bessems et al., 2014). Giulio and Hinton (2008) illustrates the various pathways for absorption, distribution, and elimination of xenobiotic compounds in fish that form the basis for selection of compartments in TK models.

2.2.5.2 Toxicodynamic models

Toxicodynamic (TD) models establish the link between concentration of a toxicant and the effects. The relationship between toxicity (T), TK, and TD is best defined as follows (Rozman and Doull (2000 b)):

Equation 3: Relationship between TK-TD

$$T = f(E, K, D)$$

where E is the exposure and function of concentration and time (E = *f*(concentration, time)). Toxicokinetics, K, is *f*(uptake, elimination), and Toxicodynamics, D, is *f*(damage, recovery). However, due to lack of the mechanistic understanding of the effects of the contaminant, the hazard or damage is estimated based on the statistical and empirically derived endpoints, such as the

predicted no effect concentration (PNEC), or no observed effect concentrations (NOEC). Knowledge of the target sites for the toxicant, the organism's metabolism, and its defense mechanism are ignored in such approaches. Such ignorance stems from the understanding that despite the biological variability, behavior at the target sites and defense mechanisms are constant across different organisms (Escher and Hermens, 2004). However, to identify the exact underlying mechanism of toxic action in an entity is easier said than done. This is owing to variability in the specific interaction of the xenobiotic with various target sites and at times non-specific interactions may also lead to toxic outcomes (Escher and Hermens, 2004). The target sites in an organism could be lipid membranes, proteins, and DNA. The subsequent effects include enzyme inhibition, membrane damage leading to disturbance to its integrity, and DNA damage (Groh et al., 2015). Fig. *5* describes the process of TD modeling, using different approaches. Jager et al. (2011) discusses the processes mentioned in Fig. *5* in detail and proposed a unifying approach to different TD methods.

2.2.5.2.1 Hazard model

Establishing a link between internal concentration and effects is accomplished by using a hazard model or a damage model. Hazard rate, h (t), is the probability that an organism dies per time interval and is determined by experimental data. The hazard rate is further linked to the probability of survival, S, by Equation *4* (simple hazard model) (Ashauer, 2007). The conceptual framework of various TD models using the hazard concept is discussed in this section.

Equation 4: Probability of survival using hazard model

$$S\,(t) = e^{-\int_0^t h(t)dt}$$

Where, h(t) is the hazard rate and S(t) is the survival rate.

Another way of linking internal concentration to effects is via the concept of damage and recovery. Damage parameter is linked to hazard rate and subsequently an organism's survival is determined, as explained in the damage-based TD models in this section. Ecotoxicological survival models are categorized in two approaches, namely, Individual Tolerance Distribution (ITD) concept and Stochastic Death (SD) models (Ashauer and Brown, 2008; Ducrot et al., 2016). Both the approaches are used to predict the consequences to a target population after repeated, chronic, and sub chronic exposure to a toxicant. ITD concept assumes that each individual has its own tolerance to a toxicant, and the effects in the individual show when this exposed concentration exceeds the tolerance. The SD concept assumes that each individual has the same probability of generating the effect for a given toxicant concentration. To better illustrate, consider a scenario where a sample of fish is exposed to a toxicant concentration that kills 50% of the population. Once the surviving fish are removed from the test conditions and placed in no toxicant condition for a sufficient time for recovery, and then re-exposed to the same prior concentration (LC 50), according to ITD, there would be no subsequent deaths as the sample already survived the concentration. Under SD concept, when the sample of survived fish is re-introduced to the LC 50, it results in death of 50% of the sample. Therefore, each time the surviving sample is re-introduced to LC 50, only 50% of the exposed population survives, such as 50% of the original sample for the first trial, 25% of original population for second trial, then 12.5% of the original population, and so on (Ashauer et al., 2013). Both these underlying assumptions for death in test organisms have been used in ecotoxicology for two decades and the SD model was also included in the OECD guidance for ecotoxicity data analysis (Ducrot et al., 2016; OECD, 2004). Interestingly, researchers such as Heagler et al. (1993) and Newman and McCloskey (2000) have experimentally demonstrated that

neither of the two approaches truly represents the biological reality of death mechanism in test populations.

2.2.5.2.2 Critical body residue model

One of the frequently used TD model is the critical body residue (CBR) model. The CBR theory links lethality to aqueous concentration and whole body residue. CBR is the internal concentration at the target tissues of an animal at the end of a toxicity test in which 50% of the test animals survive. The CBR is considered as a surrogate for the lethal dose in an animal and toxicity is better addressed with critical internal concentration (Celsie et al., 2016). The CBR theory only considers the toxicokinetics and therefore takes into account the contaminant uptake and elimination processes in the body. The internal concentration is calculated based on the following equation (Ashauer, 2007):

Equation 5: Critical body residue

$$CBR = \left({K_{in}}/{K_{out}}\right) LC50\ (1 - e^{K_{out} t_{LC50\ test}})$$

where, K_{in} is uptake rate constant, K_{out} is the elimination rate constant, and $t_{LC50\ test}$ is the duration of the toxicity test. An implicit assumption in the CBR is that the toxic action is instantaneous and completely reversible. The equation above could be used to predict the LC50 values for different durations. The validity of CBR concept for narcotic compounds was questioned by Lee et al. (2002). It was observed that the CBR model could not predict the toxicity of PAH in Hyalella Azteca.

2.2.5.2.3 Threshold hazard model

THM assumes that there is a concentration below which there is no effect or damage to the organism. This assumption is also supported mechanistically. In a steady state condition, the

internal threshold concentration is linked to external concentration. Hazard function for THM is as follows:

Equation 6: THM survival probability

$$H\ (t) = \int_0^t K_k\ max\{C_{int} - C_{int-threshold}; 0\}$$

Survival probability for an individual, S(t) is given by

$$S\ (t) = e^{-H(t)}$$

where, C_{int} is the exposed internal concentration, $C_{int\text{-}threshold}$ is the threshold concentration and K_k is killing rate constant.

2.2.5.2.4 Damage assessment model

Damage Assessment Model (DAM) is a step ahead of other effects models discussed above as it considers an important biological process of damage and recovery in an organism (Lee et al., 2002). DAM considers the toxicokinetic and toxicodynamic processes in the animal. DAM is developed with no prior assumption of reversibility of the toxicodynamics, as assumed in CBR model. This model includes parameters for damage and recovery processes in the target species. The internal concentration in DAM is estimated using a one compartment, single first order approach. Damage (D) accumulates in proportion to accumulated body residue and damage repair is proportional to the accumulated damage. Damage is calculated as follows (Lee at al., 2002; Lee et al., 2006):

Equation 7: Equation for damage

$$dD/dt = K_{accural}C_{int} - K_{recovery}D$$

The link between damage or hazard and survivability is given as follows

Equation 8: Equation for hazard

$$H(t, C) = K_1 D(t, C)$$

Equation 9: Survivability equation

$$S(t, C) = e^{-H(t,C)}$$

where, $K_{accural}$ is the rate constant for accrual of damage, $K_{recovery}$ is the rate constant for recovery or repair. K_1 is the constant obtained by fitting data curve for damage (D).

2.2.5.2.5 General Unified Threshold Model of Survival

General Unified Threshold Model of Survival (GUTS) model unifies different underlying hypotheses and assumptions mentioned above in a single model. The framework and methodology of GUTS model was detailed in Jager et al. (2011).

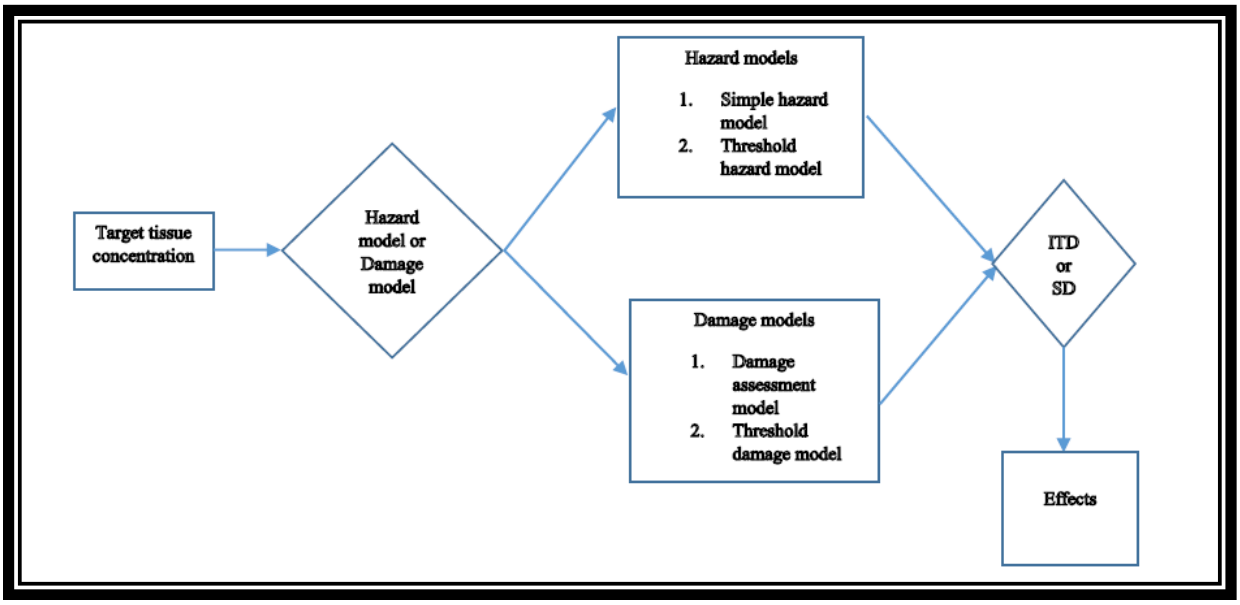


Fig. 5: Approaches in toxicodynamic modeling

ITD: Individual Tolerance Distribution, SD: Stochastic Death.

2.2.6 Bayesian Network

Environmental risk assessment is a process of estimating the probability and consequences of potential adverse impacts to environment due to anthropogenic activities. The proposed research incorporated toxicity mechanism modeling in Arctic marine species risk assessment. Such complex interdependencies between various variables are better represented using a Bayesian network (BN). BN is a probabilistic modeling approach consisting of a directed acyclic graph defining the dependencies between various variables in the network. The nodes and their relationships in a BN are defined based on an algorithm from data or through expert opinions. The BN structure developed could mimic the causal relationships in a system, thus representing complex toxicity mechanism occurring in the Arctic marine fish. The representation of the causal dependencies could enable evaluation of the effects cascading in the ecosystem. The BN also provide a convenient and coherent approach to represent the uncertainty in the process and data of the complex environmental system model. Each of the variable in the environmental system is represented as a node in the BN. The arcs connecting the nodes represent the probabilistic dependencies between the two variables. The strength of the dependencies is defined by the conditional probability table (CPT). Each node of the BN could have 2 or more states. For example, A node could have low, medium and high as its states. The states of the node could be defined qualitatively or quantitatively. The result from the BN is a distribution of the probable values for each of the states in the final node. BN can also act as a scenario synthesis tool, wherein the probability of different combination of events, such as the spill scenarios, are predicted. A BN represents the causal dependence between the nodes connected by arrows. The parent nodes are the nodes with no incoming dependence from other nodes. The probabilities of these parent nodes are the prior probabilities. The prior probabilities are calculated based on expert elicitation or based on the frequency of occurrence of the evidence. The intermediate nodes are nodes that have a

parent node and further influence other nodes (child nodes). Intermediate nodes are described by conditional probability tables (CPTs). An advantage of the BN in such modeling is that with new knowledge/data, conditional probabilities can be updated for updated results from the model. The three main components of a BN are the structure of the network, data discretization, and data parametrization (Pitchforth and Mengersen, 2013).

Many studies, such as Hoyle and Maunder (2004), Kaikkonen et al. (2020) and Marcot et al. (2001), used BN to model population dynamics in protected species. BN is especially used in studies where data can be used in conjunction with reliable field observations and beliefs. BN is ideal for developing frameworks of models with complexities in the data availability and uncertainty in the causal process in models.

2.3 Discussion

Statistical methods are based on regression analysis and appropriate chemical classification. While the mechanistic models are based on quantification of ADME processes, many input parameters in these models are at times based on statistical interpolation. Statistical methods and mechanistic methods for endpoint prediction have many inherent advantages and disadvantages. The QSAR methodology based on chemical class is useful to determine the relationship between chemical structure of a contaminant and its toxicity. The advantage of the QSAR chemical class-based models is it being relatively simple, transparent, and reproducible with a small number of descriptors, such as hydrophobicity and lipid index. The QSAR methods are known to produce acceptable results that can be used for regulatory purposes. In general, the log K_{ow} based QSAR models are in agreement with the OECD requirements for toxicity data. The drawbacks of the QSAR models include not always having a perfect definition and domain of the chemical class. For example, the functional group of hydroquinones is an outlier from the phenol class. They had

to be modeled separately with a different set of descriptors highlighting the oxidizing potential to more electrophilic quinones. When chemicals with unusual toxicity are observed, they are excluded from the data set as outliers and explained as chemical with possible biotransformation or a different mechanism of action. Another method of classifying the chemicals as mentioned above is by their modes of action based on Verhaar Classification (VC). One of the main disadvantages of the VC is the prominent presence of chemicals that do not confirm to established classification (Netzeva et al. 2007). The predictivity of the model is heavily dependent on its use for chemicals falling in its applicability domain. Variability of the descriptors and classification of reactive chemical as narcotic are some of the reasons for the failure of the model. Sometimes the chemicals with similar structure may also react differently. Some aromatic amines and phenols that are used as base material for many industrial products are classified as polar narcotic chemicals. The polar narcotic chemicals are slightly more toxic than nonpolar narcotic chemicals. However, some aromatic amines and phenols are not characterized as polar narcotic chemicals due to high reactivity, or because their structure is not classified in accordance with VC. A functional group of chemicals, such as, anilines are more toxic to daphnia magna than other polar narcotic chemicals and further, the mentioned excess toxicity is not as prominent in the case of 2-substituted aromatic amines (Ramos et al., 2002). Aruoja et al. (2014) evaluated the toxicity of anilines and phenols for the alga Selenastrum capricornutum and found that aniline toxicity does not depend on hydrophobicity while the toxicity of phenols does. These are some of the complexities involved in QSAR models. The chemical's classification could be a problem, or the chemicals of a functional group not reacting in accordance with the VC could be another problem. Furthermore, a functional group could react or be more toxic to an organism and less toxic to another organism. A review of such earlier models to determine aquatic species (such as, fish, daphnid, algae and

ciliates) toxicity was conducted by Netzeva et al. (2007). Each of the approaches mentioned have some inherent difficulties which could be summarized as follows: not all chemicals in a functional group have the same mode of toxic action and not all species in a taxon interact with the chemicals of a functional group in similar fashion.

Furuhama et al. (2015) and others have demonstrated the robustness of QSAAR methodology by identifying the descriptors that best predicted the toxicity from the training data set of aromatic amines and phenols. The study selected daphnia toxicity, molecular weight and other descriptor variables that isolated various molecular substructures in amines and phenols. Correlation between tetrahymena and pimephales toxicity data was established and it was concluded that there was a similarity between the potency of chemicals (Kahn et al., 2007). Although additional molecular descriptors in a QSAR improve the extrapolation of effects from surrogate to target organism, the development of QSAAR and QSAR are highly influenced by the training set. Another major disadvantage of the QSAR and QSAAR is the requirement for a large and structurally diverse database of existing compounds, along with ability to process multiple descriptors (Kahn et al., 2007; Furuhama et al., 2015). The chemical's mechanism of toxicity in one organism can be very different from the chemical's MOA of another organism owing to availability and abundance of target sites or selected experiment endpoints (Furuhama et al., 2015). These studies have developed interspecies QSAARs for Oryzias latipes and daphnia, for algae and daphnia, and further used these data as training set data to obtain toxicity correlation between Oryzias latipes and algae. QSAARs are not valid for strictly non-polar narcotics as they don't have such molecular descriptors that would affect to baseline toxicity.

ICE model is based on simple regression analysis of toxicity data and is easily reproducible. Just like its counterpart statistical methods, a large data set is a prerequisite, which is a limitation in the case of arctic aquatic animals. Also, statistical methods only predict the lethality/survival endpoint. A significant drawback of statistical models is their inability to consider the recovery potential of an animal while predicting the toxicity data. Empirical models also share some of the advantages of the other in silico methods, such as the ease of interpretation and implementation. Empirical models discussed above also serve the purpose of interpolation between various exposure times and doses that are within the range of the experimental data. However, these models have no ability to extrapolate to other chemicals and other organisms, as they do not consider the concentration at target tissues and various biological processes such as absorption, distribution, metabolism, depuration, detoxification, and damage.

Physiology based modeling (such as TK-TD models) has been shown to be a useful tool for toxicological research, with increasing opportunities to use these results in more scientifically based risk assessment that is less reliant on animal testing. These types of models can formalize knowledge about the toxicity and organism sensitivity, create new hypotheses, and simulate temporal aspects of toxicity, making them useful tools for risk assessment. TK-TD models allow realistic representation of exposure and effect patterns and mechanisms. Current risk assessment techniques compare the predicted environmental exposure with their elicited effects measured at constant concentration. This concentration usually is a time weighted average or maximum exposed concentration. In reality, the exposure concentration varies over time, especially so for Arctic regions, where presence of sea ice drives fate-transport of the contaminants. TK-TD models are dynamic models, so they better accommodate temporal variations in exposure concentrations. Stadnicka et al. (2012) conducted a comparison of the suitability of a one compartment TK model

and a multi-compartment TK model in the estimation of the toxicity endpoint and concluded that the multi-compartment models, i.e., the PBTK model, outperformed the single compartment models. One and two compartment TK models calculated the uptake and disposition in fish by considering the accumulation of the poorly soluble and lipophilic compounds in the lipid of the organism. Although such a methodology provided good results for some chemicals, it could not be used for compounds displaying multi-exponential kinetic behavior (Nichols et al., 1990).

TK-TD models are better equipped for meaningful interpolation and extrapolation of non-tested chemical exposure or toxicity endpoints for non-tested animals of consideration (Ducrot et al., 2016). Gergs et al. (2015) investigated three species of crustaceans and concluded that differential body sizes had a strong correlation with their variation in sensitivity. The consideration of life-stage or size dependent sensitivity of individual organisms and consequently its implication on population demographics were studied by Tarsi and Tuff (2012), Stark et al. (2004), Gergs et al. (2013), Kulkarni et al. (2013), Jager (2013), and Gergs et al. (2015). Interspecies variation in sensitivity to xenobiotics is influenced by species traits and ambient environmental factors. The species traits and environmental factors causing variation in sensitivity can be related to both the toxicokinetics and toxicodynamics. Nyman et al. (2014) discussed various factors in toxicokinetics that influence the pesticide sensitivity in aquatic invertebrates such as Gammarus pulex, Gammarus fossarum, and Lymnaea stagnalis. However, the toxicodynamic factors influencing the mechanism of toxicity in marine and terrestrial organisms had extremely limited prior research.

Organism recovery time depends on the time course of TK and TD, which makes the TK-TD models suitable for calculating organism recovery time. TK-TD models are more powerful than

the traditional dose-response models because they incorporate chemical concentrations as well as temporal dimensions. TK-TD models provide a framework to understand the causes of variability in species' responses to a chemical and the different responses triggered in a species when in contact with different chemicals. Thus, the TK-TD models overcome one of the major disadvantages of statistical methods, by explaining the statistical nearness in results with a physiological framework. TK-TD models serve well to simulate interspecies differences in toxic response, variability in tissue doses and extrapolation of toxicity endpoints. The ability of the TK-TD models in assessing variability, thereby extrapolating toxicity endpoint data for other organisms, could be used to estimate such data for the Arctic aquatic organisms.

The major disadvantage of PBTK is their demand for estimation of numerous parameters within a complex model, thus restricting their general use. The complexity in PBTK models is due to high interspecies variability in target sites and tissues, metabolism, and repair mechanism in the animals. The chemical lodged in different target tissue and organs is metabolized, and biotransformed, resulting in damage and subsequently incomplete or partial repair and recovery. Sometimes at same target sites, different chemicals may be metabolized in different fashion. TK-TD models have the potential for extrapolation across species, although this potential is currently underutilized (Veltman et al., 2014). This is attributed to lack of quantitative understanding of key biochemical and physiological processes that determine species sensitivity. Arctic aquatic ecotoxicological modeling can significantly advance if toxicokinetic and toxicodynamic parameters are quantitatively tied to species characteristics and environmental factors.

2.4 Proposed TD approach for Arctic fish

The proposed approach recommends the use of statistical methods and toxicokinetics in combination with toxicodynamics to model toxicity endpoints. Statistical methods have advantages, the biggest of which is simplicity of use. Amongst the disadvantages, the most important is the requirement for a large data set for extrapolation. Various parameters representing different physiological process in TK-TD modeling are also based on experimental observations from toxicity assays (Bessems et al., 2014). A case in point is the partition factors in various compartments (gills, tissue, liver, and blood), and coefficients for damage and recovery that are based on experimental results. For the Arctic aquatic animals, the requirement of a large data set is not met, leaving the way only for a 'species traits' and physiology-based approach that can estimate the internal concentration, and further determine the toxicity endpoint values for the chemical of concern. The challenge to be addressed for the Arctic species is estimating LC50 values without the need for toxicity experiments. To achieve this objective, the key questions to be solved in modeling are:

- What are the factors affecting the toxicokinetics in the Arctic region (e.g. sea ice) and how to incorporate them in a model?
- How can the negative ramifications from intermediate metabolites in aquatic animals be quantified?
- What is the mechanism of recovery in aquatic animals? And how can the recovery be quantified in terms of internal concentration, enzymic activity and metabolic rates?

The ecotoxicology scientific community has identified and proposed the importance of linking species traits and other biological factors to TK processes, such as absorption, accumulation and distribution, during the last decade (Dorne, 2010; Escher and Hermens, 2004). Experimental and modeling works linking the species traits to TK processes are recent (Chen et al., 2012; Rubach et al., 2012; Galic et al., 2014; Gergs et al., 2015; Nyman et al., 2014; Pery et al., 2014; Engraff et al., 2011). However, species traits affecting TD processes (damage and recovery) have not been modelled, with few exceptions. One such exception modelled toxicity due to exposure from silver in freshwater organisms (Veltman et al., 2014). The study modelled TD processes by linking mortality in aquatic species to the loss of sodium in the whole body. Readers can refer to Veltman et al. (2014) for detailed chemical interaction of silver in aquatic species target tissues.

The concentration of contaminant that can cause damage is a function as shown below:

Equation 10: Concentration of contaminant causing damage

$$\left(C^{f}_{Internal\ target\ tissue}, CM_b\right) = f(C_{exposed}, C^{a}_{metabolized}, C^{b}_{metabolized})$$

where $C^{f}_{\text{Internal target tissue}}$ is final concentration in the target tissue, $C^{a}_{\text{metabolized}}$ is the concentration of contaminants metabolized safely with no secondary toxicity, $C^{b}_{\text{metabolized}}$ is the concentration of contaminants metabolized with secondary toxicity, CM_b is concentration of metabolites from $C^{b}_{\text{metabolized}}$.

Damage due to ($C^{f}_{\text{Internal target tissue}}$, CM_b) is dependent on the recovery and repair parameter. Currently the recovery parameter is derived from experimental results. Expressing final concentration of a contaminant causing damage and its response is better when it is linked with the enzymatic activity in the corresponding tissues in the aquatic animals. The molecular

mechanism of the effects in aquatic species is measured by identifying and quantifying the biomarker responses in the species. Although the observation of the biomarker response is an outcome of the experimental setup, the enzymes and macromolecules involved can be quantified in terms of density per unit weight. This calculation helps to quantify the possible response from the animal. Such a response not only estimates the lethal effects, but also the sub-lethal effects in the species. The knowledge of active genes, transcriptors and enzymes that damage, metabolise, and detoxify is required (Billiard et al., 2008). This knowledge helps in developing a time series of cell damage. This individual cell damage is further quantified in organismal level and even further to community level. Enzymes as a link between concentration and effects in toxicant are illustrated in Figure 6.

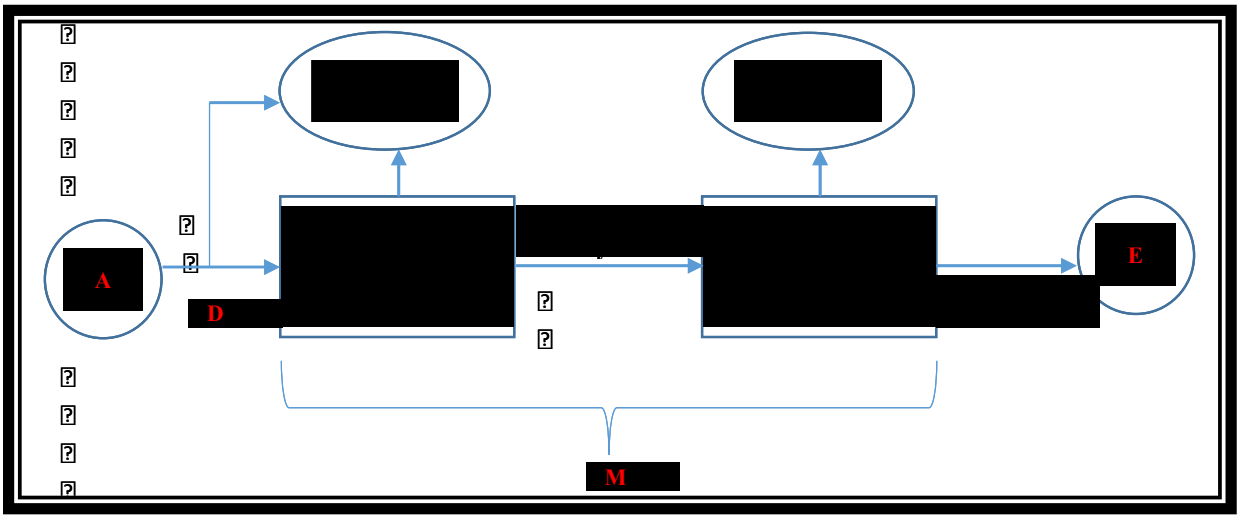


Figure 6: Enzymes activity as a link between xenobiotic concentration and its effects (*A D M E: Absorption Distribution Metabolism and Elimination of a xenobiotic in an organism).*

The mechanism of toxicity of Benzo[a]Pyrene (BaP) was well established by various studies and its toxicity mechanism is used as representative for PAH in general aswell (Varanasi, 1989). PAH is used as an example to illustrate the above process in Figure 7. The major mode of toxicity of PAH is due to interference with cellular membrane function and membrane associated enzyme

systems. The most important effect leading from the PAH/PAH metabolite exposure is covalent bonding of PAH to cellular macromolecules such as proteins, DNA and RNA, which cause cell damage, mutagenesis, tetragenesis, and cancer (Van Tiem., 2011). PAH may also be bound to the surface of plasma membrane causing perturbations in the membrane making it more permeable. The degree of carcinogenity in the fish can be related to the structure and reactivity of the major metabolites produced by the cytochrome P450 MFO and epoxide hydratase systems. DNA adducts are used as an indicator of exposure of fish to genotoxic compounds. A methodology tying the PAH concentration, PAH metabolites concentration, and enzymatic activity to the amount of target tissues may act as an analytical method for cellular damage determination and subsequent sub lethal and lethal effects in the aquatic animal body. Exposure of the fish to PAH causes molecular changes and subsequent integration of the molecular data with other physiological, environmental, and geophysical factors will lead to better TK-TD models linking concentration to lethal and sub-lethal effects. Such models limit the role of new experimental methods on Arctic species and develop an efficient effects prediction model for the aquatic species.

Research in aquatic risk assessment is moving towards incorporating realistic representation of exposure patterns and quantitative mechanistic approaches. Owing to the dynamic nature of the sea ice, the temporal and spatial variability of exposure conditions is even more predominant in the Arctic regions. The most significant factors that contribute to such variation are the presence of sea ice and harsh weather conditions. TK-TD models are better suited than statistical and empirical methods to model ecological risk in the Arctic region. Arctic aquatic risk assessment TK-TD model should consider physiological, environmental, and geophysical factors affecting the absorption, distribution, metabolism/biotransformation, and elimination (ADME in figure 6) of the

toxicants. To accomplish TK-TD modeling, a host of species-specific parameters are required. The proposed model uses known temperate species toxicity data as surrogates to estimate the Arctic species data, as shown in Figure 7.

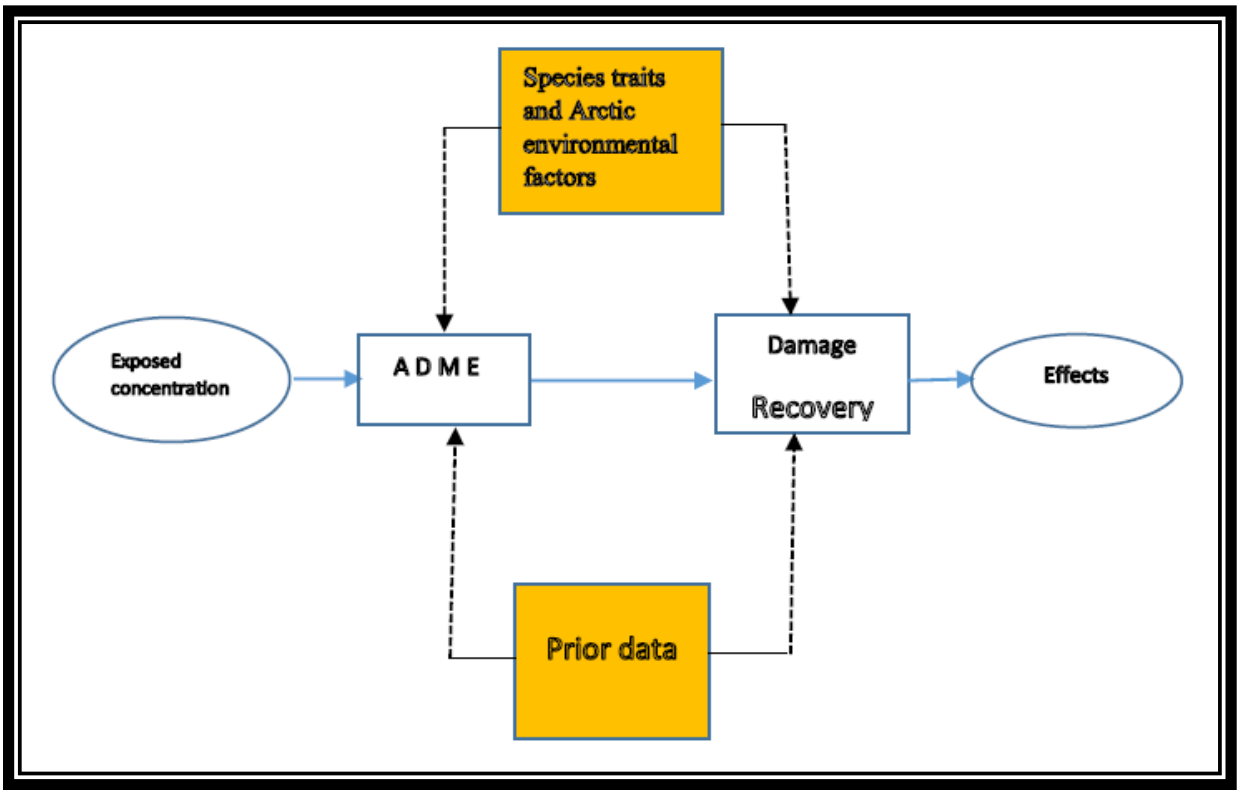


Figure 7: Overview of the proposed mechanism based Arctic aquatic risk model.

Although some researchers proposed a species trait influenced TK model, no such model has been developed. Further, species traits and environmental factors affecting TD parameters have not been investigated in detail, nor models developed. Factors affecting toxicodynamics parameters, such as damage and recovery, in the cellular level should be combined along with the factors affecting the toxicokinetics in the Arctic animals and prior data (as shown in Figure 7) to estimate effects in aquatic organisms. Such an approach paves the way for a comprehensive risk assessment model for Arctic aquatic animals.

Chapter 3: Arctic marine fish 'biotransformation toxicity' model for ecological risk assessment

3.1 Introduction

Marine transport and petroleum industry activities in Arctic regions, such as the Barents, Beaufort and Chukchi Seas, can imperil marine organisms that are exposed to oil spills. A key knowledge gap in Arctic Environmental Risk Assessment (ERA) is the limited 'No Observed Effect Concentrations'(NOEC) data for various Arctic aquatic animals. Establishing such NOEC exposure standards is difficult because of the limited available experimental toxicity data for the Arctic marine organisms. The lack of data is usually addressed by using temperate species data as surrogates to fill the gaps in toxicity data of their Arctic counterparts, although such a practice is debatable. Studies such as Bakke et al. (2016), Jensen (2011), Andersen et al. (2015), Jonsson et al. (2010) and Nahrgang et al. (2009, 2010a, 2010b, 2016) have discussed in detail how Arctic marine organisms are especially physiologically different than their counterparts elsewhere. For example, polar cod has different routes of xenobiotic biotransformation and excretion than Atlantic cod, thus influencing polar cods' toxicity properties. Xenobiotics in teleost fishes (which is over 97% of the fishes) are primarily excreted via urine and bile. Polar fishes, such as the polar cod, lack glomerular kidneys and therefore xenobiotic excretion occurs not by the urinary tract, but the hepato-biliary tract only (Nahrgang et al. 2010b, Christiansen et al. 1996, Andersen et al. 2015). Toxic effects variation in po[2]lar cod could also be attributed to other physiological conditions,

[2] This chapter is published as a research paper in Marine Pollution Bulletin. DOI: 10.1016/j.marpolbul.2019.03.039.

such as the drastically varying body lipid content and rate of metabolism. The geophysical factors, such as the presence of ice and other environmental factors, also contribute to delayed, increased, or mitigated responses to the xenobiotic removal and toxicity (Hallanger et al. 2011). Therefore,

when modeling the toxicity of Arctic marine species, all the physiological, geophysical, and environmental factors in play should be incorporated in an ecotoxicological model. A framework for such traits-based modeling was proposed by Rubach et al. (2012), where a link between traits to various physiochemical and biochemical processes was proposed as an answer for mechanistic ecotoxicity models. Traits-based approach is especially helpful when using the effect modeling in Environmental Risk Assessment (ERA).

The current study develops on the traits-based approach by identifying Arctic environmental, geophysical, and physiological factors affecting the Toxicokinetic (TK) and Toxicodynamic (TD) parameters at cellular level in polar cod, a keystone species in the Arctic region (Bakke et al. 2016, Jonsson et al. 2010, Werner 2006). The most frequently cited mechanistic ecotoxicity models are Toxicokinetic Toxicodynamic (TKTD) models. While TK deals with the quantification of toxicant distribution in various tissues and organs in an organism, TD model deals with the quantification of effects induced in an organism. Although TK and TD models are touted as the mechanistic models, they are also limited by the data from toxicity assays and could be termed as semi-mechanistic models as they do not completely encompass the knowledge of toxicity mechanisms in the TD tier of the modeling. TD parameters such as mortality percentile and maximum threshold distributions are based on statistics from observing the test subjects in toxicity assays. This study attempts to graphically represent the TK and TD processes in polar cod using a Bayesian Belief Network (BBN). Further, the BBN links various environmental, physiological and geophysical factors that are specific to the Arctic region to the TK and TD processes. Understanding the

mechanism of both toxicity and defense in cellular level is integral to mechanistic ecotoxicity modeling. Arctic marine fish toxicity and their recolonization potential have traditionally been assessed using mortality endpoints and reproduction endpoints derived from toxicity assays. However, the field of ecotoxicology is increasingly favoring use of mechanistic based modeling rather than the statistical analysis and toxicity assay approaches used in prior studies (Baas et al. 2015, Fahd et al. 2017). Fahd et al. (2017) offer a detailed explanation on various statistical and mechanistic methods in ecotoxicology. The current understanding of the mechanism of toxicity of PAH is very recent and, in cases such as polar cod, limited. Additionally, the role of seasonal and geographic variations in the defense mechanism of the polar cod is far less studied. A recent concept in the toxicity mechanism literature is of adverse outcome pathways (AOP) by Ankley et al (2010). AOP presents the chain of events after the initiation of the biological activity at the molecular level. Refer to Ankley et al. (2010), Escher et al. (2017), and Knapen et al. (2015) for details on the AOP concept. The concept of AOP merely mentions the possibility of the mechanism and provides a biological context to the toxicity assays, yet there is no explanation regarding quantification of toxicity. Ideally, a mechanistic TD model must consider all the mechanisms in cytotoxicity and adopt a trait-based approach combining both empirical data and predictive models. An organism has both mechanism of toxicity and defense in its cell (Turcotte 2008). In fish, biomarkers such as Ethoxyresorufin-O-deethylase (EROD) and Glutathione S-transferase (GST) measure the metabolic activity and changes in cell antioxidant defenses based on the exposure to contaminants and can be effectively used for aquatic risk monitoring and assessment (Strobel et al.2015). Thus, effort is made to link the biomarkers data to the process based TKTD model and incorporate this in the BBN model.

Modeling any real-life concept into a model is a challenge, and the difficulties are multiplied further in modeling of any living organisms' operations. BBNs have long been used for modeling in ecological issues, by employing their capacity to integrate expert knowledge and empirical data. They are the graphical models describing probabilistic relationships between a set of variables (Helle et al. 2011). BBNs are especially effective for modeling situations that depend on cause and effect, where some information is known and uncertainty exists in other incoming data, for example, the toxicity mechanism in fish (Liu et al. 2015, McDonald et al. 2015). The goal of the chapter is predicting the probability of cell damage in polar cod from PAH biotransformation, which could be subsequently used to determine the probability of organism failure and fish community failure in future studies.

3.2 Context of the study and baseline data

The region of interest is the Arctic Ocean and adjacent seas. The Arctic region is characterized by the presence of sea ice, harsh weather conditions, and an extreme light regime. Arctic region also has non-complex food trophic levels, with polar cod serving as a keystone species. Polar cod has both abundance and circumpolar distribution in the Arctic region and is a good choice as a monitoring species for risk assessment associated with oil spills. Additionally, polar cod holds strong association to lower trophic and higher trophic species, and to under ice and ice edge in the ocean. Polar cod feeds on the lower trophic species such as amphipods and copepods, and in turn is preyed upon by higher trophic species such as birds, seals, and polar bear. Due to this unique positioning in the food web, polar cod is selected as the species of concern for this study. The concentration of PAH spill considered in this study varies from a low of <30µg/L to a high of 60-100µg/L. Nahrgang et al. (2010) conducted toxicity assays using total PAH concentrations within

the range of 30 – 60 µg/L and documented the elicited biomarker responses. This study used the same range of total PAH exposure and the subsequent biomarker response data to develop the BN model. Further, the range of total PAH concentrations considered in this study reflect the spill scenarios of 15000-40000 tonnes of crude oil spill, assuming 3.9% weight of total PAH. Lower spill concentrations were considered to represent an environmentally relevant spill concentration. Further, smothering causes mortality of marine fish in large spills, while in lower concentrations the biotransformation processes come into effect. The sea ice data is from the Fram Strait and Kongsfjord regions as presented by Werner (2006), and Nahrgang et al. (2010b) respectively. The seasons of Winter, Summer, and Autumn are defined from December to June, July to September, and October to November, respectively. In the Arctic region Spring season follows Winter season, however there is no biotransformation/metabolism biomarker baseline data for polar cod in Spring. Therefore, the seasons in this study were categorized as shown in Table *2*. The details of the seasonal variation in ice thickness data, ocean surface salinity, algal bloom, and metabolic activity in fish from the Fram Strait and Kongsfjord regions are also shown in Table 2. These data form the baseline for the ecotoxicity model.

Table 2: Baseline data of environmental factors affecting polar cod toxicity

Season	**Ice thickness (m)**	**Salinity - Ocean surface (psu)**	**Chl a (in ice) (mg/m^2)**	**Baseline Phase I (EROD) activity (pmol/min/mg)**	**Baseline Phase II (GST) activity (nmol/min/mg)**
Winter	0.8 – 3.5	32.4 – 34.6	0.12 – 2.63	3	500
Summer	1.4 – 3.4	16.7 – 32.4	0.13 – 2.94	13	200
Autumn	1.6 – 3.3	31.5 – 32.4	0.25 – 18.40	3	350

Source: Werner (2006) and Nahrgang et al. (2010)

3.3 Methodology to develop the ecotoxicity model

The workflow of the methodology used to develop the toxicity model is shown in Figure 8. The ecotoxicity model alludes to a novel approach, where the environmental and geophysical factors of the Arctic, and the physiology of the polar cod contribute to the toxicity model. Each of the steps mentioned in Figure 8, leading to the development of a Bayesian Belief Network (shown in Figure 9) and probability of cell toxicity, are discussed further in detail.

The model is a graphical representation of the TK and TD models in Arctic scenario. The concentration of xenobiotic in the target organ in polar cod and the elicited response in the target organ is assessed using suitable biomarkers. The first step in the methodology is to determine the toxicity and defense mechanism in polar cod and how they interact with the TK and TD processes. Subsequently, suitable biomarkers representing a measure of metabolic activity and antioxidant defense activity in polar cod are incorporated in the toxicity model. The second step in developing the BBN is to identify the environmental, physiological, and geophysical factors affecting the TK and TD processes and to determine the baseline of those factors and pertinent biomarkers. The third step in the methodology is to collate data from the prior steps and use cause and effect relationships to develop an ecotoxicity model using the Bayesian Belief Network (BBN) (Figure 9). The nodes in Figure 9 represent discretized random variable and the arcs represent the probabilistic dependencies between the variables. The conditional probability tables (CPTs) of a BBN reveal the marginal probability of a variable with respect to other dependent variables for that node. All the nodes used in the model and the CPTs assigned to the nodes in the model are presented in the supplementary material.

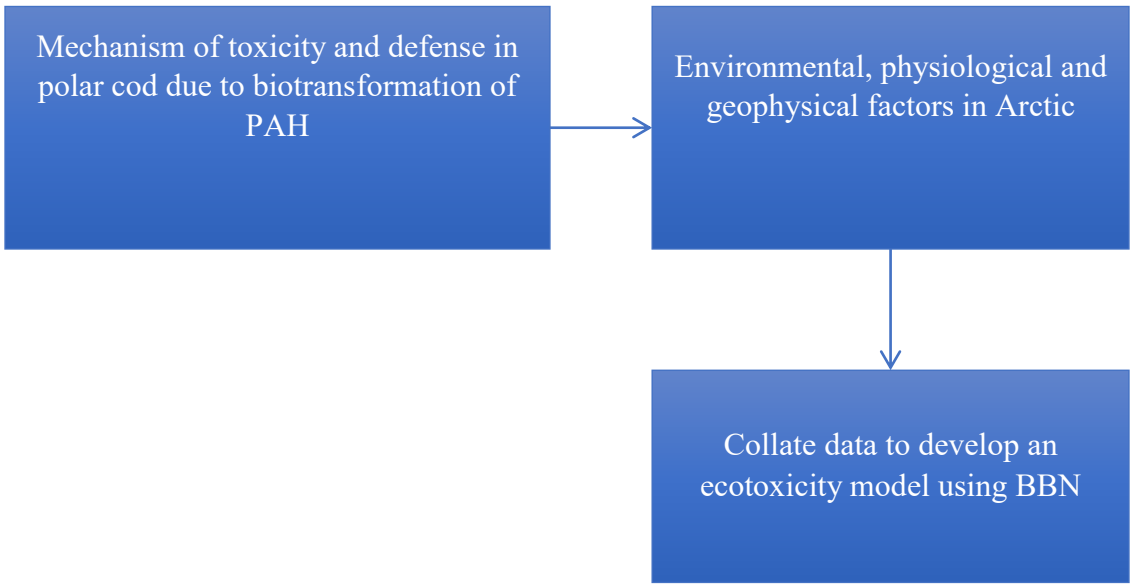


Figure 8: Workflow of the methodology for polar cod ecotoxicity model.

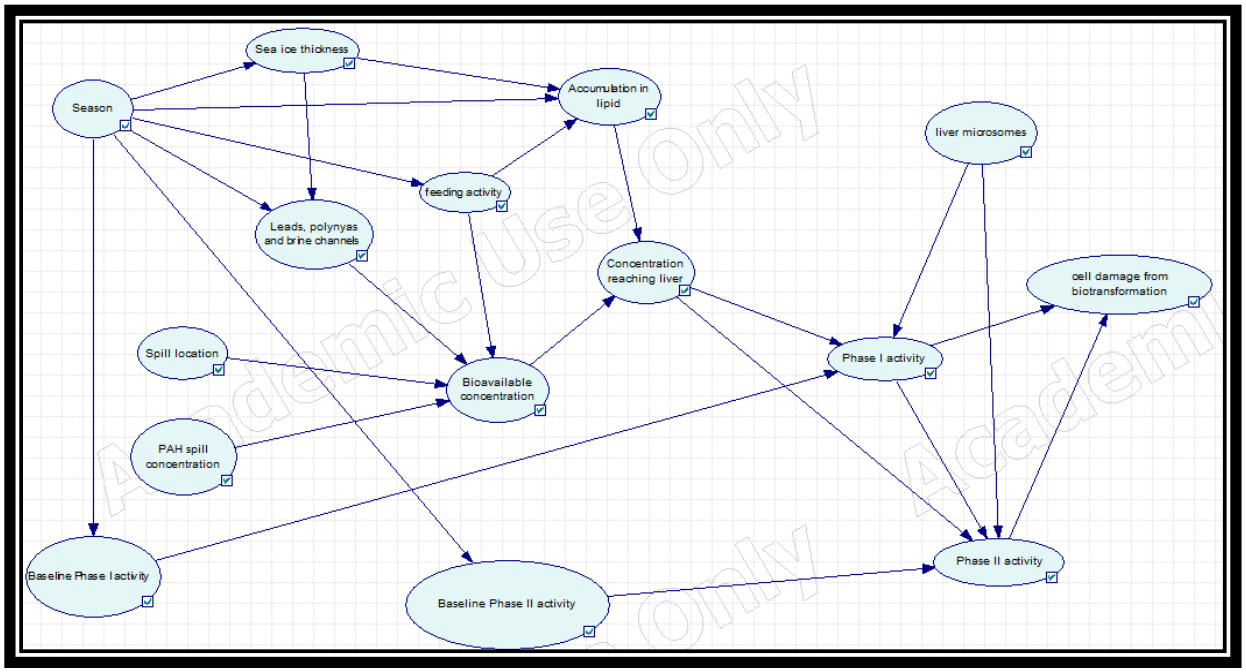


Figure 9: Relationship between various nodes in the BBN.

The relationship of polar cod to variables in the Arctic region is explored to identify and develop a cause-effect based modeling of toxicity from possible PAH exposure. A hypothetical exposure scenario is used to develop and validate the Bayesian risk assessment model for polar cod. Four exposure scenarios in three different seasons are considered as follows:

1. Oil spill on the pack ice in winter.
2. Oil spill under the pack ice in winter.
3. Oil spill on thin ice in summer.
4. Oil spill on thin ice and water column in autumn.

3.3.1 Mechanism of toxicity and defense in polar cod due to biotransformation of PAH

Death occurs due to compromise of the cell structure, i.e. cytotoxicity. Studies have identified that double breaks in DNA strands also contribute to the cell membrane compromise, however, such an effect is not considered in the BBN model yet. A cell inherently has the ability to repair itself, however, when a certain threshold number of cells in an organ fail, the organ fails. Mechanism of cytotoxicity in fish can be from lipid peroxidation due to action of PAH, oxidative stress due to biotransformation of PAH and its metabolites, and phototoxicity due to breaking of the PAH due to UV actions (Dorne 2010, Turcotte 2008, Lushchak 2011). This chapter focuses on PAH biotransformation and develops a causal relationship to various factors affecting PAH in lipid accumulation and metabolism. The factors include background metabolic activity, feeding activity, and spill location with respect to the sea ice. Biotransformation in polar cod is a two-step process where the enzymes react with the lipophilic xenobiotic, such as PAH, and convert it to water-soluble metabolites suitable for elimination. The two steps in biotransformation of a xenobiotic are termed phase I and Phase II processes. Phase I facilitates adding polar atoms to

xenobiotic compounds by one or more of the processes of oxidation, reduction, and hydrolysis, thereby enhancing the toxicants' water solubility. Various intermediary and final Metabolites generated during the biotransformation process depend on many conditions, and these metabolites contribute to any one of the possible outcomes: toxification, inert presence in the body, or safe removal. Thus, generation of metabolites enabling xenobiotic detoxification is only one of the possible three outcomes of biotransformation. The enzymes that are active in phase I for PAH biotransformation are the Cytochrome P 450 group of enzymes (Varanasi 1989). PAH are specifically biotransformed by the CYP1 group of enzymes in the Cytochrome P 450 group (Lacoste et al., 2013). During phase I of the biotransformation process, free radical oxygen ions or oxides are formed. The first step in oxidation of PAH is by insertion of an oxygen atom to form arene oxide. This is mediated by the presence of the cytochrome P-450 system of enzymes. Since production of oxygen atoms is concomitant to many natural processes, including biotransformation, they must be balanced to negate any negative impacts. Phase II reactions further enhance the water solubility of the metabolites produced during the phase I step. The Phase II conjugation reactions with gluthathione are facilitated by glutathione-S-transferase (GST). This enzymic action provides antioxidant defense in the organism. Some of the metabolites resulting from the phase I process cause cytotoxicity unless they undergo the phase II process. Cell death due to biotransformation results when the toxic metabolites resulting from phase I process exceeds the conjugating capacity of the organism via phase II process (Figure 10) (Banni et al. 2009). Mortality due to biotransformation of PAH is dependent on the actions of enzymes and is caused when cell deaths in an organism breach a threshold. The metabolites produced in the biotransformation process determine the toxic nature and effects in the organism as the process of detoxification or toxification is determined by balance of actions of Phase I and Phase II enzymes

as shown in Figure 10. Although Phase I biotransformation reactions include oxidation, reduction and hydrolysis, an example of only oxidation is used in Figure 10 to demonstrate the biotransformation toxicity mechanism framework. The biomarkers used to quantify the metabolites concentration in fish tissues are Ethoxyresurufine-O-deethylase activity (EROD) and glutathione-S-transferase (GST) assays. EROD measures the Phase I metabolite activity while GST measures the Phase II conjugation activity in the sample.

The majority of the biotransformation of xenobiotic is accomplished in the liver of polar fish. This is also true for many other fishes and other organisms. Many studies have stated that about 80% of biotransformation in fish occurs in the liver (Banni et al. 2009). While the polar fishes, such as cod, do not have xenibiotic elimination via kidneys, the gills serve as another source of toxicant removal. Xenobiotic removal via gills is not considered in this toxicity model. Limited and recent studies, such as Vieweg et al. (2017), Tomy et al. (2014), and Nahrgang et al. (2009, 2010a, 2010b) have conducted experiments on ingestion and biotransformation of crude oil in polar cod, while many earlier studies (Banni et al. 2009) have studied the biotransformation biomarkers in Atlantic cod. CYP1A is a membrane bound enzyme is predominantly present in the smooth endoplasmic reticulum (SER) of microsomes in the liver cells but are also present in lower densities in other tissues such as gills (Jensen 2014). Many studies have developed cell models and identified that about 16 – 20 % of the surface area of a cell is occupied by SER and the antioxidant enzyme is estimated to be 2% of the cytosolic protein in fish (Moore 1992). This information is used to quantify the various states of the node 'Liver microsomes' as shown in Table S1 of the supplementary material. The CPTs for the relevant nodes of 'Liver microsomes', 'Phase I activity', and 'Phase II activity' are presented in Tables S6-S8 of the supplementary material. Nodes in the

BBN for biotransformation toxicity associated with each step in the methodology are presented in Table 3.

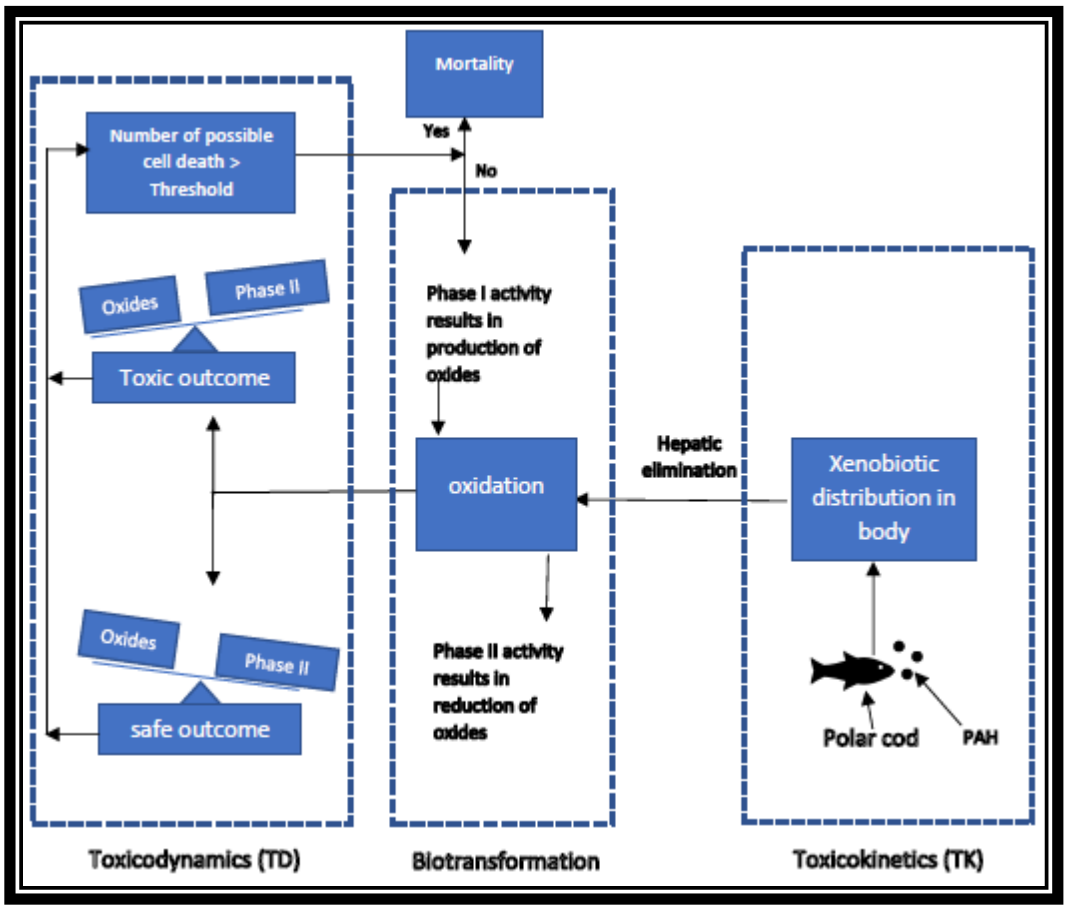


Figure 10: Mechanism of toxicity from biotransformation

Table 3: Nodes of BN for biotransformation toxicity

Nodes	**Mechanism of toxicity and defense in polar cod due to biotransformation of PAH**	**Environmental, physiological and geophysical factors in the Arctic**
Season		
Spill location		
PAH spill concentration		
Sea ice thickness		
Leads, polynyas and brine channels		
Feeding activity		
Salinity changes from ice formation and melting		
Accumulation in lipid		
Bioavailable concentration		
Concentration reaching liver		

Percentage of liver microsomal protein	[redacted]	
Phase I activity	[redacted]	
Phase II activity	[redacted]	

3.3.2 Environmental, physiological, and geophysical factors in Arctic

All the environmental and geophysical factors are analyzed and quantitatively tied to variation in enzymic activity using conditional dependencies in the Bayesian model. Changes in the Phase I and Phase II activities are evaluated based on the EROD and GST biomarkers activity from various experiments on polar cod (Nahrgang et al. 2009, 2010a, 2010b). The environmental and geophysical factors in the Arctic region and the physiology specific to marine organisms (Refer Figure 8) is further discussed in the following sections.

3.3.2.1 Transport of spilled oil in dynamic sea ice

Transport of spilled oil from the top of thick ice to the water column, and transport of spilled oil from the top of thin first year ice to the water column are the scenarios that are considered in this study. Also considered is the scenario of spilled oil under pack ice. The understanding of the oil spill exposure should be associated with the transport phenomena of the oil and ice formation processes. Since juvenile cod live under the ice pack, the phenomenon that drives the oil from the top of the ice to under the ice is important to consider. The ice thickness is a dynamic variable. The oil spill on top can reach the ocean surface under the ice through brine channels and cracks. The processes that could impact the under-ice exposure of PAH are melting of the ice, ice

formation and brine channel formation, and absorption and transport of PAH using naturally occurring cracks and crevices in the ice pack.

3.3.2.2 Polar cod and sea ice association

Polar cod is an appropriate monitoring organism for oil pollution in the Arctic (Jonsson et al. 2010). It is the most abundant fish residing under the Arctic sea pack ice (David et al. 2016). The pack ice serves as habitat for juvenile cod, i.e., up to 2 years. It has been estimated that the distribution of such cod is about 5000 individuals per square km. They are observed below the sea ice in autumn and winter. In the Arctic regions where the sea ice fully melts in late summer and autumn, they become part of the pelagic fish stock. The single most important factor impacting the TK and TD is presence of sea ice. The role of sea ice and its seasonal variations are significant in metabolism and phototoxicity. Polar cod in large numbers feed under the sea ice for algae and shrimp-like organisms. Many amphipods and copepods survive under the sea ice, thus providing opportunity for food to the polar cod. Under the sea ice and water interface also provides excellent breeding spots for the polar cod. The availability of food varies according to the seasonal variation of the sea ice thickness. In winter, when ice is the thickest, food availability is low. As the thickness of ice decreases, more sunlight penetrates, increasing the algal bloom and subsequently increasing the opportunity for food availability for polar cod. Under medium and thin sea ice, i.e. autumn and summer, the polar cod thrive with increased availability of food and increased metabolic activity (Werner 2006). Metabolic activity is the highest in autumn, then followed by summer and winter respectively. The variation in polar cod baseline metabolic activity is in accordance with the food availability in the region. Ice melt and increased open waters in Summer and Autumn pave way for increased availability of chlorophyll a (Chl a) which is essential for growth of algae (Berge et

al. 2015). The copepods and amphipods in the Arctic region thrive on the algal bloom and are preyed upon by polar cod. Thus, feeding activity in polar cod corresponds with the increased Chl a in the ice and ocean surface (Table *2*).

The increased feeding activity results in increased possibility of PAH accumulation in lipids and increased metabolism of PAH through dietary exposure. The polar cod spawn in early winter; therefore, the body lipid reserve accumulated is utilized during the winter season. This could result in increased availability of lipid stored PAH in metabolism process.

3.3.2.3 Changing salinity

The seasonal variation of ice causes changes in the salinity of the water near the surface. In early winter when the ice forms, it pushes brine out into the seawater via brine channels, thereby, increasing salinity of the water. When the pack ice starts melting in summer and autumn, it lowers the salinity of the water at the interface. In Autumn, the salinity levels are above summer levels, but gradually increase due to the fresh ice formation. Salinity is a critical abiotic factor affecting the biological processes such as lipid metabolism and oxidative stress (Lee et al. 2017). Changes in salinity could put the juvenile polar cod under stress and thereby cause change in metabolic activity. The impact of decreasing salinity on enzyme activity is not well understood in polar cod. Lee et al. (2017) studied the effects of changing salinity on oxidative stress and lipid metabolism in a rotifer. The study observed significant increase in GST activity for increasing salinity. Apart from the impact of oxidative stress, lipid content can also be affected due to changes in salinity. Kheriji et al. (2003) observed that fish acclimatized to lower salinity contained 55% less fatty acid than those acclimatized to seawater at the same temperature. However, no study has comprehensively studied the oxidative stress and lipid composition variation due to salinity and temperature in polar cod. The scope of this chapter deals with oxidative stress due to

biotransformation of PAH. The PAH stored in lipid could lead to toxicity due to lipid peroxidation, is discussed in the next chapter. However, the PAH stored in lipids could attenuate the concentration of biotransformed PAH and this phenomenon is considered in this chapter.

3.3.2.4 Extreme light regime and seasonal temperature variations

The Arctic regions face an extreme light regime of extended hours of sunlight or darkness. Prolonged periods of sunlight expose the marine organisms to ultraviolet (UV) rays, UVA and UVB. Juvenile polar cod are especially susceptible to this exposure when they ingest the PAH. The interaction of the UV rays with the PAH in juvenile polar cod lead to increased instances of toxicity (Willis et al. 2014). The PAH metabolites generated due to UV action and their toxicity is out of the scope of this chapter. The under ice and water surface interface is a stable temperature environment owing to the insulating effect of the ice cover. Therefore, it can be assumed that the impact of changing temperature regimes is insignificant.

The CPTs for the nodes pertinent to environmental, physiological, and geophysical factors are shown in Tables S2-S11 of the supplementary material.

3.3.2.5 Bayesian Belief Network for polar cod toxicity

Bayesian belief network (BBN) is a tool for modeling complex mechanisms such as toxicity in an organism. BNN are directed acyclic graphs, where available quantitative knowledge is used to generate posterior probability. A systems approach is presented in this study to understand the toxicokinetics and toxicodynamics of PAH in Arctic fish, and the toxicity model is presented as a causality-based model. Causal relationships linking various relationships between TK, TD,

biotransformation, and the multistep pathway in TK TD are presented as a belief-based network in Figure 11.

The primary input nodes or parent nodes of the BBN are ‘Season’, ‘Spill location’, and ‘PAH spill concentration’. All remaining nodes are related to parent nodes via conditional dependencies. The season in the Arctic greatly influences the sea ice thickness and the opening in the ice pack. Season also influences the feeding activity and metabolic rates in the fish. It was observed by Nahrgang et al. (2010b) that the seasonal fluctuations exist in the baseline Phase I and Phase II activity. As is evident from Table *2*, the baseline Phase I activity is highest in summer, while baseline Phase II activity is highest in winter, followed by autumn. Spill location and spill concentration influence the ‘Bioavailable concentration’ node, along with other nodes such as ‘Feeding activity’ and ‘Leads, polynyas and brine channels’. The ‘Bioavailable concentration’ node positively affects the ‘Concentration reaching liver’ node, while ‘Accumulation in lipid’ inversely affects the ‘Concentration reaching liver’. ‘Phase I activity’ node is dependent on ‘Concentration reaching liver’, ‘Baseline Phase I activity’, and ‘liver microsomes’ present for Phase I. The ‘Phase I activity’ node influences ‘Phase II activity’ node, along with ‘Baseline Phase II activity’, ‘liver microsomes’ for metabolism and ‘Concentration reaching liver’. All the nodes in BBN are presented in Table 3.

Conditional dependencies in the model are presented in the supplemental material. As an example, conditional dependencies of the node ‘Concentration reaching liver’ are presented in Table 4.

Table 4: Conditional dependencies of 'Concentration reaching liver' node

Bioavailable concentration	**Low**			**Medium**			**High**		
Accumulation in lipid	Low	Medium	High	Low	Medium	High	Low	Medium	High
Low	0.9	0.9	0.95	0.15	0.3	0.35	0	0	0
Medium	0.1	0.1	0.05	0.85	0.7	0.65	0.1	0.3	0.35
High	0	0	0	0	0	0	0.9	0.7	0.65

The concentration reaching the liver is directly dependent on the bioavailable concentration and inversely dependent on the accumulation of PAH in hepatic lipids.

3.4 Results and discussion

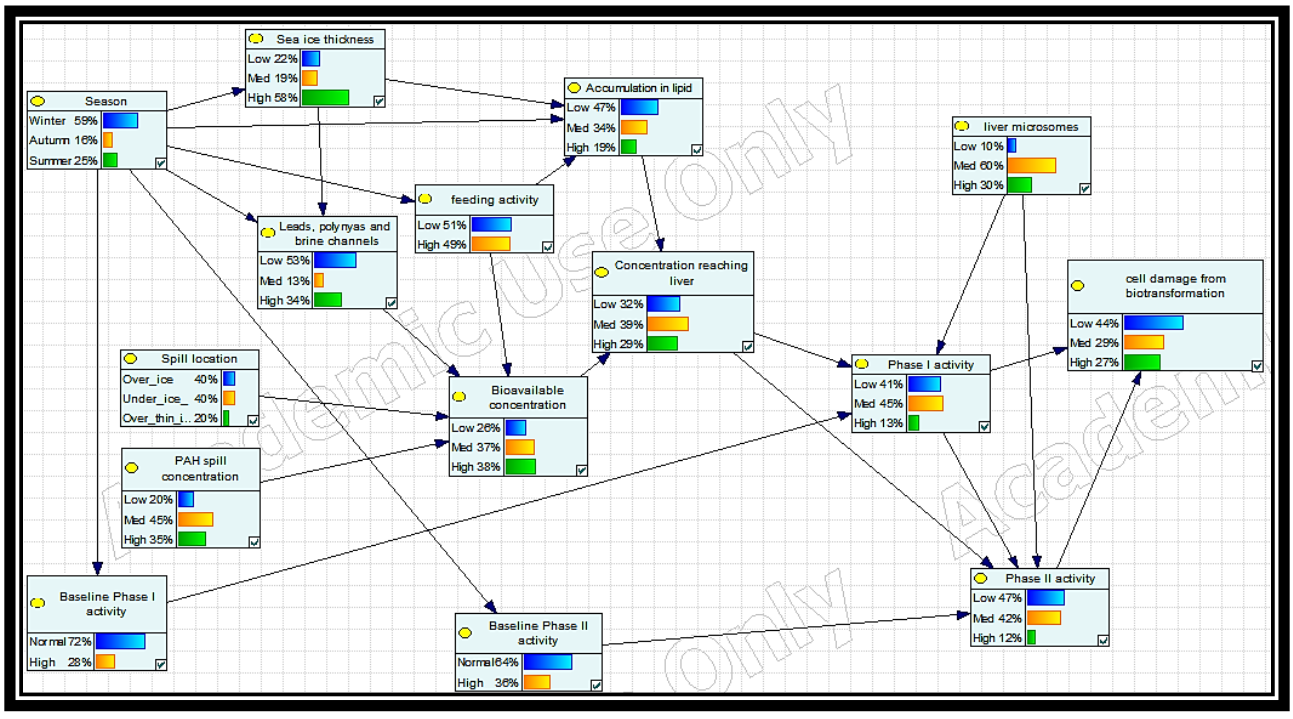


Figure 11: Bayesian network-based biotransformation toxicity model

The current model is a first analysis of causal dependencies starting from the point of oil spill, to seasonal variation in spill attenuation and bioavailability, and then integrating the bioavailable concentration with metabolic activity, and finally to toxicity.

3.4.1 Scenario 1: Oil spill on winter pack ice

During winter in the Fram strait and Svalbard region, the ice pack depth varies from 2.5 to 3.5 m. The leads and openings in the pack ice are at their minimums in winter season and the surface area openings in ice pack are under 10%. Feeding activity is low during this time of the year. During early winter, polar cod accumulates lipids and uses the lipids through the winter spawning activity. Presence of increased lipids in the fish could attract lipophilic chemicals like PAH, which could be stored benignly in the lipids until the lipids are metabolized at which point, the PAH are metabolized by phase I and phase II enzymes. The input parameters for the season node are winter, microsomal concentration and PAH spill concentration node state is medium.

Spill on ice: Assuming a spill is on the pack ice, the results of cell death as obtained from the BN model are reported in Table *5*. The probability is high, about 57%, for low impact in cell deaths. The most sensitive nodes are Phase I enzyme activity, presence of leads, polynyas and brine channels, followed respectively by nodes such as bioavailable concentration, concentration reaching liver, and liver microsomal percentage. The important environmental processes contributing to the possible toxicity are the possibilities of leads and brine channel formations, which can increase the possibility of PAH traveling from the ice surface to the water. The background metabolic activity is very low in winter.

The biological processes contributing to lessen the impact of cell death are less presence of microsomal fraction in liver and less baseline Phase I activity. Any of the states in a given node can be set as an evidence, at which point, such a node is termed an evidence node. Choosing 'liver microsomes' as an evidence node and varying evidence from low, medium and high states, it can be observed that the probability of cell damage increases respectively. This trend was observed in all seasons as shown in Table 6.

Spill under the pack ice: Assume a spill under the pack ice in winter, with conditions similar as mentioned earlier. Presence of a spill under the pack ice is an important and more plausible scenario than the oil spill being present on the ice. Ship traversing through the iced waters could result in spilled oil moving and accumulating under the ice. The underside of the pack ice is rough with many undulations. The spilled oil is trapped in these undulations. Ocean surface water movements further spreads the spill under the pack ice. A major contributor for surface water movement is wave action, however, the presence of ice dampens the wave action. Although ocean currents also cause spilled oil movement, such a case is not considered in this study. The presence of leads and openings play different roles in PAH exposure to polar cod when the spill is over the ice pack as opposed to under the pack ice. The openings in the pack ice provide conditions for mixing of oil and water, local turbulence in the water-ice interface causing oil in which can increase the exposure of the resident fish to oil. Such movement and activity within the spill oil can cause increased dissolution in water and subsequently increased exposure to the polar cod. Under the given assumption of PAH concentration, the probability of exposure of the polar cod to spilled oil is higher in summer and autumn than in winter. For the same amount of exposure to PAH, concentration reaching polar cod liver in winter is higher than other seasons. This trend is attributed to the feeding behavior of the polar cod. In winter, the lipid accumulation is minimal,

hence the probability of higher concentration of PAH reaching the liver. In summer and autumn, the feeding activity is far higher and a lot of energy is stored in form of body lipids. However, the higher lipid content also attracts the lipophilic PAH, which then is stored in lipids. This can decrease the concentration of PAH reaching liver as is evident from the Table *7* below. However, the probability of spill exposure and subsequent bioavailability of PAH in polar cod will be higher than when the spill is on the ice pack.

3.4.2 Scenario 2: Oil spill on thin ice in winter

Pack ice is prominent in Winter in the Arctic region. However, such ice also could develop small and large leads. Thin Sea ice starts forming in these openings between the pack ice. When oil spill is trapped between the pack ice chunks this scenario may arise. The input parameters selected in this scenario are winter, medium spill concentration, and medium presence of liver microsomes. In the event of the presence of thin ice in winter and a spill on the thin ice, the probability of cell damage is 44% low, 30% medium and 16% high. As the ice cover thickness decreases, the probability of cell damage increases owing to more concentration reaching the liver and thus more possibility of biotransformation of PAH. The most sensitivity node for this scenario is 'Phase I activity' in liver microsomes.

3.3.3 Scenario 3: Oil spills on thin ice in summer

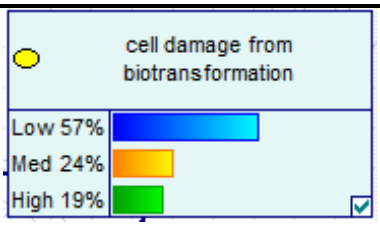
Feeding activity and openings in ice, and baseline metabolite activity. In this study, Summer is considered from July to September and the ice thickness varies from 1.4m to 3.4m. During these months, the feeding activity is on the rise from its level in Winter. Sea ice is present as thin ice in this scenario and the input parameters for this scenario are medium spill concentration, and medium presence of microsomes in liver. The most sensitive node for this scenario is also the 'Phase I activity'. The probability of cell damage is 38%, 33%, and 29% for low, medium, and high probability states. The most sensitive node as in previous cases is the 'Phase I activity' node.

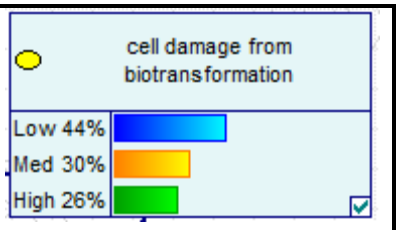
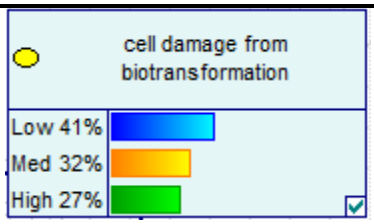
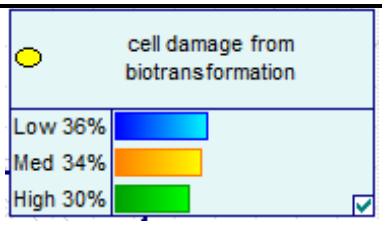
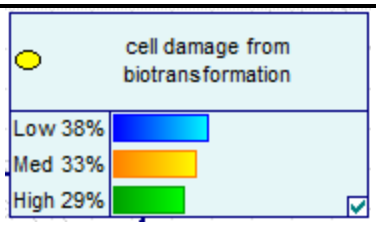
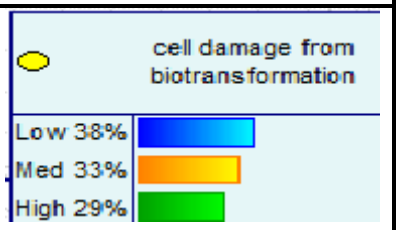
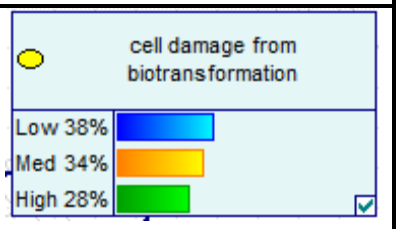
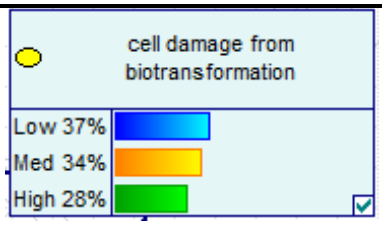
The commencing of the biotransformation process starts with phase I activity. The baseline phase I activity is already higher in Summer season, therefore limited PAH could be biotransformed. The higher baseline phase I activity can ultimately contribute to lowering cell damage than when lower baseline activity be present.

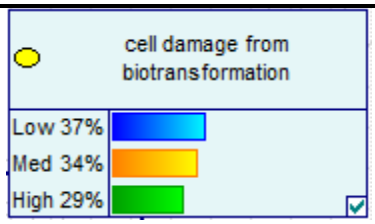
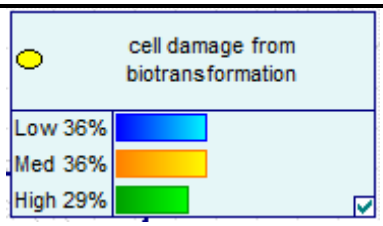
3.3.4 Scenario 4: Autumn and spill over thin ice

The autumn season has the highest probability for low sea ice presence and the sea ice thickness could vary from 0.8m to 3.3m. Consider medium spill concentration and medium liver microsome presence as the input parameters for this scenario. The probability for cell damage is 37%, 34%, and 29% for low, medium and high probability states. Amongst the three seasons considered, Autumn season presents highest probability for cell damage. The factors that contribute to escalation of the cell death are increased spill concentration, low sea ice presence, lower baseline phase I activity, lower lipid accumulation, and higher baseline phase II activity. The most significant node from the model that influences cell death is the percentage of liver microsomes. This could be understood by the fact that lower presence of microsomal tissues presents lower opportunity for metabolism of PAH and higher probability for lipid storage of PAH.

Table 5: Results of cell damage when considering medium 'liver microsomes'

Season	**Spill location**	**Spill concentration**	**Cell damage**
Winter	On thick ice	Medium	

	On thin ice	Medium	
	On thick ice	High	
	On thin ice	High	
Summer	On thick ice	Medium	
	On thin ice	Medium	
	On thick ice	High	
	On thin ice	High	

Autumn	On thin ice	Medium	
	On thin ice	High	

The presence of microsomal tissue is consistently observed as a sensitive factor leading to cell damage. Therefore, the worst-case scenario of a spill on thin ice in Autumn, with medium spill concentration selected for further investigation and microsomal tissue levels were changed to see how it impacts the probability of cell damage. The results of varying the states of the microsomal tissue node are presented in Table 6. The presence of sea ice plays an important part in temporarily mitigating the toxicity to organisms, as is evident from Table 5. The probability of cell damage is lower when the spill is on thick ice than on thin ice in all seasons, although this trend is more evident in winter season. This is owing to the higher probability of pack ice presence with minimal leads, brine channels, and openings in the ice surface. Alternatively, in summer and autumn the openings in sea ice are maximum, thereby causing higher exposure probability. The baseline levels of phase II GST activity are higher in winter. The balance of metabolites warrants equal or greater production of GST conjugates. Since the baseline is already higher, the ability of the organism to conjugate could be limited if the concentration of exposure is high. The concentration used in this model was at lower environmental relatable concentrations, thus the baseline Phase I or Phase II levels did not play a significant role in the model.

Table 6: Autumn scenario with changing 'liver microsome' states

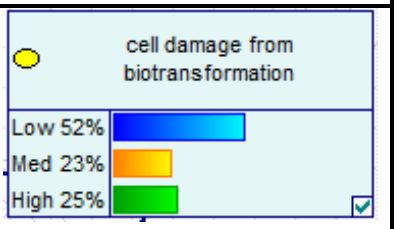
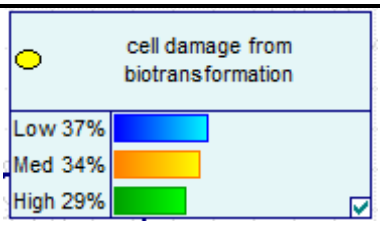
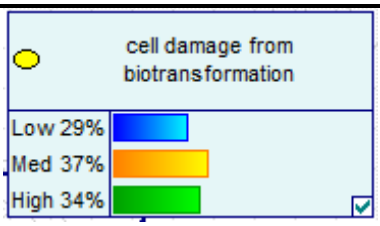
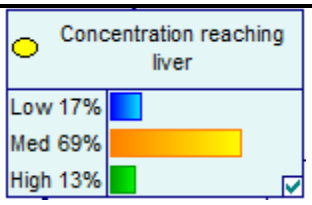
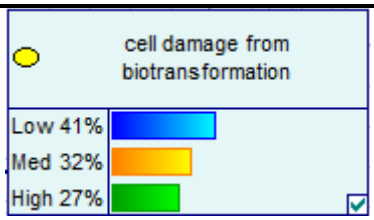
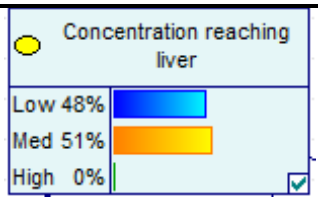
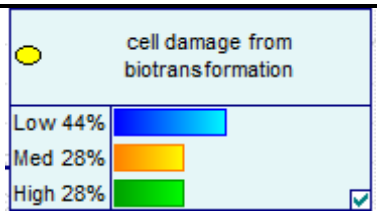
Season	Spill location	Spill concentration	Liver microsome	
Autumn	Over thin ice	Medium	Low	
			Medium	
			High	

Table 7: Spill under pack ice

	Concentration in liver	Biotransformation cell damage
Winter		
Summer		

Comparing various scenarios of spill concentration and spill location in varying seasonal conditions, it could be ascertained that the scenario with thin ice, many leads and openings in Autumn could be termed as a worst-case scenario (Figure 12). The presence of a high percentage of microsomal tissues also impacts the cell damage probability as seen from Table 5 and Table *6*. This could be attributed to higher presence of CYP1A enzymes in such tissues. Table 7 represents the results for spill under the ice scenario. The probability of PAH concentration reaching liver is higher is winter than in summer, as evident from the Table 7. This could be understood to be the effect of lipid accumulation phenomenon of polar cod in summer and autumn. The subsequent cell damage is slightly higher in winter season.

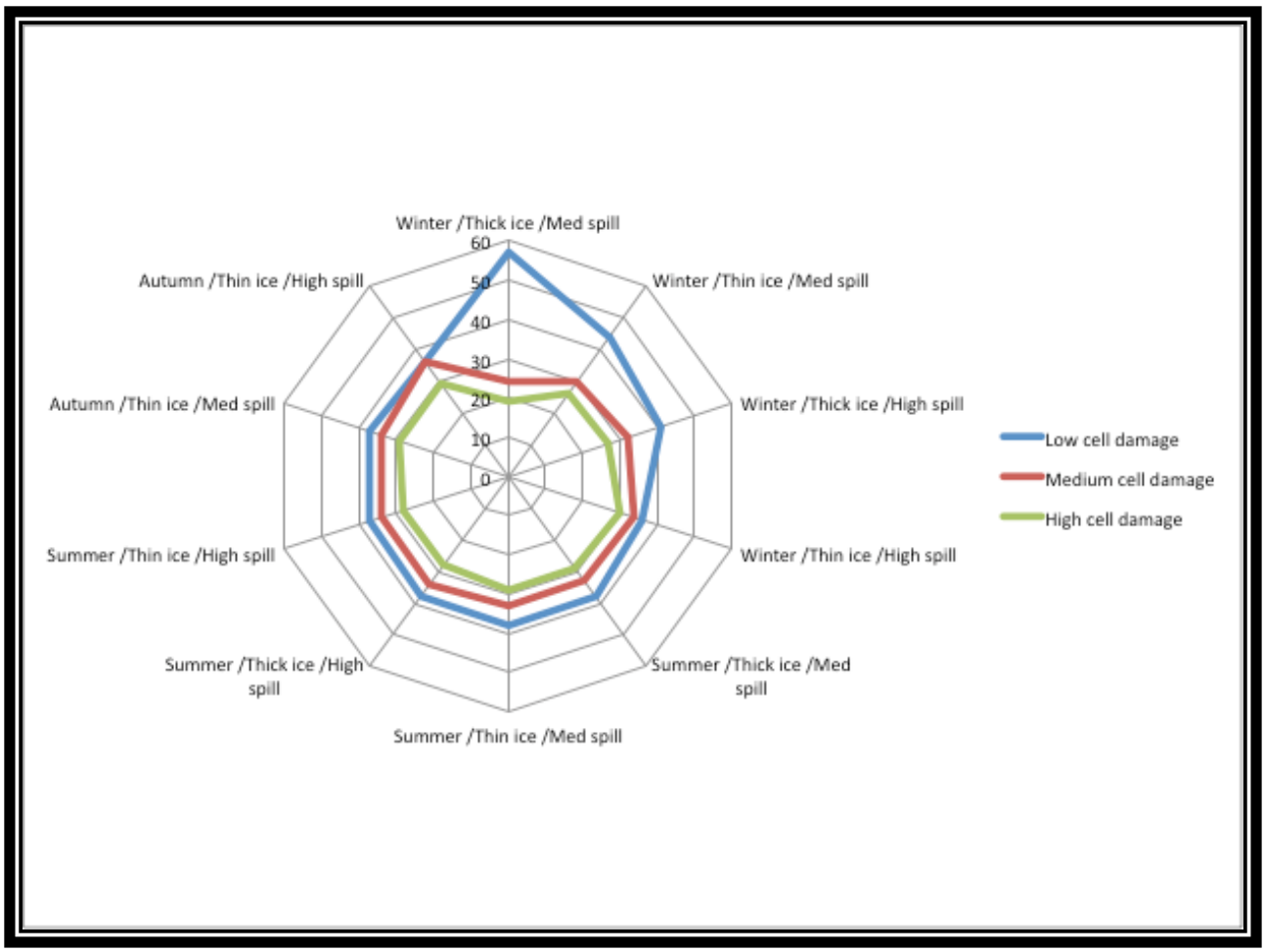


Figure 12: Probability of cell damage in all seasons, varying spill concentration and ice thickness

3.5 Conclusions

The process of biotransformation is very complex and there are many pathways in biotransformation as mentioned in Varanasi (1989) and Giulio (2008). The pathway catalyzed by CYP1A was selected in this study because this pathway was identified as a major contributor in biotransformation by various studies such as Varanasi (1989), Lacoste et al. (2013), and Sorhus et al. (2021). The BBN model can be used to circumvent the need for toxicity assays and also present a mechanism-based approach to determine the toxicity in aquatic species. When biomarkers for other biotransformation pathway can be identified, they can be added into the BBN model. More biomarkers that are sensitive to Phase I, Phase II and detoxification processes for a given chemical

of concern can be added to the model for increased accuracy in results. The BN model, using biomarkers, geophysical features and physiological features as nodes, could act as an effective tool to assist in evaluating the damage to the health of marine ecosystems impacted by oil spills.

This work can be further improved by considering following points:

1. Capturing the dynamic nature of ice in a given season is a challenge in the present Bayesian model. However, a continuous time variable could also be incorporated in the model in future.
2. The biggest challenge is to use the probability of toxicity to interpret a meaningful estimation of probability of cell death, and then extrapolate this to the organism. This extrapolation is to be further used to identify impact of recolonization or thriving of a colony by incorporating population models.

Chapter 4: Risk assessment of polar cod using ecotoxicological biomarkers and Bayesian Network

4.1 Introduction

The accidental spill of crude oil in the Arctic region due to the operations of the shipping and petroleum industries poses a risk to the marine species (Bakke et al. 2016, Jensen 2014). Conducting the environmental risk assessment for various marine species and predicting the possible effects on the species survival is imperative for planning remediation and mitigative measures after a spill. However, the lack of toxicity data of various polar marine species could hamper the exercise of conducting the environmental risk assessment. The general practice of using temperate region fish toxicity data for polar region counterparts is debatable owing to many physiological distinctions of the polar fishes (Fahd et al. 2019). The purpose of this study is to develop a risk assessment model that circumvents the use of toxicity assays for Arctic marine species. The model uses a Bayesian-based approach to predict the probability of cell deaths in individual polar cods that are exposed to Polycyclic Aromatic Hydrocarbons (PAH) in an oil spill. The results from the Bayesian network (BN) are further interpreted to determine the probability of cell deaths in a population of the organism. By analogy, the percentage of deaths in a population of aquatic species is equivalent to the probability of cell deaths from the model.

The general steps in environmental risk assessment (ERA) are hazard identification, exposure modelling, toxicological modelling, and risk characterization (EPA 1992). Hazard identification deals with identifying the toxicant of interest, species of interest, and site-specific factors defining

the susceptibility scope of the risk. The exposure modelling and toxicological modelling can be termed together as the analysis step of the ERA, as shown in Figure 13. The purpose of exposure modelling is to measure the spatial and temporal distribution of the toxicant and the species of concern. The goal of toxicological modelling is to quantify the adverse outcomes elicited by the toxicant. Risk characterization uses the results from the analysis step of the framework to evaluate the likelihood of the adverse outcomes in a target population due to exposure. Risk characterization also includes various assumptions made in the study, along with a discussion of the uncertainties, strengths, and weaknesses in the analysis. Figure 13 shows the framework of ecological risk assessment with details of steps taken in this study. This chapter is published as a research paper in the journal Marine Pollution Bulletin.[3]

[3] Fahd F., Veitch B., Khan F. Risk assessment of Arctic aquatic species using ecotoxicological biomarkers and Bayesian network. Marine Pollution Bulletin, 2020, 156: 111212.

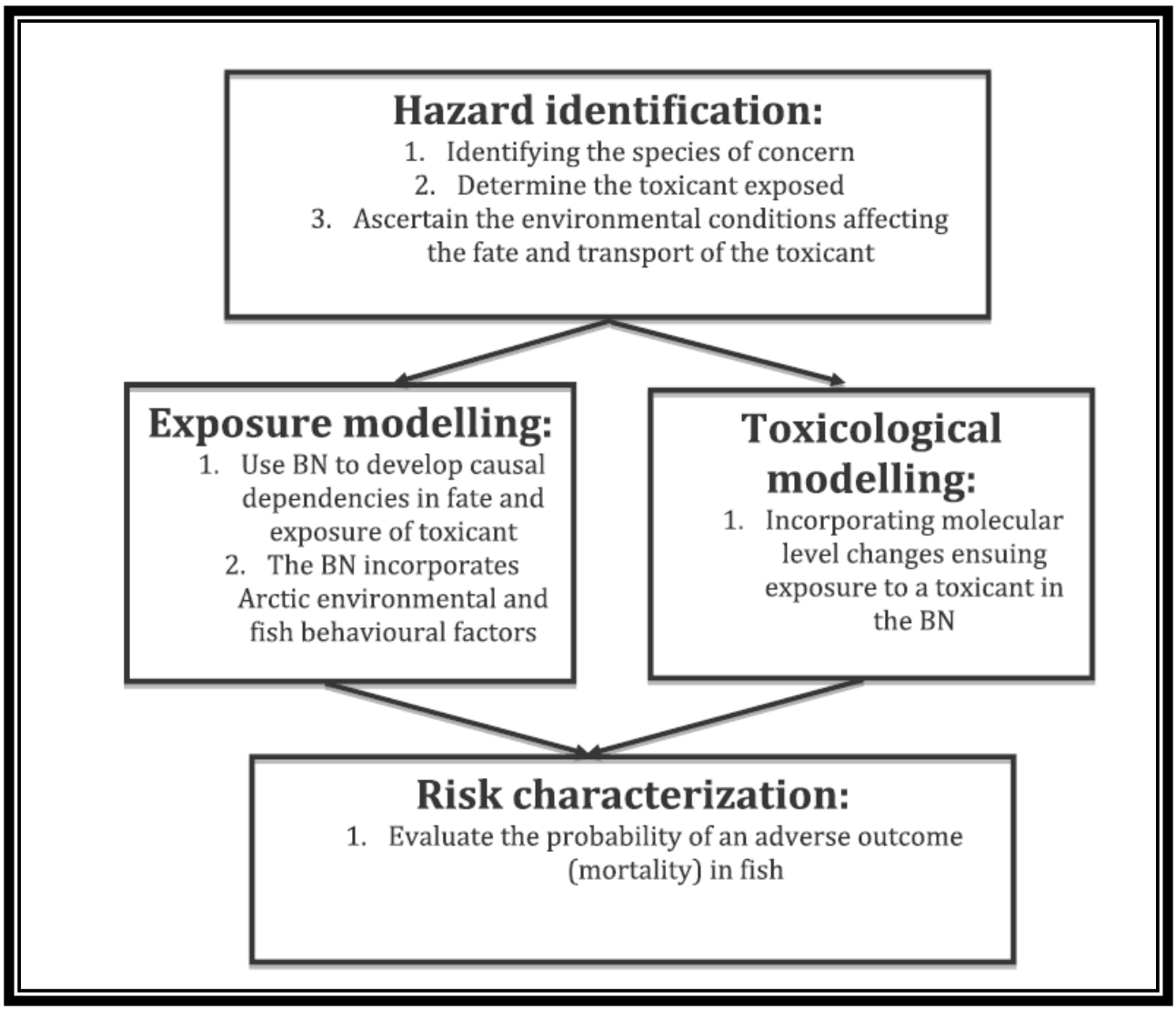


Figure 13: Framework of the Arctic region aquatic ecological risk assessment

Bayesian networks (BNs) are conceptual cause-effect models with each node representing variables in the system. The series of arrows connecting these nodes represent the causal/conditional dependencies based on the process or mechanism. BN is used to calculate how probable an event is and how its probability changes with respect to subsequent system changes or the presence of the evidence (Pollina and Hart 2005). Conditional probabilities represent the strength of the relationship between the nodes connected by arrows. Two or more states can be

attributed to each of the nodes. Prior probabilities can be assigned to each variable, which can be determined based on data, expert opinion, or a combination of both. Upon updating the BN, prior probabilities are upgraded to a new set of observations. Recent studies, such as Lu et al. (2019) and Afenyo et al. (2019), used BN to model spilled crude oil recovery effectiveness in the ice covered waters. BN was used Although BNs have been used by risk analyst in the fields of forest management (Tantipisanuh et al. 2014), fisheries management (Underwood et al. 2015), and other environmental issues (Fox 2010), limited work is accomplished in using BNs in ecotoxicological assessment of Arctic oil spill scenarios (Nevalainen et al. 2017). Sources of uncertainty in the ecotoxicology include poor understanding of the complex mechanism of toxicity in the organism and lack of dose-response data for the Arctic aquatic species. BN is one of the methods in analyzing uncertainty in ecotoxicology. An essential characteristic of BN is that it can readily be updated upon learning new information, whether of the toxicity data or causal dependencies in the BN. Also, some of the ecotoxicology biomarkers data are measured in different units. Some biomarkers are based on the total count of genes, while a few are measured as the reagent used per mg per minute, while some others are calculated based on visual observations by counting of fluorescence at specific wavelengths. BN provides an excellent platform for various biomarkers to be linked to obtain cell toxicity, as explained in the section below.

4.2 Hazard identification

One of the significant hazards of shipping and oil exploration in Arctic regions is the possibility of oil spills. In the event of an oil spill, while there are many aquatic species risking exposure, this study focuses on the risk assessment of Boreogadus Saida (polar cod). The Arctic food chain is short and non-complex. Polar cod serves as an essential food source to most marine bird species, seals, walrus, and polar bears (Hop and Gjosaeter 2013, Bakke et al. 2016). Various studies have

identified polar cod as a notable species in the Arctic region, and it can act as an excellent indicator for risk assessment of different trophic levels in the region (Christiansen et al. 2014, Tomy et al. 2014). PAH is an important constituent of crude oil with respect to causing deleterious effects to the marine organism (Jensen 2014). PAH and its metabolites can have harmful effects on fish by the production of intracellular reactive oxygen species (ROS) during various biochemical processes, such as biotransformation (Vieweg et al. 2017). The ROS are also generated in the detoxification process when an organism is exposed to various xenobiotics. Although the production of oxygen radicals is integral to the normal functioning of the fish, the naturally occurring oxygen radicals in the fish are neutralized by the action of antioxidant enzymes (Amado et al. 2009). The balance between the pro-oxidant actions and antioxidant actions of the enzymes is what causes normal functioning or homeostatic functioning of an organism (Carney Almroth 2008; Fahd et al. 2019). Mortality in polar cod is due to the production of unconjugated reactive oxygen species (ROS) in target organs such as liver and lipids. Fahd et al. (2019) details the importance of ROS balance for homeostatic functioning of the organism and mechanism of biotransformation toxicity in polar cod.

Apart from the PAH induced effects, mortality in polar cod due to oil spill exposure is also affected by the extreme environmental conditions and the physiological conditions of exposed aquatic organisms. For example, polar cod are exposed to a variety of environmental stressors such as elevated levels of oxygen in the cold polar waters and high ultraviolet radiation in the Arctic summers, which can increase the probability of ROS production. Polar fish-specific physiological conditions, such as varying lipid content over seasons and presence of elevated levels of unsaturated fatty acids to maintain membrane fluidity, also increase the organisms' susceptibility to ROS driven toxicity (Camus et al. 2003). Studies such as (Strobel et al. 2015 and Vieweg et al.

2017) have observed higher baseline antioxidant levels in polar fish when compared to temperate fish. High baseline levels of antioxidant activity could result in impeding detoxification processes in the organism leading to excessive deleterious effects.

4.2.1 The geographic extent of study and species of concern

The geographic area of the study is the Arctic region, and the species of interest in the study is polar cod, which is the most abundant fish species in the Arctic region (Hop and Gjosaeter 2013). Polar cod feed on amphipods and zooplankton, such as copepods. The habitat distribution of polar cods is divided into sympagic and pelagic phases. Polar cod spawn in early winter and the early life stages and juvenile polar cod are mostly associated with the sympagic life cycle. The adults are also pelagicly distributed in summers (Hop and Gjosaeter 2013). The sea ice thickness data and algal bloom data variations by the seasons, as shown in Table 8, were collected from the region around Svalbard and the Fram Strait by Werner (2006). Three seasons, namely, winter, summer and autumn, are used in this study as polar cod data was not collected by Nahrgang et al. (2010) during the spring season.

Table 8: Sea ice data, seasons, algal bloom data and other pertinent data

Season	**Depth of sea ice at the location (m)**	**Algal bloom in ice (mg/m^2)**	**Baseline Phase I activity (pmol/min/mg)**	**Baseline Phase II activity (nmol/min/mg)**	**Reference**
Winter	0.8-3.5	0.12-2.63	3	500	Werner (2006) and Nahrgang et al. (2010)
Summer	1.4-3.4	0.13-2.94	13	200	
Autumn	1.6-3.3	0.25-18.4	3	350	

4.3 Methodology

The framework for the methodology adopted in this chapter is presented in Figure 13. Hazard identification was discussed in the section 4.2, while the toxicological modeling and Exposure modeling are discussed below.

4.3.1 Toxicological Modeling

The Bayesian network developed for toxicological modelling is based on the principles and processes of biotransformation and detoxification in the polar cod.

The pathways of biotransformation and detoxification are shown in Figure 14. Biomarkers, which are the measurable indicators of PAH biotransformation and detoxification, are used as nodes in the BN model. The biomarkers include ethoxyresorufin-O-deethylase (EROD) and Glutathione-S-transferase (GST) for Phase I and Phase II biotransformation processes, respectively. Other biomarkers, which are the measurable indicators of reactive metabolites are hydroxyl radical (OH) and peroxyl radicals (ROO), Total Oxygen Scavenging Capacity (TOSC) OH, and TOSC ROO respectively. These biomarkers are also used as nodes in the BN model. This study is a first attempt to make a quantitative toxicity model using a mechanism-based approach. Although other quantitative models exist wherein various ‘weights’ are assigned to the biomarkers based on toxicological importance (Piva et al. 2011, Vieweg et al. 2017), these approaches are not based on the toxicity mechanism.

4.3.1.1 Pathways for ROS production and detoxification:

In an oil spill of substantial quantity, the initial deaths of the exposed organisms are caused by smothering and asphyxiation due to the oil. Under smaller concentrations, the exposure concentration of PAH reaches the various target tissues in the organism. Many studies have alluded to the concept of using contaminant concentration at the target tissue level as an indicator as opposed to the contaminant concentration in the exposed water (Ducrot et al. 2016). The study takes the same idea and further tries to estimate the amount of contaminant activated in various target tissues and then safely biotransformed and removed from the body. The amount of contaminant that is not negotiated safely by the organism could determine the effects induced in the organism. When an organism is exposed to PAH, a mechanism for detoxification is initiated. The presence of PAH triggers initiation of the CYP1A enzymes, which act on the PAH via oxidation or reduction processes and insert an oxygen atom in the PAH compounds (Varanasi 1989). These actions are termed Phase I metabolism in the organism. The CYP1A enzymes are mostly present in the microsomes of the liver. There is limited Phase I activity reported in the gills of the fishes, and it is mainly from the xenobiotics absorbed and circulated through the blood in the organism (Ingebrigtsen et al. 2000). The two possible outcomes once the Phase I metabolism is activated are the generation of benign non-reactive metabolites and reactive metabolites (Varanasi 1989, Giulio and Hinton 2008). The reactive metabolites are conjugated by the action of Glutathione S-Transferase (GST) enzymes. However, if the total reactionary metabolites are more than the amount of GST enzymes present for conjugation, then those metabolites contribute to the production of superoxide anions (O_2^-). Some of these anions are further converted to hydrogen peroxide with the help of superoxide dismutase (SOD) enzymes. Another set of enzymes, namely catalase (CAT), safely converts the hydrogen peroxide to water and oxygen

(Santana et al. 2018). While some superoxide anions and hydrogen peroxide participate in Fenton reaction in the presence of Fe^{2+} to produce hydroxyl radical ($HO\cdot$). The hydroxyl radical is one of the worst possible outcomes that could be produced in the body as these are extremely reactive (Santana et al. 2018). These radicals can cause cell death due to DNA damage (Madureira et al. 2014). They also contribute to the creation of lipid peroxyl radicals. The lipid peroxyl radicals generated can be safely conjugated using the glutathione peroxidase (GPx) enzymes. A biomarker malonaldehyde (MDA) indicates the extent of lipid peroxidation in the polar cod. Pertinent biomarkers quantifying the effects in the polar cod are used as indicators of detoxification activity in the organism. The framework of the part of the BN model for the toxicological modelling step is based on Figure 14, while further nodes are added representing biomarkers for ROS and environmental and physiological conditions pertaining to the Arctic region.

4.3.1.2 Biomarkers effectively reflect the risk in an organism:

The 'risk or damage' considered in this study is due to the cell's death in the organism. It is assumed that upon the death of 35% of the cells in an organ, death occurs (Priante et al., 2019). Cell deaths are caused by two possibilities, namely, DNA damage and cell membrane compromise (Banni et al. 2009, Hooper et al. 2012). Identifying the risk to polar cod from mortality can be better facilitated by integrating the mechanism of toxicity with the biological endpoints such as biochemical and physiological biomarkers. Intracellular biomarkers have been used as indicators of PAH exposure for many marine organisms (Connon et al. 2012; Hook et al. 2014). Although the use of biomarkers is not new, their use in conjunction with the aquatic toxicity mechanism for the Arctic region is novel. Recent studies have used intracellular biomarkers in polar cod as

indicators of the trends of toxicants removal (Hook et al. 2014, Regoli et al. 2011). Jensen 2014 conducted crude oil exposure experiments with Atlantic cod and observed a significant increase in the concentrations of the biliary metabolites and an increase in EROD activities and CYP1A concentrations. Although the CYP1A responses have increased with the increase in crude oil exposure, they were not dosed dependent, according to Jensen (2014). This was true for fishes exposed to higher concentrations of crude oil owing to inhibition substance, or possibly because the responses to the highest oil concentration surpassed the maximum threshold induction in the body. Other studies, such as Vieweg et al. (2017), have observed that the CYP1A activity, represented by EROD, was dose-dependent for the polar cod used in experiments. The antioxidant defense responses, such as the GST and CAT, varied in the experiments and showed only a slight increase with the exposure and duration.

However, owing to the limited experimental work associated with the Arctic marine organism, some biomarkers may not show appropriate exposure dependent trends. Studies such as Nahrgang et al. (2009 and 2010) have demonstrated that the toxicant exposures were in line with the biomarker responses elicited. Nahrgang et al. (2010,2016) showed that EROD activity is in line with the PAH exposure, while the GST and CAT activity in polar cod were in some contradictions. However, the same study found that the total oxygen scavenging capacity (TOSC) of the polar cod liver tissue increased following the PAH exposure. The TOSC experiment determines the actual mitigating capacity of the liver tissue to hydroxyl (TOSC OH) and peroxidation (TOSC ROO). The results from lipid peroxidation assay indicate that the level of malonaldehyde (MDA) in the liver and gills of fish increased significantly when compared to their control samples (Otitoloju and Olagoke 2011). Data of the biomarkers and their elicited response for the given exposure are

presented in Table 9. The data from this table are used to classify the states of nodes such as ‘Phase I activity’, ’Phase II GST’, ‘Catalase (CAT)’, ‘TOSC ROO’, and ‘TOSC OH’.

Using multiple biomarkers to analyze a mechanism helps decrease the uncertainty and increase the precision of prediction in a BN. Therefore, biomarkers such as CAT, TOSC, GST, GPx, and MDA, which contribute in some fashion to the toxicant removal in the polar cod, are to be simultaneously used in the BN.

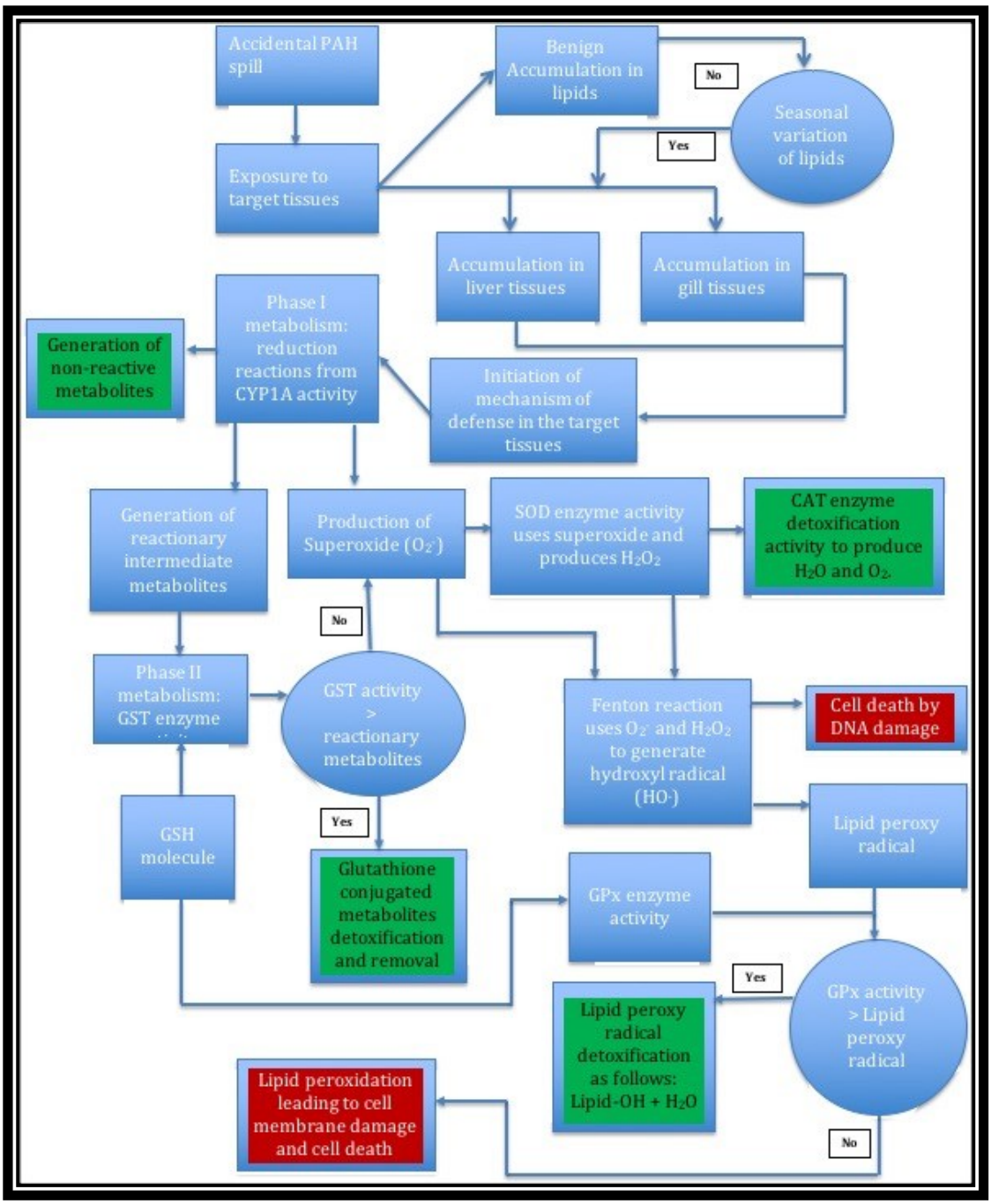


Figure 14: Pathways of ROS production and detoxification in polar cod

(Green boxes represent safe outcome while the red boxes represent risk)

4.3.2 Exposure modelling

The environmental and physiological factors influencing the polar cod aquatic exposure and subsequent toxicity that are incorporated in the model are discussed in this section.

Sea ice: The most prominent environmental and geophysical factor in the Arctic region is sea ice. The presence and depth of the sea ice are dependent on the season in the region. Data collected by Werner (2006) from the Fram Strait varies the sea ice from 3.5m in winters to 0.8-1.6 m in the summer season, respectively.

Exposure of polar cod to crude oil is highly dependent on the location of the oil spill concerning the sea ice. A spill on the sea ice must traverse the depth of the ice to reach the ice-water interface. The movement of the oil in the ice is facilitated by the presence of cracks and leads in the ice, thus delaying and minimizing the exposure concentrations to the polar cod. The cracks and leads in the sea ice are also season dependent. A spill under the ice accumulates the oil in the rough under the surface of the ice. In such an event, the potential exposure of the oil to polar cod is very high as juvenile polar cod use crevices in the sea ice undersurface as habitat (Hop and Gjosaeter 2013). Refer to Table 9 for the data on pertinent nodes, such as, 'Sea ice thickness,' and ‘Leads, polynyas, and brine channels.'

Baseline enzyme activity: The enzyme activity occurring in the body is limited by the presence of phase I and phase II enzyme in the liver microsome and cytosol, respectively. Higher baseline enzyme phase I and phase II activity result in less effective biotransformation capacity of the organism. There is a regional and seasonal variation in the baseline enzyme activity in the polar cod, as observed from the data shown in Table 8 and Table 9.

Feeding activity and PAH accumulation in lipids: PAH accumulation in lipids is directly related to the spawning season for the polar cod. Feeding activity varies in accordance with the abundance of food availability. During the summer months, there is much availability of food leading to a feeding frenzy amongst the polar cod. Also, the increased feeding is to accumulate the fat reserve that could be used when spawning in the following months of early Winter (Hop and Gjosaeter, 2013). Hop and Gjosaeter (2013) reported liver lipids in polar cod in general to be about 60-65% and about 3% of lipids in muscles. Increased feeding activity could translate to increased exposure of PAH via a dietary route under favorable conditions. The abundance of food availability and measure of feeding activity is based on proxy field observation of algal bloom in the season. The seasonal variation of algal bloom for the region of study is shown in Table 8. Although PAH accumulation in lipids in polar cod is not measured directly, the Hepatic Somatic Index (HSI) of the sampled fish is used as a proxy for PAH accumulation in lipids.

4.2.3 Data for the model

Each node in the model is assigned with three states, namely, Low, Medium (Med), and High. Firstly, the assigned states for the nodes are to be defined as to what constitutes as Low, Med, and High. Table 9 lists all the nodes and assigns a range of values characterizing the nodes. The discretization of data in Low, Med, and High states is based on reference studies or on assumptions made from the observations in the reference studies. For example, Banni et al. (2009) observed that about 85% of the PAH concentration reaching in the fish is metabolized in the liver.

Using the observation from Banni et al. as the value for the high state in the node ‘Concentration reaching liver’, Medium and Low state values were assumed.

Table 9: Characteristics of the states of the nodes in the ecotoxicity model for polar cod.

Nodes	States	Comments	References
Season	Winter, Summer, Autumn	Winter considered from December to June, Summer from July to September and Autumn from October to November.	Werner (2006) and Nahrgang et al. (2010)
Spill location	Over ice, under ice and over thin ice.	Assumptions made in this study. This study did not consider spill under the thin ice as it is assumed that the case would be similar to spill on thin ice.	
PAH spill concentration	Low, medium and high.	Low – <30 µg/L Med – 30-60 µg/L High- 60- 100 µg/L	Nahrgang et al. (2010) and Rodd et al. (2017)
Sea ice thickness	Low, medium and high.	Low- 0.5 -1.5 m Med - 1.5-2.5m High- 2.5-3.5m	Werner (2006)
Leads, polynyas and brine channels	Low, medium and high.	The percentage of open areas or cracks from the surface to the bottom of the ice Low - <10% Med - <25% High -<40%	Assumption
Feeding activity	Low and high.	Low - <15% High- >15%	Cusa (2016) and assumptions.
Accumulation in lipid	Low, medium and high.	The concentration of PAH trapped with the lipids in muscles and liver lipids. Low - <10% Med - <20% High- <30%	Hop and Gjosaeter (2013) and assumptions.
Bioavailable concentration	Low, medium and high.	Low - <60% Med -60-80% High ->80%	Banni et al. (2009) and assumption.
Concentration reaching liver	Low, medium and high.	Low - <60% Med -60-80% High ->80%	Banni et al. (2009) and assumption.
Liver microsomes	Low, medium and high.	The liver is around 2 to 7% of the dry weight of the fish. The endoplasmic reticulum takes about 15-20% of the liver area.	Priante et al. (2019) and assumption.

		Low – 5-7% Med – 7-15% High – 15-25%	
Phase I activity	Low, medium and high.	The phase I activity is measured in induction and increase in folds of the CYP1A enzyme activity Low – 8 Med – 9 High – 10	Rodd et al. (2017)
Phase II activity	Low, medium, and high.	Phase II is measured in fold increase in GST activity. Low – 3 Med – 5 High – 7	Nahrgang et al. (2010)
Baseline Phase I	Normal and high	The phase I activity is measured in pmol/min/mg. Low - <6 High – 7-15	Nahrgang et al. (2010)
Baseline Phase II	Normal and high	The phase II activity is measured in nmol/min/mg. Low – < 300 High – 300-600	Nahrgang et al. (2010)
TOSC OH	Low, medium and high	The activity is measured in TOSC unit/mg protein. Low- <250 Med – 250-500 High – 500-750	Vieweg et al. (2017)
TOSC ROO	Low, medium and high	The activity is measured in TOSC unit/mg protein. Low - <200 Med -200-400 High – 400-600	Vieweg et al. (2017)
Catalase (CAT)	Low, medium and high.	The activity is measured in µmol/min/mg protein. Low - <150 Med – 150-300 High – 300-450	Vieweg et al. (2017)
Cell death from DNA damage	Low, medium and high.	Low - <15% Med – 15-30% High - >30%	Madureira et al. (2014) and assumption.

Cell death from lipid peroxidation	Low, medium and high.	Low - <15% Med – 15-30% High - >30%	Madureira et al. (2014) and assumption.
Total cell death	Low, medium and high.	Low - <15% Med – 15-30% High - >30%	Madureira et al. (2014) and assumption.

4.3.4 Developing the conditional dependencies in the model

The conditional probability tables of various nodes and the reason for choosing the probability values of each state of the node are described in Supplementary materials document section 1.0. The CPTs for all the nodes are listed in Tables S1 through S21.

4.4 Results and Discussion

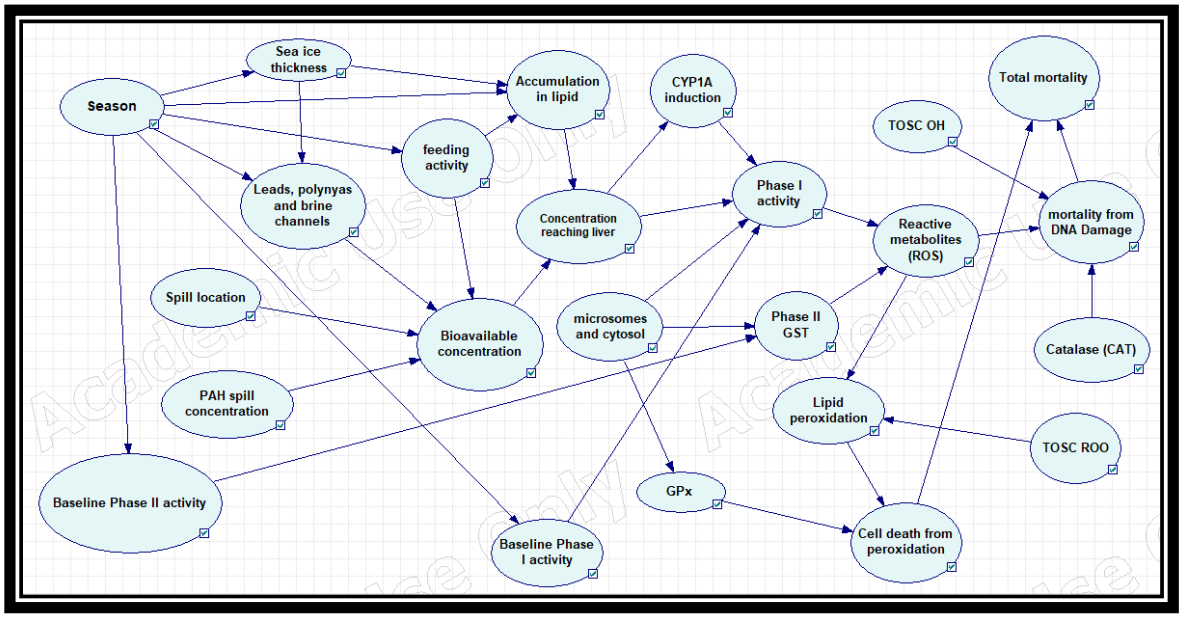


Figure 15: Bayesian network based ecotoxicity model for polar cod

4.4.1 Risk Characterization:

In a hypothetical event of a shipping accident in the Arctic region, the following scenarios are considered in this study:

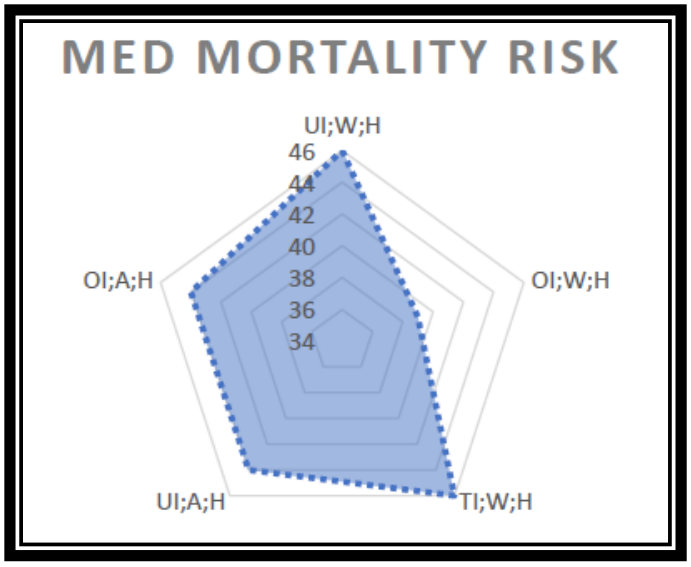
1. Spill over thick ice
2. Spill under thick ice
3. Spill over thin ice

For the given scenarios, the results of the probability of polar cod death for different seasons, exposure concentrations and physiological conditions are predicted in this study. In winter, the oil must traverse the thick ice and reach the ice-water interface to cause exposure to the polar cod and the openings in the ice are minimal.

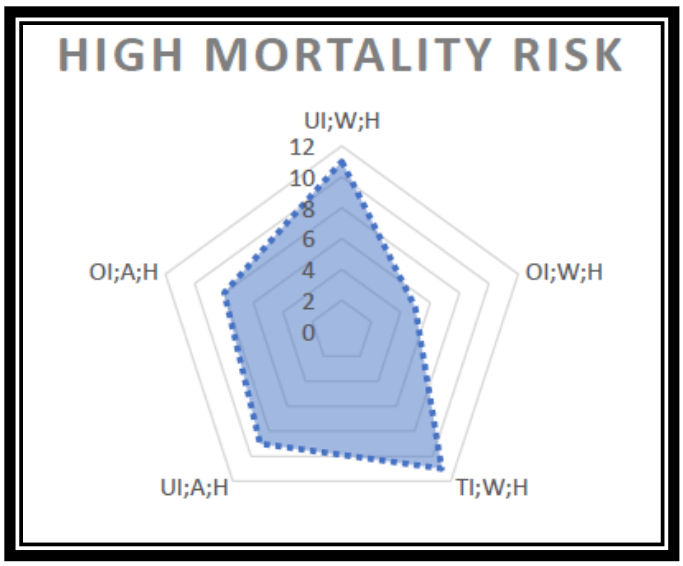
Therefore, in the scenario of spill over thick ice, for all the levels of a PAH spill, the probability of a safe outcome was high, as shown in Figure 16. Changing the seasons did not bring a significant change in the probabilities of 'No risk' outcome, although the estimated probability of cell damage in polar cod is highest in the Autumn and lowest in Winter for any spill concentration (Figure 16). This is owing to the increased cracks and openings in the sea ice in autumn, leading to increased PAH exposure. The concentration of PAH in a spill reaching the liver of the fish elicits the phase I and phase II actions. Thus, higher concentrations of PAH in the liver could lead to more ROS generation. The variations of the PAH reaching the liver vary significantly for winter, summer, and autumn, as shown in Table S22 of Supplementary data document. It is observed from the model that concentration reaching the liver is highest in autumn and lowest in winter, and it follows the same pattern as the probabilities of cell deaths for seasonal variation. The polar cod generate and store extra lipids during the summer to facilitate spawning in early winter, and the lipids tend to accumulate the PAH, drawing them away from the metabolism process. However, based on the conditional probabilities, the BN model predicted higher PAH concentration reaching the liver even when the liver lipids are the highest.

In the event of an oil spill in the sparse ice season, the oil can be entrapped in the growing sea ice in the following winter season. Also, an oil spill under the ice is more likely to be observed in a real-life scenario. The probability of cell damage is low to medium for high PAH exposure and low cell damage for low PAH exposure. The difference between low and high PAH exposures is varying considerably, unlike the scenario of spill over thick ice (Table S22).

Thin sea ice is observed towards the end of winter and in the summer season. Thin sea ice could also be observed during the first-year ice formations. The breaks in sea ice can cause the movement of the oil to reach the ice-water interface in summer and autumn. Considering a scenario where an oil spill occurs over the thin ice during winter, for low PAH exposure concentration, the probability of cell death is 60% low and 37% medium. For high PAH exposure concentration, the risk is moderate, with the probability of cell death being 45% for both low and medium.



(A)



(B)

Figure 16: (A) Percentage of polar cod at medium risk from high exposure scenario; (B) Percentage of polar cod at high risk from high exposure scenario.

UI: Spill under thick ice

OI: Spill over thick ice

TI: Spill over thin ice

H: High PAH exposure concentration

W: Winter

A: Autumn

Biomarker evidence:

Biomarkers are used as evidence nodes and as proxies for physiological conditions of the polar cod. The total oxygen scavenging capacity of hydroxyl radicals, peroxyl radicals, and catalase enzyme activity are set as evidence. The worst-case scenario from Figure 16 and Table S22 is used to model the sensitivity of the evidence nodes in the model by toggling TOSC for both hydroxyl radicals and peroxyl radicals and catalase activity between low and high states. The

probability of cell damage when evidence nodes are ‘Low’ is moderate to high, and when evidence is 'High,' the risk is low, as shown in Table 10.

Sensitivity analysis: Sensitivity analysis was considered for the nodes of ‘Cell Death from DNA Damage’ and ‘Cell Death from Peroxidation'. The sensitivity analysis was conducted using the GeNie software by BayesianFusion, LLC. The sensitivity of the node cell death from peroxidation is mostly uniformly influenced by its various nodes except for the case of microsome and cytosol content and concentration reaching liver nodes. From Figure 17 and Figure 18, it is evident that Cell damage is highly influenced by microsome and cytosol content, phase I metabolites, and phase II metabolites. The most ideal node states that estimate a low cell death by DNA damage is low PAH exposure, low microsomes, and high cytosol. Since microsomes house, the CYP1A enzymes and cytosol houses the phase II detoxification enzymes, low microsomes could result in less reactive metabolites, and high detoxification ensures low damage due to cell death.

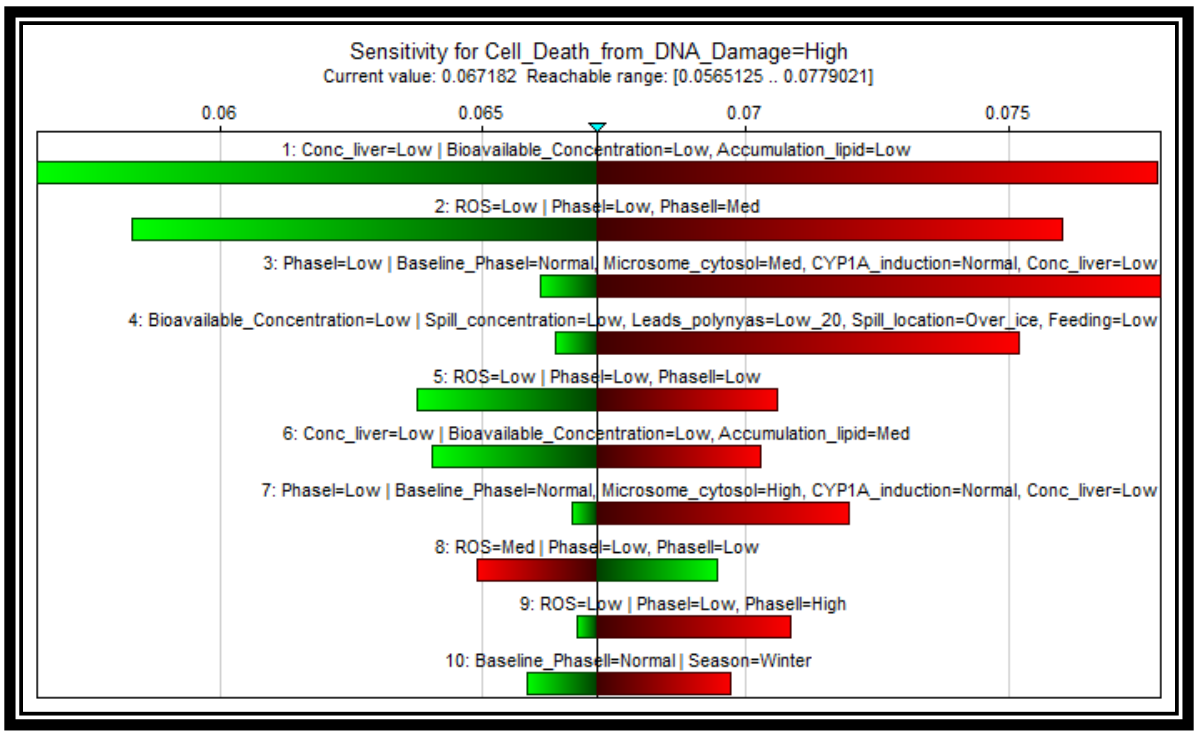


Figure 17: Sensitivity analysis of 'Cell death from DNA damage' node

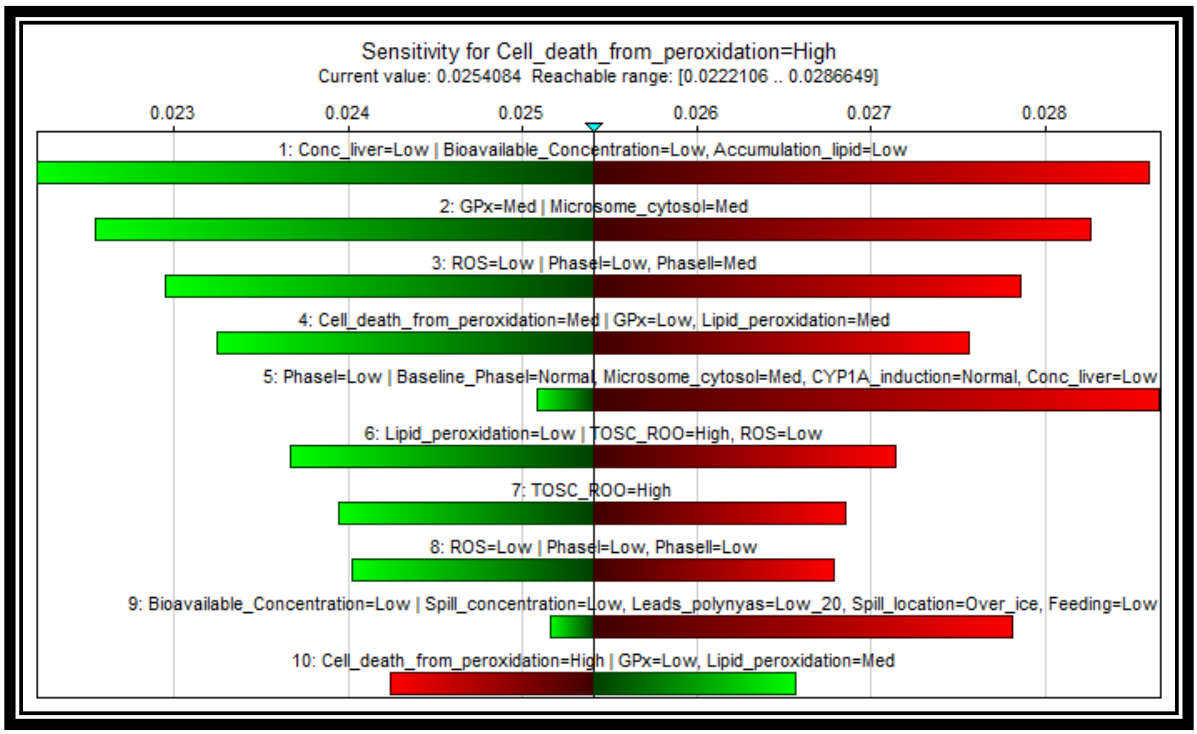
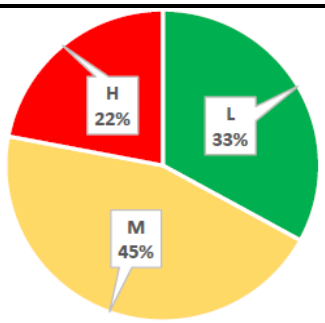
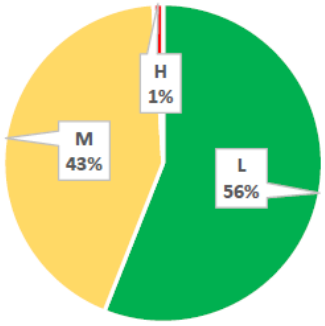
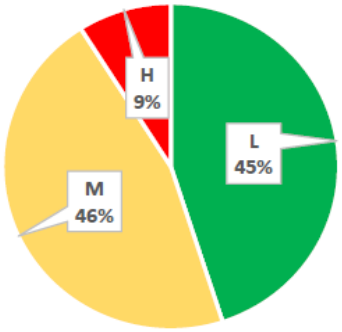
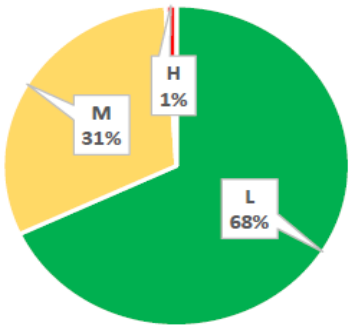


Figure 18: Sensitivity analysis of 'Cell death from peroxidation' node.

Table 10: Sensitivity impact of biomarker evidence nodes used in the ecotoxicity model

Spill location	**Season**	**TOSC OH**	**TOSC ROO**	**CAT**	**Total cell death (L-Low, M-Medium, H-High)**
Under ice	Summer	Low	Low	Low	
		High	High	High	
Over thick ice	Winter	Low	Low	Low	
		High	High	High	

4.4 Validation of the model

The model can be validated at the conceptual level, i.e., validation of the conditional probabilities or data trends of various nodes, and of the final results, which is mortality in polar cod. The model is validated using two studies on fish ecotoxicology. The first is a Joint Industry Program (JIP) conducted by Camus (2017), where in-ice open mesocosm field experiments were conducted in the Svalbard region. The experiments lasted from January 2015, when sea ice is prevalent, to ice melting season of July 2015. The study examined, amongst other things, the amount of PAH reaching the ice-water interface, the survival of zooplankton under ice, and ice algae primary production. Camus (2017) also studied the sensitivity and resiliency of juvenile polar cod when exposed to PAH concentrations of about 120 µg/L. For the quantity of crude oil spill used in the field experiments, only a fraction of the PAH reached to the bottom of the sea ice. This observation is in line with the data interpreted from the nodes 'Bioavailable concentration' and 'Concentration reaching liver' and their CPTs. No significant change in ice algae production was observed from the control sample. This implies that the presence of oil did not impede the food availability for copepods and amphipods, which are consumed by the polar cod. High feeding by the polar cods takes place in autumn to sustain through the spawning period in February. Table 9 shows that the data ranges used in the model are verified by the JIP conducted by Camus (2017). For the given exposure scenario by Camus et al., no significant increases in polar cod mortality were observed over the control sample. This observation also is in line with the results from the model where, for the worst-case scenario, the probability of high risk was 10%.

Apart from the field study, a cell-based model for stress response in cells is used for verification of the BN based ecotoxicity model developed in this study. Madureira et al. (2014) exposed a murine cell line to the environmentally relevant concentration of 50 nM (12.6 µg/L) of BaP and an extreme exposure scenario of 5µM (1.26 mg/L). The stress response of the cells was studied, and the DNA adducts formed were quantified by the study. The cells exposed to 50nM generated around 4 adducts per cell and recovered almost fully after initial exposure, unlike the case for 5µM exposure scenario, where the adducts generated about 280. The BN model shows similar results as 50 nM exposure scenarios, i.e., the risk of mortality is low.

4.5 Conclusions

Seasonal sea ice played a major role in containing PAH exposure to polar cod and subsequently leading to lesser possibility of risk. Apart from the seasonal sea ice, other physiological factors also played major role in determining mortality risk in polar cod. The physiological factors, such as presence of higher Phase II activity, and higher oxyradical scavenging ability, played greater impact on PAH risk mitigation in polar cod than seasonal environmental factors could.

Environmentally relevant concentrations were used in this study. Higher concentrations in the model were not used for two reasons. First, the initial oil spill quantity is usually large, and it kills the animals in contact by smothering or asphyxiation. The role of biotransformation and cellular response is hardly present. Second, it was observed that large concentrations of oil exposed to various fish had inhibited certain cellular responses. Since this study integrates the cellular responses with the PAH exposure to determine the toxicity risk, a scenario where certain cellular

responses are inhibited does not work for the toxicity model developed. The bioavailability and toxicity of oil are dependent on the type of oil constituents, and its biodegradability, which are beyond the scope of this research.

No distinction is made to water exposure and dietary exposure of PAH in this study. However, studies such as Bakke et al. (2016) studied the disposition of BaP and phenanthrene in polar cod. They identified the significant distribution of the PAH in bile and intestine, while some of the PAH is also distributed in the gills for waterborne exposures. This distinction could be further incorporated in future studies.

New approaches in the genomics and bioinformatics can also be incorporated in the model as a biomarker node in future works. The higher number of biomarker nodes would increase the reliability of the predicted mortality percentage in the polar cod risk assessment studies. The biomarker data for the model is based on very few biochemical studies, as more studies are conducted, the data for the nodes would point to a more precise trend in effects estimation in polar cod.

Chapter 5: A food chain-based ecological risk assessment model for marine oil spills in the Arctic environment

5.1 Introduction

The Arctic is melting and becoming more attractive and accessible to human activities. The relatively pristine Arctic region is open to shipping and oil and gas exploration activities (Hoop et al., 2011; Chapman, 2003; Gardiner et al., 2013). Accidental oil spills may occur during these activities and impose severe impacts on the Arctic aquatic ecosystem (Lee et al., 2015; Helle et al., 2020; Nevalainen et al., 2017). There is an urgent need to assess the potential impacts these activities will have on the Arctic apex aquatic mammals and the food web of those animals. The challenges and knowledge gaps in oil spill ecological impact assessment in the Arctic region can be categorized broadly as the following:

- Lack of knowledge in oil spill fate and transport modelling in ice-infested waters: The presence of sea ice and its uncertainties hampers the clean-up in ice infested waters (Afenyo et al. 2017). Studies such as Sorstrom et al. (2010), Dickins (2011), and Singsaas et al. (2020) have conducted field and laboratory-scale experiments to study the oil spill fate and transport in ice infested waters. Afenyo et al. (2016) has described the oil spill transport process in ice-infested water in terms of spreading, dispersion, advection, sedimentation, and encapsulation. The presence of ice cover significantly impacts the weathering and transport processes in the Arctic region. Sea ice either could impede or facilitate the oil exposure to marine animals, based on the location of an oil spill and other environmental conditions (Fahd et al. 2019).
- Lack of aquatic toxicity data on Arctic species: Toxicity data for Arctic marine species is limited (Fahd et al. 2019). Toxicity data, such as the No Observed Effects Concentration

(NOEC) of crude oil to various species, is based on experimental studies or modeling based on a surrogate species. The knowledge gap in the toxicity data of Arctic species is also filled by using temperate fish data. Fahd et al. (2017, 2019, 2020) proposed that Arctic marine species toxicity data could be estimated based on the probability of cellular damage and metabolite interactions in the organism. The metabolite interactions, quantified by ecotoxicological biomarkers, are represented in causal relationships using a Bayesian Network (BN). Fahd et al. (2019, 2020) demonstrated the BN model by estimating mortality for Boreogadus saida (Arctic cod) populations for various assumed oil spill scenarios.

- Unique features of the food chain and the feeding behaviours of the marine species: The Arctic food web is comparatively simple. Therefore, the impact on one trophic level develops cascading effects on other trophic levels (Nevalainen et al. 2017). Apart from the animals' susceptibility to oil exposure, the non-availability of prey further compounds the population dynamics of the animals in the region. The availability of prey also impacts the ability to reproduce offspring; thus meeting energy requirements for the apex predators plays a significant role in their thriving. Arctic animals are over-dependent on one animal as a major source of energy. For example, a major part of the whale diet comprises of small fish, such as polar cod and capelin, while the seals are pivotal to the survival and reproduction energy requirement of polar bears. Studies such as Amstrup et al. (2008) have reported reduced body condition, reproduction, and survival for polar bears in ranges where sea ice reduction was observed. Sea-ice by itself may not be directly related to the population density. However, sea ice is an indicator of seals' presence, which forms a main source of nutrition for the polar bear. Across the range of the polar bears, the relationship

between ringed seals and polar bears is such that an abundance of ringed seals appears to regulate the density of the polar bears in the region.

This study has attempted to tackle the above challenges and fill the knowledge gap by developing a Bayesian network (BN)-based approach for ecological risk assessment of oil spills' impact on Arctic animals. The BN is based on the current understanding of the Arctic food chain. The BNs developed in previous chapters focussed on the risk assessment of polar cod, while the current BN model focuses on the cascading effects of polar cod in the food chain. The rest of the chapter is organized as follows. Section 5.2 presents the Arctic food chain first, based on which the BN-based ecological risk assessment approach is proposed. In Section 5.3, this method is applied to a hypothetical oil spill near Svalbard. This is followed by results and discussions in Section 5.4. Finally, Section 5.5 concludes the study. This chapter is published as a research paper in the journal Marin Pollution Bulletin[4].

5.2 The proposed methodology

The Arctic food web was developed based on Steiner et al. (2019), Amstrup et al. (2008), and Bluhm and Gradinger (2008), as shown in Figure 19. Understanding the idiosyncratic responses of the lower trophic level Arctic marine animals is crucial to predicting risks to the polar bear and beluga whale populations and essential for efficient conservation strategies (Chevalier et al. 2018). Identifying susceptible concentrations of PAH to individual Arctic mammals and the probability of exposure of the distribution of animals provides little insight unless they are combined with cascading effects in the marine food web. Overall impacts on the ecosystem are evaluated.

[4] Fahd F., Yang M., Khan F., Veitch B. A food chain-based ecological risk assessment model for oil spills in the Arctic environment. Marine Pollution Bulletin, 2021, 166, 112164.

Modeling studies, such as Carroll et al. (2018) and Gallaway et al. (2017), simulated the impacts on polar cod and northeast Arctic cod fisheries. Studies such as Nevalainen et al. (2017) and Helle et al. (2020) have linked the susceptibility and vulnerability of polar bears (individual and population) to the exposure of oil spills. No study has considered synergistic effects of prey availability changes in the food web with oil spill impact on apex marine mammals. Polar cod, an endemic Arctic keystone species (Cusa et al. 2016, Huserbraten et al. 2019), is selected. Its responses to oil spills are used to predict the mortality in polar cod populations. The cascading impacts of the changing polar cod populations in the Arctic apex marine mammals' food web is then studied.

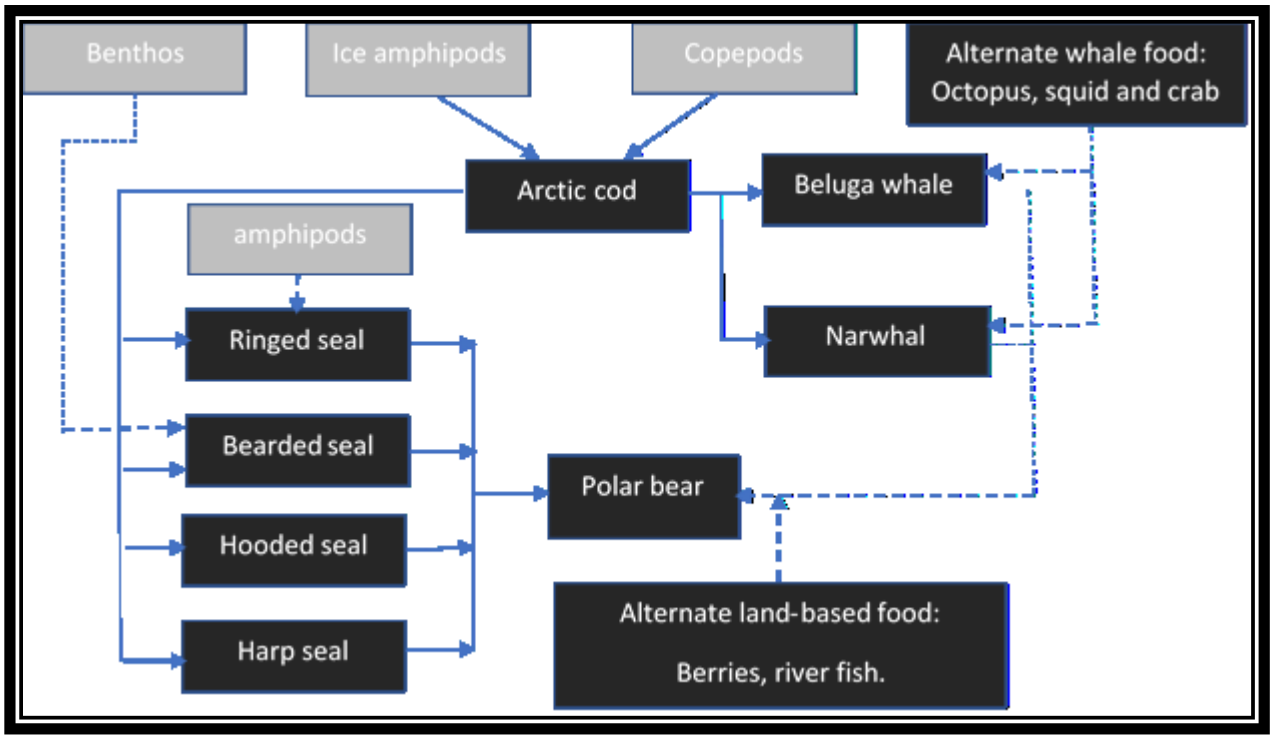


Figure 19: Arctic marine food chain considered.

(Note: Dashed line represents alternate food options. Grey colored boxes are out of the scope of the study but used to present the completeness of the food web).

Figure 20 presents the framework of the model developed in this study. Oil spill conditions and environmental conditions are used as model input to check for the immediate risk of an oil spill to

the Arctic animal species. To measure the indirect risk (i.e., from changing prey availability), prey requirement and availability are estimated for each trophic level in the food web based on their annual energy requirements. The species in the higher trophic level are assessed form immediate risk and impact on their food abundance.

The direct and indirect risk from oil spill exposure is defined as:

Risk (Direct oil spill exposure) = f (exposure, susceptibility)
Risk (Direct and indirect) = f (exposure, susceptibility, change in food abundance)
The changes in the food availability along with the acute impact of oil spills for apex Arctic mammals are modeled using a Bayesian network (BN).

Two separate BNs were developed for the two tiers of the study. The first tier assesses the impact of oil spill using the current stock of polar cod in the hypothetical spill area of around Svalbard Island. The second tier of the study assesses the predicted recruitment in the fish stock due to oil spill exposure and evaluates the impact on the apex predators. The adult polar cod mortality to oil spill scenarios was estimated using the results of the BN model of Fahd et al. (2019 and 2020). The probability of acute impact to polar bears and whales, i.e., the direct risk from oil spill exposure, was estimated and modified for current scenarios based on expert opinions from Nevalinen et al. (2017) and Helle et al. (2020). Assumptions were used by the researchers to approximate for scenarios considered; with new information on animal behaviour, the probability tables can be updated in the BN model.

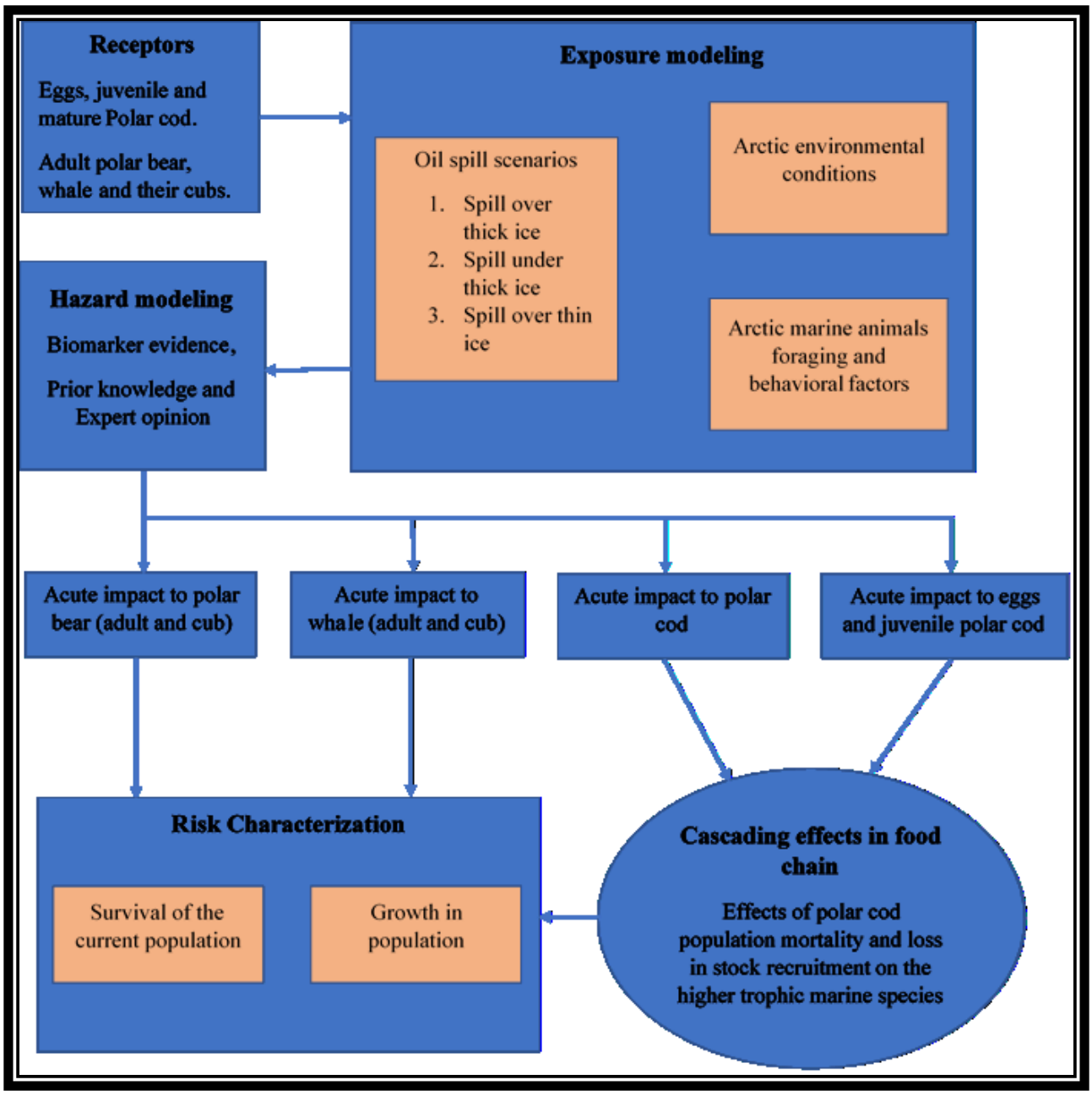


Figure 20: Framework of the survival and population growth BN model for Arctic marine mammals

5.3 Case study

5.3.1 Geographical context of the study

A hypothetical spill is assumed near Svalbard. As shown in Figure 19, polar cod is a keystone Arctic species of fish and serves as the primary energy source to many of the top predators in the region (Gallaway et al., 2017). Previous studies have investigated the impact of sea ice and

recruitment in the polar cod stock (Huserbraten et al. 2019). The Arctic cod eggs and larvae in the Barents Sea region drift with the ocean currents to the spawning assemblages around the Svalbard Island as modelled by Huserbraten et al. (2019). A spill around the spawning assemblages can prove detrimental to the recruitment of polar cod stock. The hypothetical spill area is shown in Figure 21. The line in Figure 21 represents the spill along the coast, while '+' in the figure represents the spawning assemblages around Svalbard. The area of a hypothetical spill is selected around the spawning assemblages near Svalbard Island to model the worst-case scenario.

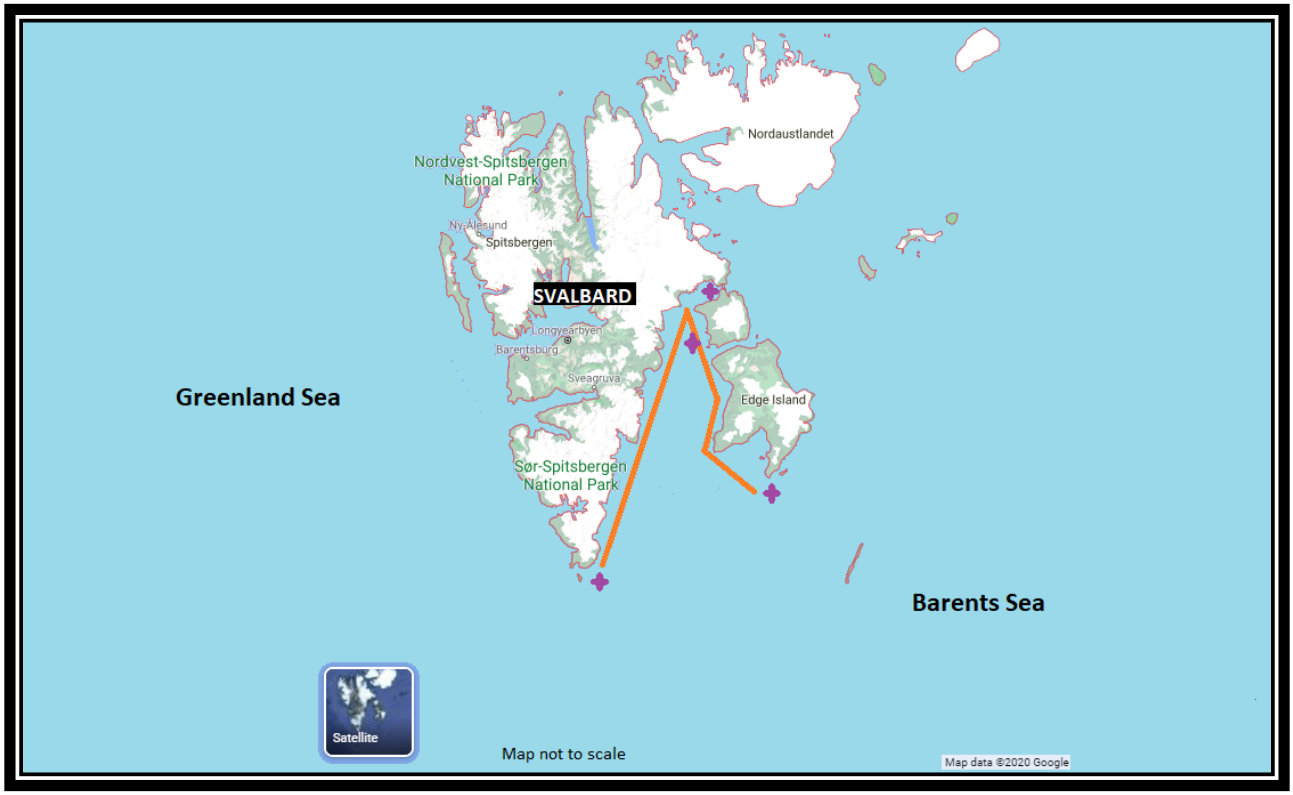


Figure 21: Hypothetical spill area assumed for the study (Source: Google map)

5.3.2 Hypothetical spill conditions

The spill area is selected as the coastline of Svalbard Island, as shown in Figure 21. The length of the coastline along the spill was calculated to be about 780 km using QGIS software. The hypothetical spill conditions, such as a spill volume of 42,000 m^3 of crude oil, in the current research were like those assumed by Helle et al. (2020). The PAH concentration in the hypothetical

spill was determined based on the experiments conducted by Nahrgang et al. (2009, 2010, 2016, 2019) wherein filtered seawater was passed through crude oil laced rock columns into the tanks holding polar cod. The crude oil spill simulation used in the experiments was 3, 6, and 12 g crude oil kg^{-1} gravel corresponding to low, medium, and high treatments. The corresponding total PAH concentrations calculated for each treatment were 15 µg/L, 18 µg/L, and 40 µg/L PAH concentration in water, respectively. Establishing a context in terms of the field oil spill for the considered exposure concentrations in this chapter, the spill conditions leading to such a PAH concentration in the ocean are 15000 tonnes, 18000 tonnes, and 40,000 tonnes for low, medium, and high PAH concentrations, assuming 3.9% weight of PAH in crude oil (Huesemann et al. 2002). This case study estimates the probabilities of acute impact or mortality to the polar bears and whales for spills in different seasons based on the probabilities reported by Helle et al. (2020).

Apart from the spill size (in PAH concentration), the initial spill scenarios considered in the study relate to sea ice and season. These factors also play an essential role in the life cycles of marine species such as polar cod, seals, polar bears, and whales.

The sea ice scenarios considered were as follows:

- Spill over thick ice
- Spill under thick ice
- Spill over thin ice

5.3.3 Developing the Bayesian network (BN) model for Arctic marine species

5.3.3.1 The model

The BN is developed for the spill scenarios based on Figure 19 and Figure 20 for the current stock of fish (Tier 1) and shown in Figure *22*. The Bayesian Network for recruitment fish stock (Tier 2)

is presented in Figure 23. The current stock refers to the quantity of polar cod inhabiting the area around the Svalbard Island. The polar cod spawn only once in their lifetime; hence the recruitment stock refers to the quantity of polar cod hatched from the current stock and inhabiting the region after the spill.

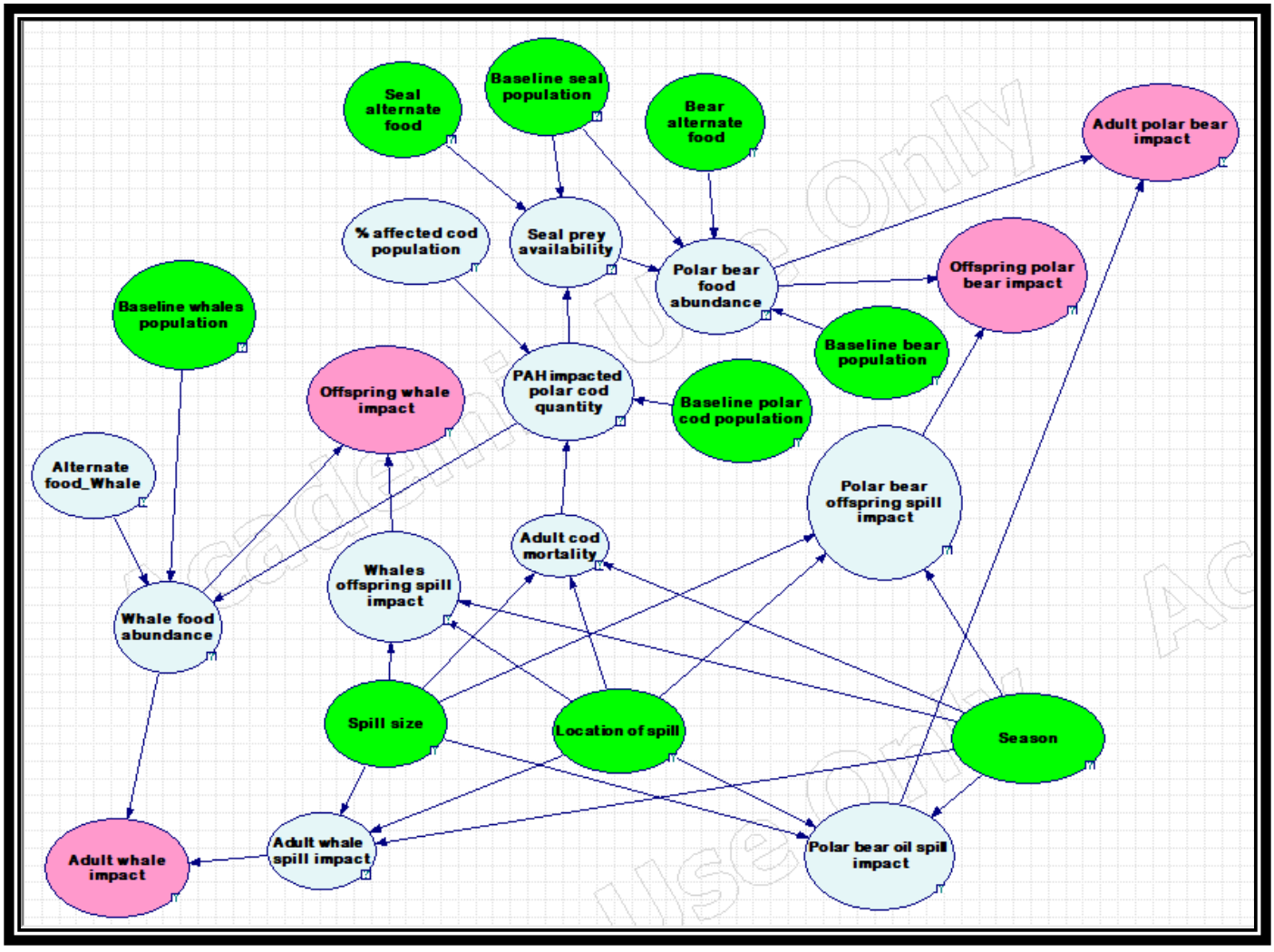


Figure 22:BN for the apex marine mammals oil spill impact using current fish stock

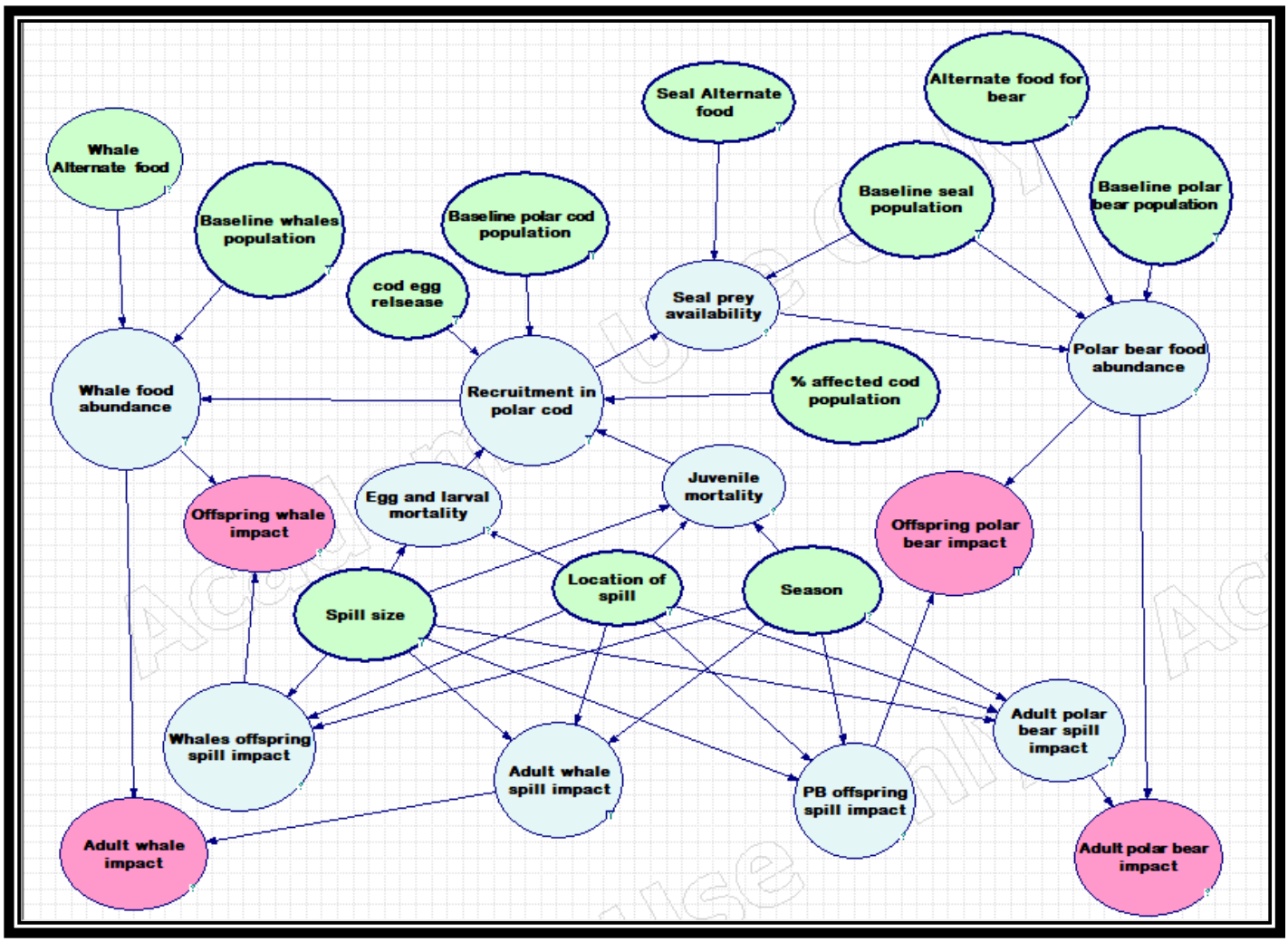


Figure 23: BN for the apex marine mammals oil spill impact using recruitment fish stock

(Note:The teal colored nodes are the parent nodes or nodes representing initial spill conditions, environmental conditions and animal behavioural conditions. Blue colored nodes are intermediate nodes and pink colored nodes present the outcome of the model.)

5.3.3.2 Data discretization and parametrization for the model

Data for the BN risk model for the apex marine species is based on expert opinion, available literature studies, and some assumptions. The expert opinion is based on the questionnaire prepared by Helle et al. (2020) and developed from the opinions of over 20 experts.

The nodes in the network shown in Figure 22 and Figure 23 could have two or more states. The discretization of the data refers to converting the continuous data sorted into different intervals

or ordinal groups defining the states of a node in the network. The states of the BN nodes are based on information on the lifecycle, habitat, and feeding behaviours of the species considered. Parameterisation refers to adding values to each state of the nodes in the network. The animal lifecycle, behavioural, and fecundity data is discussed below. Discretization and parametrization for each node in the network are accomplished based on these data. The CPTs for each of the nodes are detailed in the Supplementary data document.

Polar cod

Polar cod is a fish associated with cold sub-zero Arctic waters. Arctic cod is a small fish with lengths up to 300 mm and in some cases, up to 460mm have been recorded. Arctic cod is the most abundant and circumpolar distributed fish in the region. The polar cod plays a major role in the energy transfer in the Arctic food web by transferring the energy from the planktons to the apex marine mammals (Steiner et al. 2019, Parker-Stetter et al., 2011). The Arctic cod act as a high-energy prey, due to their high lipid content, for the upper trophic levels in the Arctic food web. The polar cod is a major food source of marine mammals such as the ringed seals, narwhals and beluga whales (Hop and Gjosaeter 2013). The standing biomass of the Arctic cod in the Barents Sea varies between 0.5-1.5 million tonnes (Hop and Gjosaeter 2013). From 1986 to 2016, the yearly variations in the polar cod stock in the Barents Sea region is provided by MOSJ (2019). The Arctic cod has a life span of 7 years, with maturity at about 3 years. The Arctic cod spawns only once during its lifetime (FAO, 2015). While there are fish stocks in the Barents Sea area, the Svalbard region is identified as one of the spawning assemblages for the Arctic cod. The spawning usually occurs in January and February, with an incubation period varying between 30 to 60 days (FAO, 2015). The relationship of the Arctic cod with sea ice is significant. Polar cod mainly feeds on the amphipods. Ice algal bloom causes an increase in amphipods, which feed on them. Although

the Arctic cod are present in the ice-covered areas of the ocean, only the larval and juvenile stages of the fish are directly associated with the ice for food and protection.

The node 'Baseline Arctic cod' has an interval of 0.3 million tonnes of fish stock between its states, which vary from 0.5 to 1.5 million tonnes. The relative frequency of occurrence, i.e., probability, for each of the states of the node 'Baseline Arctic cod' is calculated based on MOSJ (2019). The first tier of apex mammals risk assessment is accomplished using the oil spill induced mortality in the current stock of Arctic cod, as shown in node 'Adult cod mortality'. The prior probabilities for the node are influenced by the season, size, and location of the spill. These probabilities are obtained from the results of Arctic cod toxicity modeling by Fahd et al. (2020). The second tier of apex marine mammals' assessment combines the probabilities of 'Egg and larval mortality' and 'Juvenile cod mortality' to determine the recruitment in the Arctic cod population and subsequent risk to apex mammals. The 'Egg and larval mortality' and 'Juvenile cod mortality' nodes are assigned the following states: baseline, low, medium, and high mortalities.

The baseline mean of instantaneous mortality rates in eggs and larval stages of northeast Arctic cod (Gadus morhua) were estimated to 0.17 d^{-1}, with a 95% confidence interval between 0.15-0.19 d^{-1} (Langangen et al. 2014). An instantaneous mortality rate of 0.19 d^{-1} was used for Arctic cod (Boreogadus saida) in this study. Nahrgang et al. (2016) conducted experiments on the eggs and larvae hatching and survival for 3g/kg gravel (low spill concentration) was 39% and for 6 g/kg gravel (Medium spill concentration) was 24%. The experiment by Nahrgang et al. (2016) did not investigate the effects of what could be considered as high spill concentration, which is 12 g/kg gravel crude oil. An assumption of 10% survival for 12g/kg gravel of crude oil was made for this study. Based on the experimental data (Nahrgang et al. 2016), instantaneous mortality rates in

Arctic cod eggs and larvae for low, medium, and high spill concentration were established as 0.204, 0.217, and 0.230 d^{-1}. The baseline mortality rate for juvenile cod is reported as 0.009 d^{-1} (Gallaway et al. 2017). Based on the experimental data from Nahrgang et al. (2016), the mortality rates for juvenile Arctic cod are estimated to be 0.015, 0.03, and 0.04 d^{-1} for low, med, and high states of the node 'Juvenile mortality'.

Seals

Six species of seals live in the Arctic, namely, ringed, bearded, spotted, hooded, harp, and ribbon seals. As denoted by the node 'Baseline seal quantity', the seal population considered in this study varied between 0.5 million, 0.75 million, and 0.9 million for low, medium, and high states of the node (Laidre et al. 2015). The seal population was estimated based on the population studies from the Barents Sea. In the absence of such data, the populations of seal species in the Greenland Sea were used (Laidre et al. 2015). The birthing season for the seals is in spring, ranging from February to April (NSIDC, 2020). Some seal species depend entirely on the sea ice for survival. Many seals birth their offspring on ice and nurse them on ice around the breathing holes; the seals forage for food along the ice edge and under the ice for fish such as polar cod and shrimp (NSIDC, 2020). Therefore, changing ice conditions, especially in spring could impact the presence of the seals in the region and subsequently also impact the food availability of polar bears in the region. The conditional probabilities of 'seal prey availability' and 'polar bear food abundance' is detailed in the supplementary data document. Understanding the role of the predator in the ecosystem depends on identifying and quantifying its diet (Ryg and Ortisland 1991). The annual energy budget of the ringed seals was calculated by Ryg and Ortisland (1991) and it was observed that the food consumption rates varied seasonally. The energy requirements calculated by Ryg and Ortisland (1991) considered the maintenance, growth, and feeding of the offspring. The annual gross energy

consumption of the females exceeds the energy consumption of the male seals in the experiments. It was also observed that average energy consumption was three times the energy required to basal metabolic rate (BMR). The average consumption per individual in the seal population was calculated to be 4.6×10^9 joules gross energy per year (Ryg and Ortisland, 1991). Assuming the 1 kg of fish to produce 810 kJoules gross energy (Dyck and Kebreab, 2009), the quantity of fish consumption for the seal population is estimated. This data is further used to obtain the probabilities of the node 'Seal prey availability'.

Polar bear

Polar bear is an apex predator in the Arctic marine food web. Polar bears birth their cubs in winter, mostly in December-January. Polar bears primarily prey on ringed seals and bearded seals resting on the sea ice. Polar bears turn hyperphagic in spring when there is plenty of young seal pups (Dyck and Kebreab, 2009). In the regions where little to no sea ice is present in the summer, polar bears prefer to spend the time onshore foraging for land-based food sources. These include berries and fruits, some nesting birds and eggs, small land animals, and river fishes in some cases (Dyck and Kebreab, 2009). The energy requirements and budget of the polar bear with different body masses were studied using three diets: berries, Arctic charr, and seal. The energy budget calculations assumed that the polar bears were restricted to land. The gross energy content from ringed seal raw blubber was calculated to be 34,430 kJ per 1 kg. The diet (kg) required to cover the daily energy loss was calculated for polar bears with masses varying from 100 kg to 650 kg. The data was used to predict the probabilities in the nodes 'Polar bear food abundance'. A 500 kg polar bear would need to consume 1 kg of seal blubber or 4 kg of fish to maintain its body mass (Dyck and Kebreab, 2009). Hilderbrand et al. (1999) reported that captive brown bears consumed an average of 10.8 kg of fish per day and estimated that a polar bear of up to 650 kg would have

energy surplus and gain mass in such a scenario. The polar bear population in the Barents Sea is estimated to be about 2644, with a 95% confidence interval between 1899-3592 (Laidre et al. 2015). The states of the node 'Baseline polar bear quantity' are defined based on the population data from the Barents Sea.

Whales

Three whale species, Delphinapterus leucas (Beluga whale), Monodon Monoceros (Narwhal), and Balaena mysticetus (bowhead whale), are endemic to the Arctic region all year. Beluga whales are the most abundant whales in the Arctic waters and only they were considered in this study (Kastelein et al., 1994). The lifespans of the belugas range from 15 to 30 years; they attain sexual maturity at the age of 5-7. Belugas give birth every three years on average. The habitat of beluga whales varies seasonally. As sea ice breaks up, beluga whales swim along the ice edges and also penetrate the leads. When the sea ice becomes sparse or disappears during summer, belugas are found along the coastline and in shallow waters and river estuaries. In autumn months, they move to locations of feed in deep waters. In winter, they prefer the sea ice areas. From mid-August, the belugas move back to the deep waters. The belugas were observed to be in polynyas and loose pack ice. The aerial survey also observed that the belugas preferred ice cover of 4/10 to 8/10 concentration. Barber et al. (2001) observed that the belugas avoided ice cover of 10/10 concentration. Belugas have the lowest body fat content in summer. In late summer, intensive feeding increases their blubber content. Arctic cod is the main diet of belugas along with other fishes such as capelin and saffron cod. The amount of food consumption depends on the sex, sexual activeness and age group of the belugas. Kastelein et al. (1994) found belugas of about 200 kg ate around 4.5% of their body weight. While the belugas around 1400 kg ate 1.2% of the body weight. Calving time for belugas could occur in late spring or early summer. The peak of the calving season

is observed to be mid-June to early July. The information on the relationship between the whale species and sea ice was used to develop the CPTs for the nodes 'Adult whale spill impact' and 'Whale offspring spill impact' nodes.

5.4 Results and discussion

5.4.1 Oil spill initial conditions

Sea ice is a ubiquitous geophysical feature in the Arctic region with a crucial role in the foraging, resting, and breeding behaviors of marine mammals. The spill scenarios were selected to reflect several possibilities of spill in ice infested waters, such as spill over and under thick sea ice and spill over thin ice. The oil spill release quantities considered in this study are 15000, 18000, and 40000 tonnes of crude oil in the polar cod spawning areas around Svalbard Island. The results from all scenarios are presented in the supplementary data document. The best- and worst-case scenarios of each of the species of food web are discussed in this section.

5.4.2 Impact on the current polar cod stock

It is reasonable to assume that a percentage of the fish stock in the region was exposed to the spill instead of all the fish stock in the region. The node 'fish stock affected' in the model has probabilities assigned showing 99% of the probability the spill causes less than 20% of the stock to be affected. The mortality in exposed adult cod was lowest when the spill occurred over thick ice and for the low spill volume and was highest when the spill occurred under the ice. However, for the given best and worst scenarios for polar cod, the change in its population and subsequently the increased risk was not drastic.

5.4.3 Recruitment stock impact

The significant factors affecting the recruitment are average fecundity in each female cod, mortality in egg and larval stages, and juvenile cod mortality. The spill scenario causing the least

mortality in juveniles and eggs/larvae occurs for the spill over thick ice, and the scenario with the highest risk is when the spill is under the ice. A variation in the average fecundity from low (9000 eggs per female) to high (25000 eggs per female) causes a significant increase in the recruitment stock. Low eggs per female resulted in lower fish stock (in million kg) than the baseline stock, while medium fecundity resulted in increased fish stock compared to the baseline fish stock. An average of 25000 eggs per female increases the stock from 500 million kg to 2100 million kg. Such drastic changes in the fish have previously been reported in the region of study. Refer to the supplementary data document for the baseline stock variation in the last two decades.

5.4.4 Risk to apex predators

The species in the higher trophic level are assessed for direct risk and impact on their food abundance. The conditional probabilities for the direct risk, given spill size, location, and season, were based on expert opinions and assumptions. The risk from only the oil spill is termed as the baseline risk. This case study also aims to investigate the additional risk due to changes in the prey availability in the food web.

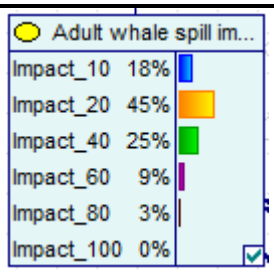
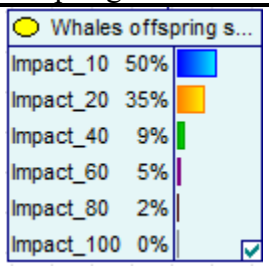
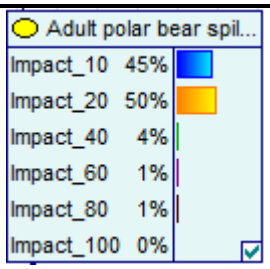
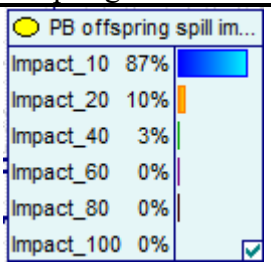
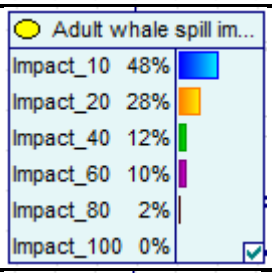
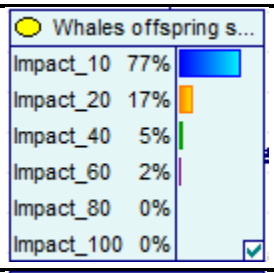
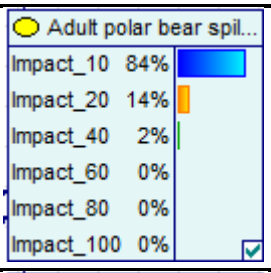
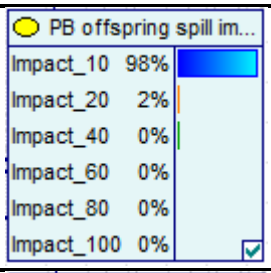
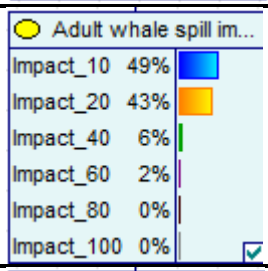
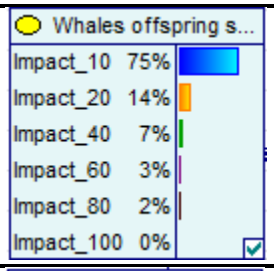
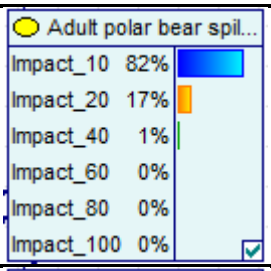
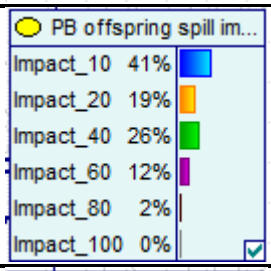
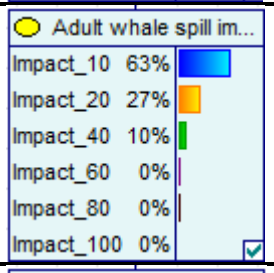
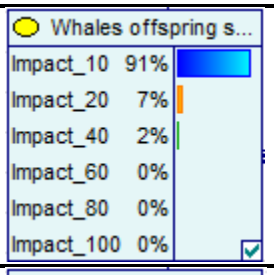
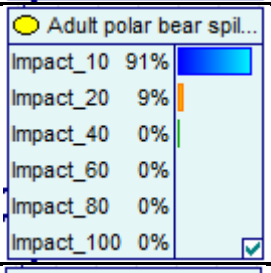
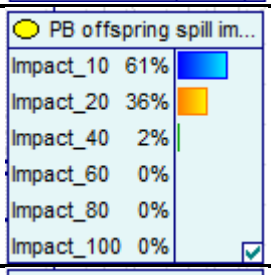
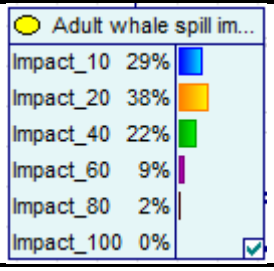
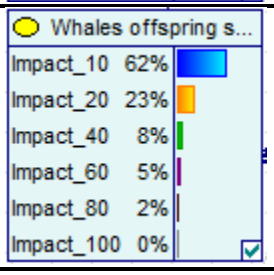
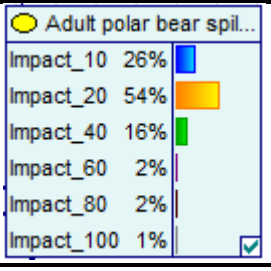
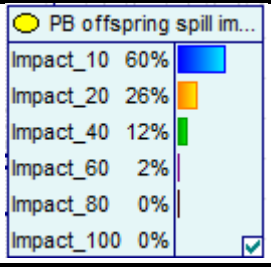
5.4.4.1 Direct risk/baseline from oil spill

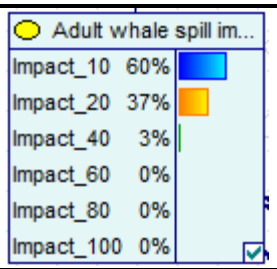
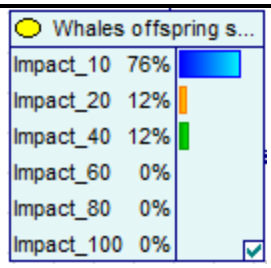
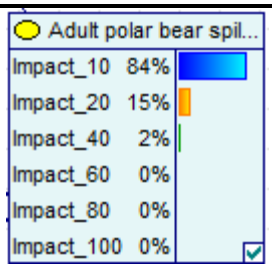
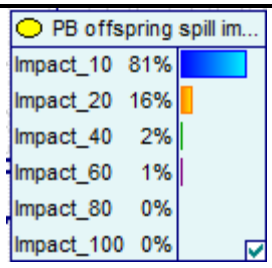
Impact on whales

The baseline risk from the oil spill for whales is presented in Table *11*. The worst-case scenario for both the adult whale species and their offspring is a high-volume spill under thick ice. The next worse case is a spill over thin ice. The scenario of spill over thick ice showed the least baseline risk in the whale species. Comparing the baseline risks in the adults and offspring shows that the predicted risk for adult whale species for all the spill scenarios was higher. Beluga whales are known to inhabit the areas of thin and thick ice as well. The risk from spill over thick ice, although less compared to other scenarios, could be due to the frequent visits to the breathing holes by whale

species. As the whale forage for food under the ice, the spill under thick ice causes the highest exposure and, subsequently, higher risk to both the adults and offspring.

Table 11: Baseline risk from oil spill for whales

Spill size	Spill location	Risk to Adult whales	Risk to whale offspring	Risk to polar bear	Risk to polar bear offspring
High	Under ice				
Low	Under ice				
High	Over thick ice				
Low	Over thick ice				
High	Over thin ice				

Low	Over thin ice				

Impact on Polar bear

Direct risk assessed for adult polar bears indicated the worst-case scenario is a high volume of spill over thin ice, followed by a spill under thick ice, and lastly, the scenario of a spill over thick ice. The scenarios posing a higher direct risk to polar bear offspring are spill over thick ice, followed by a spill over thin ice, and finally a spill under thick ice. It is estimated that adult polar bears would avoid the spill over the thick ice; however, they can be exposed to a spill under the ice due to their hunting habitat around the ice edge. It is also observed that the polar bears prefer thick ice as habitation and hunt the seals on thin ice floes. Based on such observations, the risk from a spill on thin ice is predicted to be higher than a spill under the ice. The polar bear cubs are housed in the ice caves on thick sea ice. Polar bear cubs' behaviour in their habitat could potentially expose them to a spill over thick sea ice. Since the cubs do not hunt seals along the ice edge, the scenario of spill under the ice is predicted to be of least risk.

5.4.4.2 Indirect risk from cascading effects of oil spill

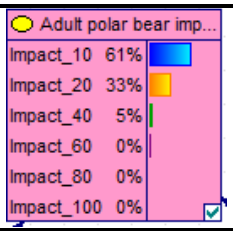
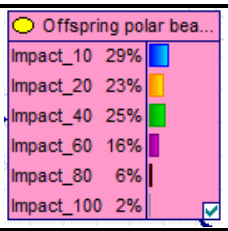
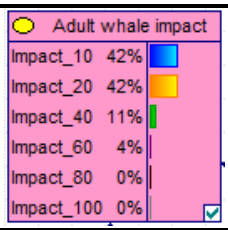
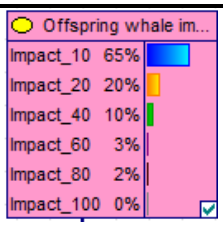
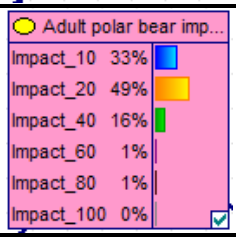
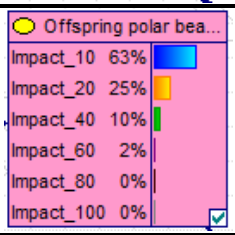
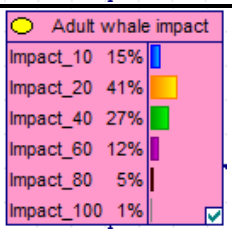
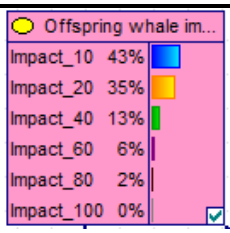
The indirect risk from the synergistic effects of decreasing food availability with the risk from oil spill exposure follows similar trends as the direct risk discussed previously, albeit with higher risk probability. Table *12* presents the indirect risk for the high-volume crude oil spill with varying spill locations on the sea ice. The CPTs for these outcome nodes (adult polar bear impact, polar

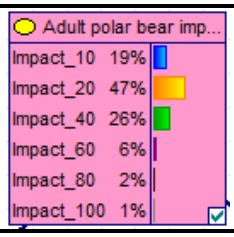
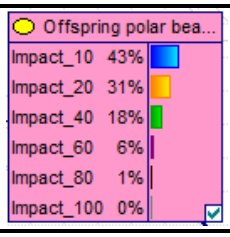
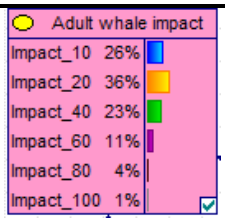
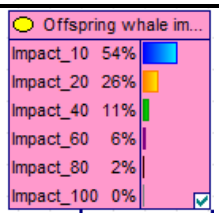
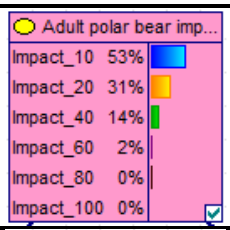
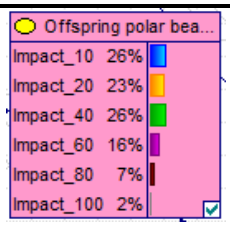
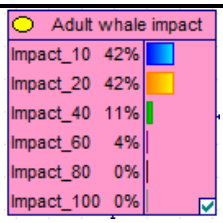
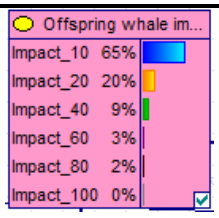
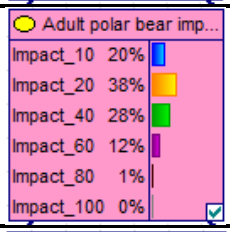
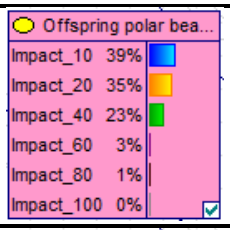
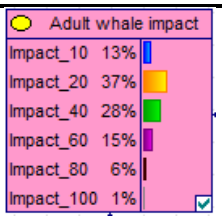
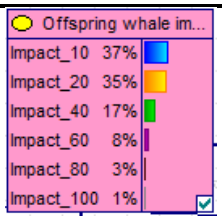
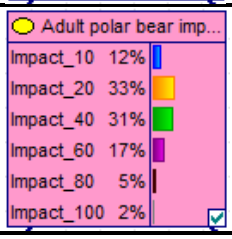
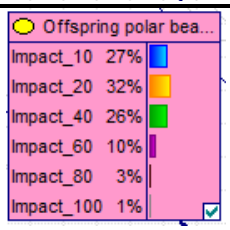
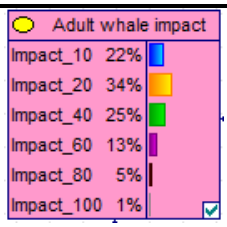
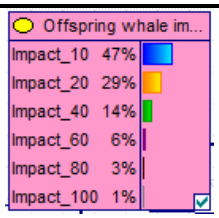
bear offspring impact, adult whale impact, and whale offspring impact) are presented in the supplementary data document.

Using the Tier 1 of the BN toxicity model, the risk to adult and offspring polar bear and whales was estimated to be higher than the risk from the baseline oil spill scenario. In Tier 2 of the toxicity model, the risk to adult and offspring apex marine species was even higher than the risk estimated from the Tier 1 model (Table 12).

The probability of the polar bear food availability to be lower than or up to minimum maintenance was estimated to be 54% for current stock. The same probability was 75% for recruitment stock. Subsequent mortality risks to polar bear cubs were predicted to be higher in the Tier 2 recruitment model. However, the risk for the adult polar bear showed a marginal increase in risk for the recruitment model.

Table 12: Additional risk from cascading effects of the oil spill in food chain

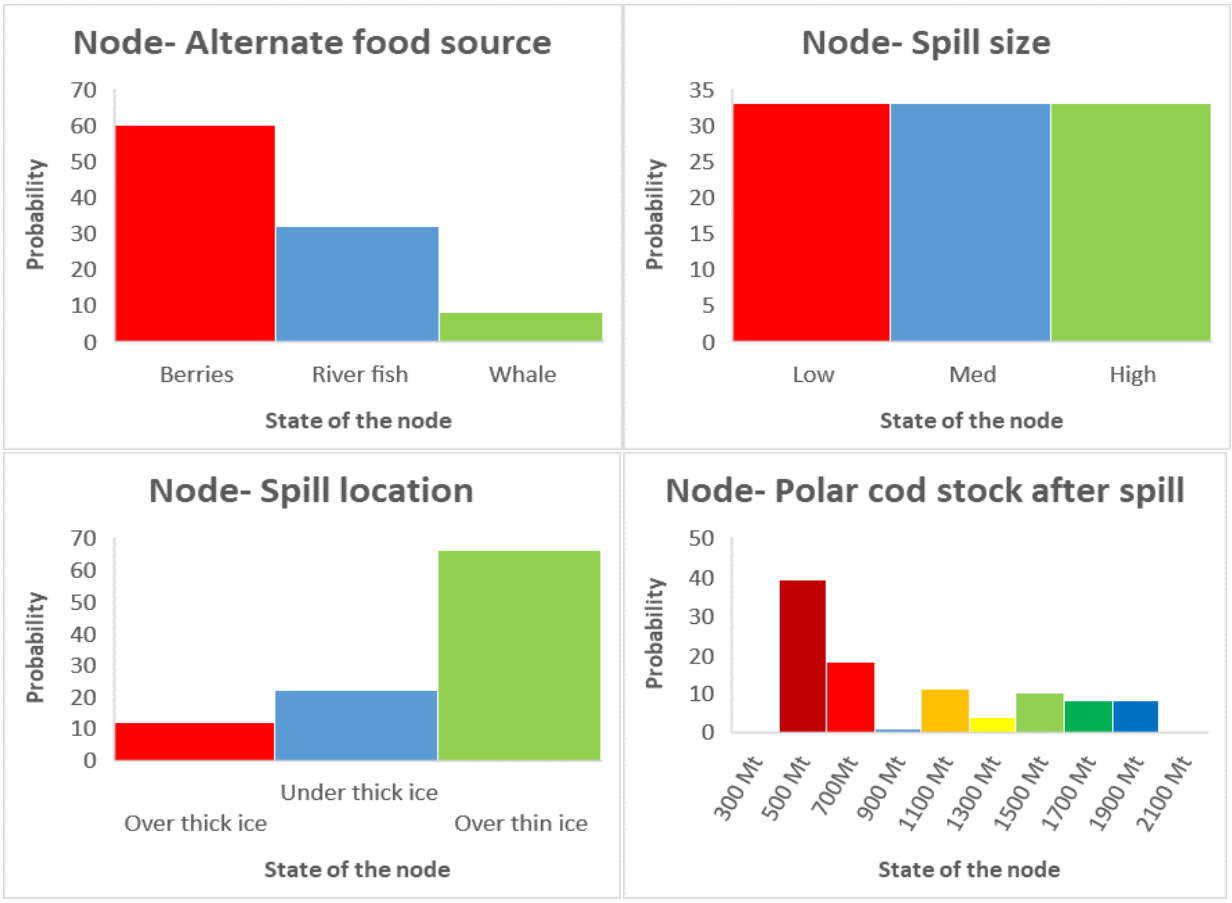
Tier 1					
Spill location	Spill size	Risk to adult polar bear	Risk to polar bear offspring	Risk to adult whales	Risk to whale offspring
Over thick ice	High				
Under ice	High				

Over thin ice	High				
Tier 2					
Spill location	Spill size	Risk to adult polar bear	Risk to polar bear offspring	Risk to adult whales	Risk to whale offspring
Over thick ice	High				
Under ice	High				
Over thin ice	High				

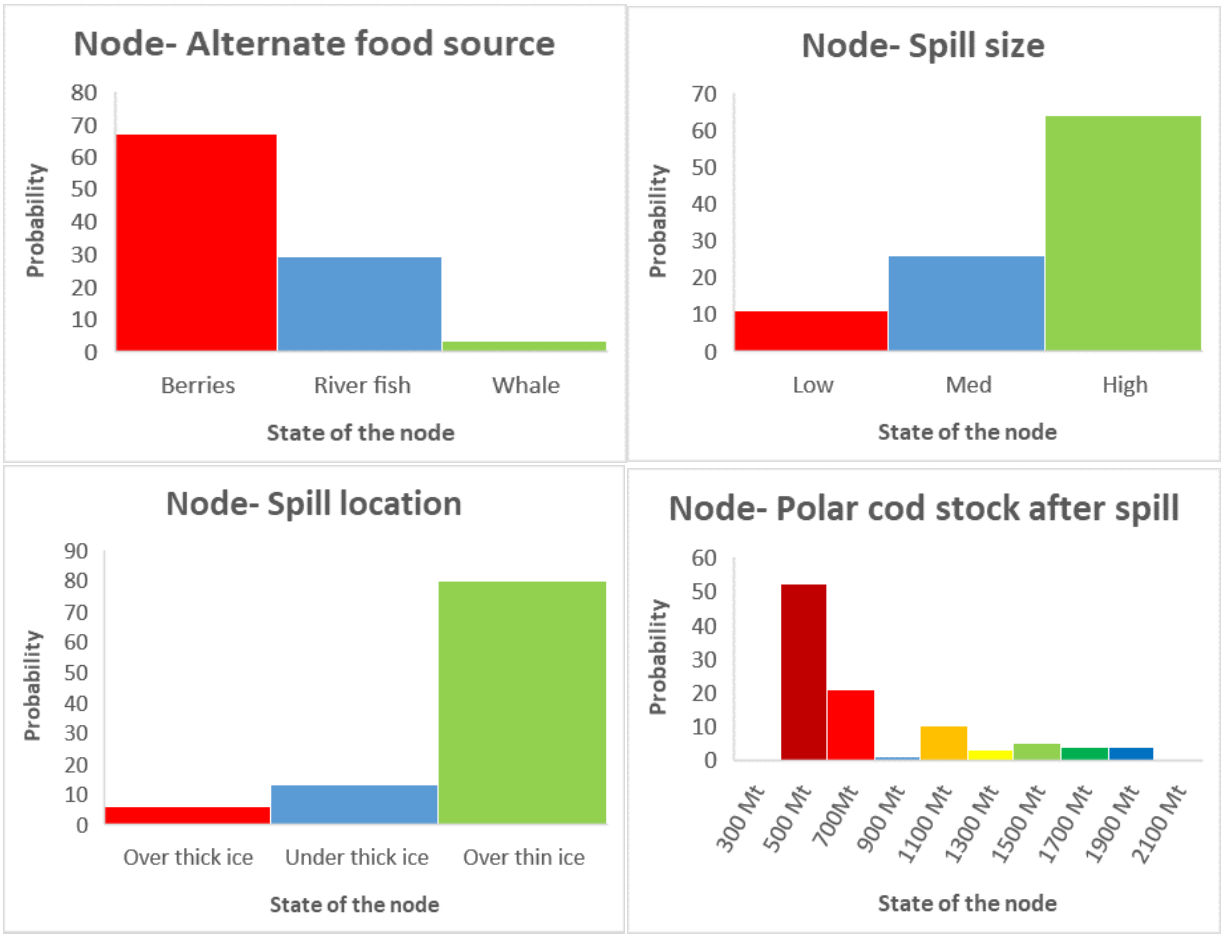
5.4.4.3 Sensitivity analysis

The sensitivity analysis is performed using the software developed by Bayesfusion (Bayesfusion, n.d). The factors affecting the probability of risk to apex predators are the presence of sea ice, season, quantity of Arctic cod, alternate food availability, and location of the spill with respect to sea ice. The sensitivity of the outcome nodes ‘adult whale impact’ and ‘whale offspring impact’ shows that the nodes that have maximum effect on the outcome are ‘whale food abundance’, ‘spill size’, and ‘location of spill’. To further study the relationship between these sensitive factors and risk, the BN model was set with 50% risk to target species as evidence. The resulting changes in

probabilities, i.e., posterior probability of the sensitive nodes was back calculated by the BN model as shown in Figure 24 and Figure 25.

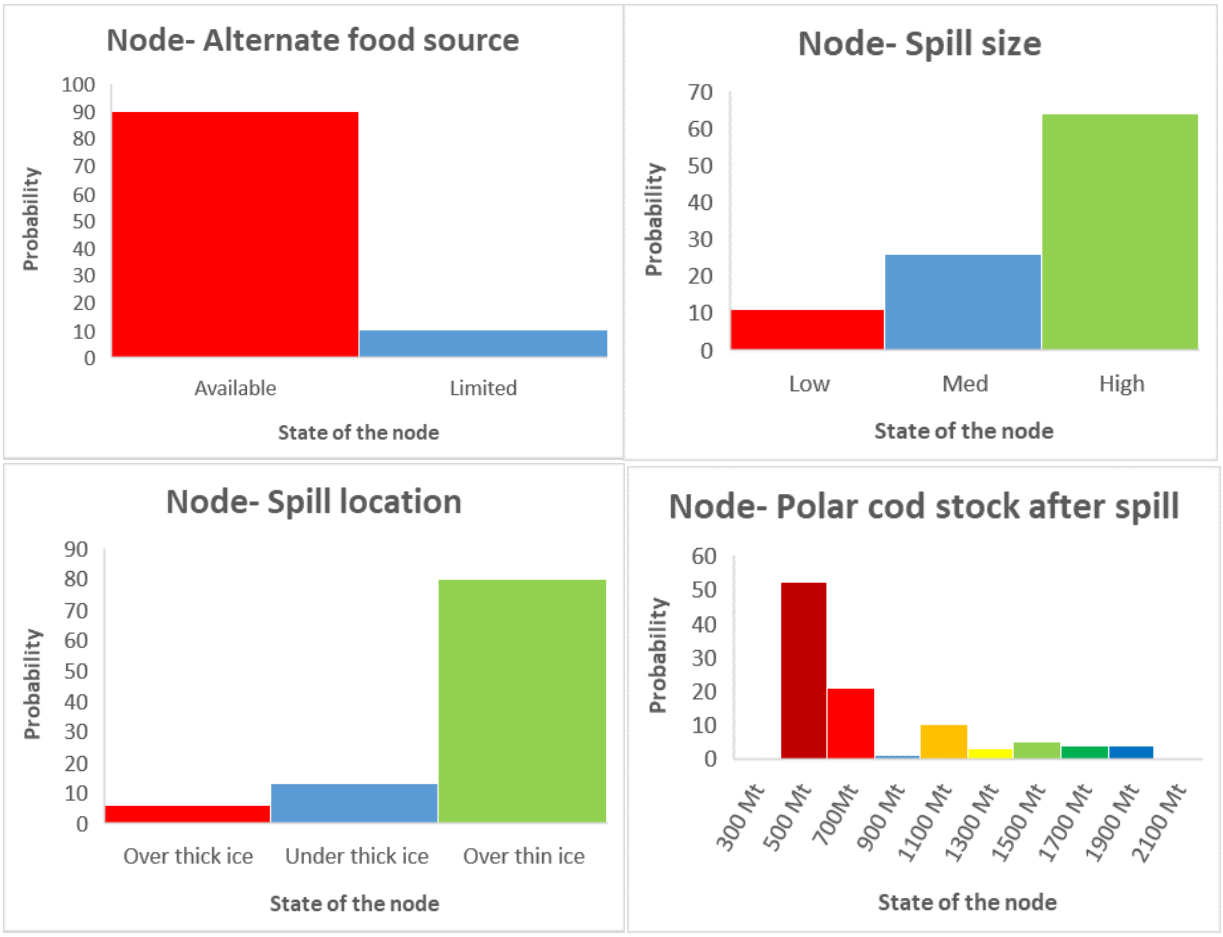


(A)

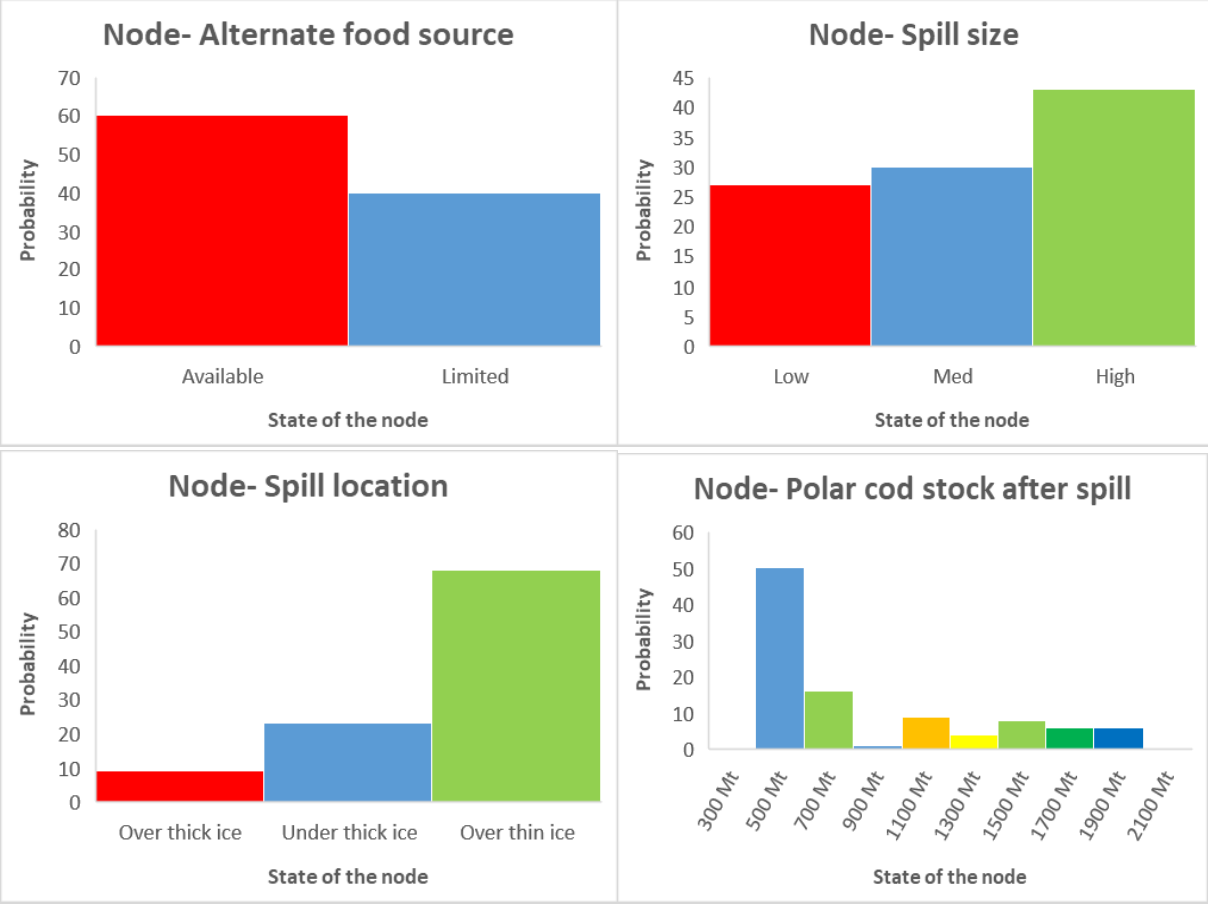


(B)

Figure 24:(A) The prior probabilities of sensitive nodes for polar bear; (B) Posterior probabilities of sensitive nodes when the 50% of polar bear population at risk is set as evidence


Node- Alternate food source
Probability
100
90
80
70
60
50
40
30
20
10
0
Available
Limited
State of the node
Node- Spill size
Probability
70
60
50
40
30
20
10
0
Low
Med
High
State of the node
Node- Spill location
Probability
90
80
70
60
50
40
30
20
10
0
Over thick ice
Under thick ice
Over thin ice
State of the node
Node- Polar cod stock after spill
Probability
60
50
40
30
20
10
0
300 Mt
500 Mt
700Mt
900 Mt
1100 Mt
1300 Mt
1500 Mt
1700 Mt
1900 Mt
2100 Mt
State of the node

(A)



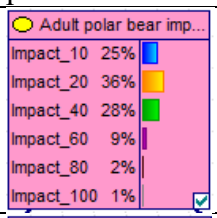
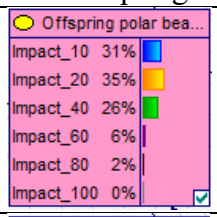
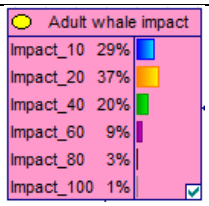
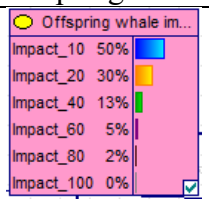
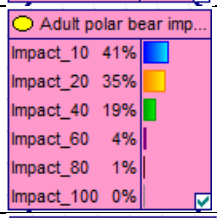
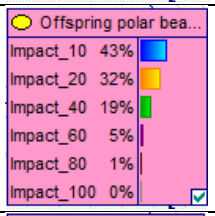
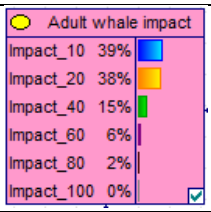
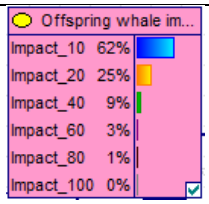
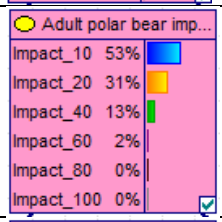
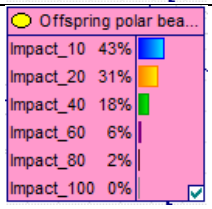
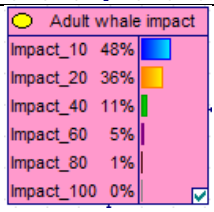
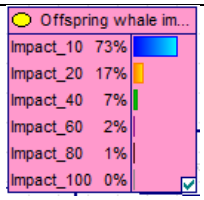
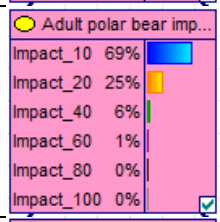
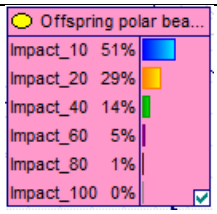
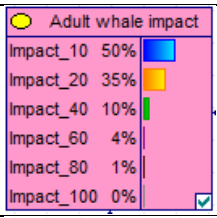
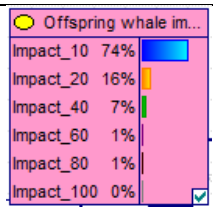
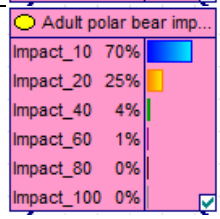
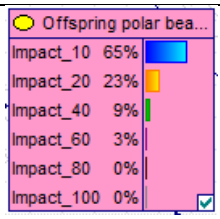
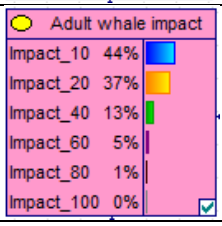
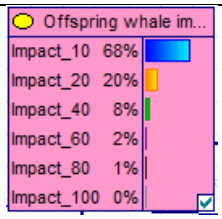
(B)

Figure 25:(A) The prior probabilities of sensitive nodes for whales; (B) Posterior probabilities of sensitive nodes when the 50% of whale population at risk is set as evidence.

Sensitivity analysis of the risk to the polar bears and their offspring shows that the kind of prey availability and quantity of food availability plays a significant role in determining risk. The location of the spill is also a factor in determining the total risk to polar bears. Comparing the risk sensitivity of the apex predators shows that the food availability node has a greater influence on the risk of polar bears than whales. This observation further emphasizes the importance of seals availability to polar bear survival.

To observe the sensitivity of the fish stock's presence to the apex predators, the node ‘stock after recruitment’ was set to 300 million tonnes and 1900 million tonnes, and the results were compared. Lower fish stock elicited a higher risk of mortality to polar bears. The risk increased significantly for polar bear offspring, as presented in Table *13*.

Table 13: Effects of varying fish stock on the risk to apex predators

Fish stock (million tonnes)	Risk to adult polar bear	Risk to polar bear offspring	Risk to whales	Risk to whale offspring
300				
700				
1100				
1500				
1900				

5.5 Conclusions

This study develops an approach to quantitatively assess the combined impacts of oil spill impacts and cascading effects on the food web. A comprehensive insight into the impacts in the region could facilitate the identification of significant lower trophic species, and enhanced conservation methods for apex marine predators.

Notable findings from this study are:

1. A spill at an Arctic cod spawning assemblage could lead to recruitment collapse in fish stock and subsequent increased risk to polar bears and whale species.
2. The non-availability of food to apex predators or imbalance in the food web could lead to drastic changes in the survival of polar bears and whale species. Amongst the apex predators, the effect on polar bear could be more devastating than whale species.
3. The risk to survival for whale and polar bear offspring is lower than their adults when cascading effects in the food web are taken to account.

Limitations of the study:

The probabilities considered for the nodes did not take gender of the polar bears into account. The sex of polar bears determines different exposure probabilities based on hunting cub rearing activities. The probabilities used for various nodes were based on literature or expert opinions, however, some assumptions were also made of the conditional probabilities in this study. The advantage of using a BN model is that with the availability of new information, these probabilities could be easily adjusted in the model to generate new risk probabilities in apex predators.

Chapter 6: Conclusions and recommendations

6.0 Conclusions

The proposed shipping activities through the Arctic region via the NSR could result in shorter travel distances. However, these shipping operations can open new environmental challenges for the region, especially since such operations coincide with high biological productivity time of spring and summer. Owing to the simple food web, any disturbance in the populations will have cascading effects on all the other species in the food web.

Most of the Arctic environmental risk studies so far had focused on modeling the oil spill fate and transport, and the remediation of contaminated waters, while a JIP by Arctic Oil Spill Response Technology, conducted experiments of oil transport in sea ice infested water. Few studies have focused on the mortality and ecotoxicological risk in marine species such as polar cod, polar bears, seals and whales. This thesis addresses an important question regarding the shipping operations in the Arctic region. Shipping operations in open water or navigable ice regions carry a real threat of an oil spill in the sensitive Arctic region. The purpose of this thesis was to investigate the risk to Arctic marine species using novel in-silico methods to model mortality in sentinel species. Use of in-silico methods circumvented the need to use the traditionally obtained No Observed Effect Concentration (NOEC). To achieve this goal, some important research questions that were addressed were regarding the unavailability of the toxicity data for the Arctic species and the need to develop novel in-silico methods for assessing toxicity in Arctic marine species. This thesis addressed the objectives laid down and produced the following research outcomes.

1. The suitability of the current ecotoxicological models for Arctic marine species was studied and presented here. A novel cellular metabolite activity and BN based model was proposed.
2. A novel in-silico method for ecotoxicological risk for polar cod was developed, and the risk to polar cod populations from various crude oil spill scenarios was estimated.
3. Moving a step further, the recruitment in a polar cod stock resulting from various spill scenarios was estimated and plugged in the food-web based risk model for apex marine species (polar bear and whales).

In the present study, the impacts of sea ice, extreme light regime, various polar region-specific physiological characteristics in polar cod and their effects on xenobiotic distribution and metabolism were studied. A Bayesian belief network was developed to model individual polar cod toxicity. The enzyme activity in the polar cod liver and other pertinent organs is used as a proxy for cellular damage and repair and is subsequently linked to toxicity in polar cod. Seasonal baseline variation in enzyme production is also taken into consideration. Three factors that significantly impacted the mortality risk in polar cod:

1. Presence of sea-ice and location of spill with respect to the ice.
2. Physiological factors responsible for biotransformation and excretion of the PAH reaching the target tissues.
3. Seasonal variation in background metabolic activity.

The polar cod risk model predicted lowest risk for the ‘Spill on thick ice’ and highest risk for the ‘spill under ice’ scenarios. The risk of mortality increased by over 10% for ‘spill under ice’ scenario than when the spill was over the thick ice. While presence of sea ice largely impacted the risk of mortality in polar cod, physiological factors in the polar cod also had significant impact on the probability of exposure and subsequent mortality. This physiological factor was the ability of

the polar cod liver to biotransform the PAHs reaching the target tissues. Biomarkers such as the EROD, CAT, GST, and GPx elicited the ability in the polar cod to biotransform and excrete the PAH in target tissues. When the biomarkers activity is the highest, the risk to polar cod decreased by about 18% for the worst case scenario of spill under ice.

While this thesis has focused on ecologic risk, it also identified and considered associated uncertainties. The main uncertainties identified include toxicological processes in the ecotoxicological modelling in marine species, and lack of validated data. The uncertainties in processes could lead to a different structure of the BN model and is a greater concern than the non-availability of validated data.

The process of biotransformation is very complex, and there are many pathways in biotransformation. However, the pathway catalyzed by CYP1A is selected in this study because this pathway is identified as a major contributor to biotransformation. Biomarkers for other biotransformation pathways can be added to the model structure when identified. Adding more biomarkers sensitive to phase I, phase II, and detoxification processes for a given chemical of concern to the model is expected to increase the accuracy of the results.

This study integrated the cellular responses to the PAH exposure, quantified as biomarker activity, to determine the toxicity risk. However, there is a possibility of a false negative response from biomarker activity owing to inhibition of cellular responses for any of the various reasons thus rendering the model less accurate. This issue could be resolved by inducting different biomarker

nodes alluding to a toxicity pathway in BN model thus reducing the uncertainty in risk number generated. The bioavailability and toxicity of oil are dependent on the type of oil constituents and its biodegradability, which are beyond the scope of this research. The model's biomarker data is based on very few biochemical studies; as more studies are conducted, the nodes' data should point to a more apparent trend in effects estimation in polar cod.

A novel approach of quantifying the prey availability across the food web and the additional stress it generates on top of the exposure to the oil spill is estimated. The risk to polar bears and whales is determined. The model predicted a recruitment collapse (for the scenarios considered), causing a higher risk of mortality of polar bears, beluga whales, and Narwhals in the Arctic region. Whales (adult and calves) were predicted to be at higher risk when the spill was under thick ice, while adult polar bears were at higher risk when the spill occurred on thin ice. A spill over the thick ice caused the least risk to whale and adult polar bears. The spill's timing and location have a significant impact on the animals in the Arctic region due to its unique sea ice dynamics, simple food web, and short periods of food abundance. The model can help resource managers project the changes in polar cod populations and their responses to future oil spills. In the event of a future oil spill, the model can be used to asses losses to the polar cod stock, apex marine species and the Arctic food web.

While most of the earlier studies focussed on the determining probability of exposure and risk to fish, polar bears and whales, no study had focussed on determining the ripple effects on such an oil spill on the prey availability and stress in the food chain in the Arctic region. This research adopted the data from previous experimental works on food and energy budget of the marine species and used that data to generate possible risk (survival and population growth) to the apex marine species. This novel approach gives comprehensive insight into the oil spill impacts in the

region, facilitate the identification of significant lower trophic species, and enhanced conservation methods for apex marine predators. Arctic Council, an intergovernmental forum, addresses the common concerns and challenges and enhances cooperation in the eight Arctic-rim states. The strategy adopted by the Arctic Council is published as the 'Arctic Environmental Protection Strategy' (AEPS), a multilateral and non-binding agreement among the eight Arctic states. This model could contribute to the Arctic environmental protection and serve as a comprehensive marine risk model from an oil spill. In future, more factors such as Arctic peoples hunting behaviors and yearly fish catch quantities could also be included in the BN model to get a more realistic impact on the apex marine species populations. Such studies could help in creating measured and regulated anthropogenic activities in this sensitive region.

The probabilities considered for the nodes did not take gender of the polar bears into account. The sex of polar bears determines different exposure probabilities based on hunting cub rearing activities. The probabilities used for various nodes were based on literature or expert opinions, however, some assumptions were also made of the conditional probabilities in this study. The advantage of using a BN model is that with the availability of new information, these probabilities could be easily adjusted in the model to generate new risk probabilities in apex predators. Other factors, such as translocation amongst the marine species, were also not considered in this study.

6.1 Recommendations for the research

Investigation of the pathways of toxification and detoxification in other Arctic marine species and the addition of more biomarkers as nodes in the BN model can create a more robust model for ecotoxicological risk determination of the Arctic marine species.

Additional factors that influence the stress on the food web-based BN model, such as translocation and divergence in food preferences, must be investigated to assess a complete and comprehensive understanding of the risk to marine species due to exposure from crude oil spills.

Reference

Afenyo M. K. (2017). Fate, transport and risk of potential accidental release of hydrocarbons during Arctic shipping. Doctoral thesis, Facykty of Engineering and Applied Science, Memorial University of Newfoundland.

Afenyo M., Khan F., Veitch B., and Yang M. (2017). Arctic shipping accident scenario analysis using Bayesian Network approach. Ocean Engineering, 133, 224-230.

Amado L. L., Garcia M. L., Ramos P. B., Freitas R. F., Zafalon B., Ferreira J. L., Yunes J. S., and Monserrat J. M. (2009) A method to measure total antioxidant capacity against peroxyl radicals in aquatic organisms: Application to evaluate microcystins toxicity. Science of the total environment, 407, 6, 2115-2123.

AMAP, 1998. AMAP Assessment Report: Arctic Pollution Issues. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway. xii+859 pp.

AMAP, 2010. Assessment 2007: Oil and Gas Activities in the Arctic - Effects and Potential Effects. Volume I. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway (vii + 423 pp).

Amstrup, S. C., Marcot B. G., and Douglas D. C. (2008). A Bayesian Network Modeling Approach to Forecasting the 21st Century Worldwide Status of Polar Bears Pp. 213–268 in DeWeaver E. T., Bitz C. M. and Tremblay L. B., eds. Arctic Sea Ice Decline: observations, Projections, Mechanisms, and Implications. Geophysical Monograph, 180. American Geophysical Union, Washington DC.

Andersen O., Frantzen M., Rosland M., Timmerhaus G., Skugor A., and Krasnov A. (2015). Effects of crude oil exposure and elevated temperature on the liver transcriptome of polar cod (Boreogadus saida). Aquatic Toxicology, 165, 9-8.

Ankley G. T., Bennett R. S., Erickson R. J., Hoff D. J., Hornung M. W., Johnson R. D., Mount D. R., Nichols J. W., Russom C. L., Schmieder P. K., Serrano J. A., Tietge J. E., and Villeneuve D. L. (2010). Adverse outcome pathways: A concenptual framework to support ecotoxicology research and risk assessment. Environmental Toxicology and Chemistry, 29, 3, 730-741.

Aruoja V., Moosus M., Anne K., Sihtmae M., and Maran U. (2014). Measurement of baseline toxicity and QSAR analysis of 50 non-polar and 58 polar narcotic chemicals for the alga Pseudokirchneriella subcapitata. Chemosphere, 96, 23-32.

Ashauer R. (2007). Predicting effects of fluctuating or pulsed exposure to pesticides on aquatic organisms. PhD thesis, Department of Environmental Sciences, University of York.

Ashauer R., and Escher B. I. (2010). Advantages of toxicokinetic and toxicodynamic modelling in aquatic ecotoxicology and risk assessment. Journal of Environmental Monitoring, 12, 11, 2056-2061.

Ashauer R., Hintermeister A., Potthoff E., Escher B. I. (2011 a). Acute toxicity of organic chemicals to Gammarus pulex correlates with sensitivity of Daphnia magna across modes of action. Aquatic Toxicology, 103, 38-45.

Ashauer R., Thorbek P., Warinton J. S., Wheeler J. R., and Maund S. (2013). A method to predict and understand fish survival under dynamic chemical stress using standard ecotoxicity data. Environmental Toxicity and Chemistry, 32, 4, 954-965.

Ashauer, R., Agatz, A., Albert, C., Ducrot, V., Galic, N., Hendriks, J., Jager, T., Kretschmann, A., O'Connor, I., Rubach, M.N., Nyman, A.M., Schmitt, W., Stadnicka, J., Van Den Brink, P.J., Preuss, T.G., 2011b. Toxicokinetic-toxicodynamic modeling of quantal and graded sublethal endpoints: a brief discussion of concepts. Environ. Toxicol. Chem. 30 (11), 2519–2524.

Ashauer, R., Brown, C.D., 2008. Toxicodynamic assumptions in ecotoxicological hazard models. Environ. Toxicol. Chem. 27 (8), 1817–1821.

Ashauer, R., Escher, B.I., 2010. Advantages of toxicokinetic and toxicodynamic modelling in aquatic ecotoxicology and risk assessment. J. Environ. Monit. 12 (11), 2056–2061.

Austin T., Denoyelle M., Chaudry A., Stradling S., and Eadsforth C. (2015). European chemicals agency dossier submissions as an experimental data source: refinement of a fish toxicity model for predicting acute LC50 values. Environmental Toxicology and Chemistry, 34, 2, 369-378.

Baas J., Spurgeon D., and Broerse M. (2015). A simple mechanistic model to interpret the effects of narcotics. SAR and QSAR in Environmental Research, 26, 3, 165-180.

Bakke M. J., Nahrgang J., and Ingebrigtsen K. (2016). Comparative absorption and tissue distribution of ^{14}C-benzo(a)pyrene and ^{14}C-phenanthrene in the polar cod (Boreogadus saida) following oral administration. Polar biology. 39, 6, 1165-1173.

Banni M., Bouraoui Z., Ghedira J., Clerandeau C., Guerbej H., Narbonne J. F., and Bousetta H. (2009). Acute effects of benzo[a]pyrene on liver phase I and phase II enzymes, and DNA damage on sea bream Sparus aurata. Fish Physiology and Biochemistry, 35, 293-299.

Barber DG, Saczuk E, Richard P (2001) Examination of beluga-habitat relationships through the use of telemetry and a geographic information system. Arctic 54:305–316.

Barron M. G., Bejarano A. C., Conmy R. N., Sundaravadivelu D., Meyer P. (2020). Toxicity of oil spill response agents and crude oils to five aquatic test species. Marine Pollution Bulletin, 153, 110954.

Barron M. G., Stehly G. R., and Hayton W. L. (1990). Pharmacokinetic modeling in aquatic animals: models and concepts. Aquatic Toxicology, 18, 61-86.

Bayesfusion (n.d) https://support.bayesfusion.com/docs/QGeNIe.pdf (Last accessed: November, 25th 2020).

Bender M. L., Giebichenstein J., Teisrud R. N., Laurent J., Frantzen M., Meador J. P., Sorensen L., Hansen B. H., Reinardy H. C., Laurel B., Nahrgang J. (2021). Combined effects of crude oil exposure and warming on eggs and larvae of an arctic forage fish. Scientific Reports, 11, 8410.

Berge, J., Renaud, P.E., Darnis, G., Cittier, F., Last, K., Gabrielsen, T.M., Johnsen, G., Seuthe, L., Weslawski, J.M., Leu, E., Moline, M., Nahrgang, J., Soreide, J.E., Varpe, O., Lonne, O.J., Daase, M., Petersen, S.F., 2015. In the dark: a review of ecosystem processes during the Arctic polar night. Prog. Oceanogr. 139, 258–271.

Bessems J. G., Loizou G., Krishnan K., Clewell III H. J., Bernasconi C., Bois F., Coecke S., Collnot E. M., Diembeck W., Farcal L. R., Geraets L., Gundert-Remy U., Kramer N., Kusters G., Leite S. B., Pelkonen O. R., Schroder K., Testai E., Wilk-Zasadna I., and Zaldivar-Comenges J. M. (2014). PBTK modelling platforms and parameter estimation tools to enable animal-free risk assessment. Regulatory Toxicology and Pharmacology, 68, 119-139.

Billiard S. M., Meyer J. N., Wassenberg D. M., Hodson P. V., and Di Giulio R. T. (2008). Non additive effects of PAHs on early vertebrate development: mechanisms and implications for risk assessment. Toxicological Sciences, 105, 1, 5-23.

Bluhm B. A., and Gradinger R. (2008). Regional variability in food availability for Arctic marine mammals. Ecological Applications, 18, 2, 77-96.

Brandvik P.J., SØrheim K.R, Reed M., 2006. Short state-of-the-art report on oil spills in ice-infested waters (final). SINTEF A06148.

Brinkmann M., Eichbaum K., Buchinger S., Reifferscheid G., Bui T., Schaffer A., Hollert H., and Preuss T. G. (2014). Understanding receptor-mediated effects in Rainbow trout: in vitro- in vivo extrapolation using physiologically based toxicokinetics models. Environmental Science and Technology, 48, 3303-3309.

Brown K.G., and Foureman G. L. (2005). Concentration-time-response modeling for acute and short-term exposures. Regulatory Toxicology and Pharmacology, 43, 45-54.

Camus L. (2017). Unique Arctic communities and oil spill response consequences: oil biodegradation and persistence and oil spill response consequences resilience and sensitivity. Arctic response technology Oil Spill Preparedness. http://arcticresponse.wpengine.com/research-project/environmental-effects-from-arcticoil-spills/ Last accessed 5/21/19

Camus L., Birkely S. R., Jones M. B., Borseth J. F., Grosvik B. E., Gulliksen B., Lonne O. J., Regoli F., and Depledge M. H. (2003). Biomarker responses and PAH uptake in Mya trunca following exposure to oil contaminated sediment in an Arctic fjord (Svalbard). Science of The Total Environment, 308, 1-3, 221-234.

Carney Almroth B., Albetsson E., Sturve J., and Forlin L. (2008). Oxidative stress, evident in antioxidant defences and damage products, in rainbow trout caged outside a sewage treatment plant. Ecotoxicology and Environmental Safety, 70, 3, 370-378.

Carroll J., Vikebo F., Howell D., Broch O. J., Nepstad R., Augustine S., Skeie G. M., Bast R., and Juselius J. (2018). Assessing impacts of simulated oil spills on the Northeast Arctic cod fishery. Marine Pollution Bulletin, 126, 63-73.

Celsie A., Mackay D., Parnis J. M., and Arnot J. A. (2016). A fugacity based toxicokinetic model for narcotic organis chemicals in fish. Environmental Toxicology and Chemistry, 35, 5, 1257-1267.

Chapman P. M. (1993). Are Arctic marine invertebrates relatively insensitive to metals?. Environmental Toxicology and Chemistry, 12, 611-613.

Chapman P. M., and McPherson C. (1993). Comparative zinc and lead toxicity tests with Arctic marine invertebrates and implications for toxicant discharges. Polar Record, 168, 45-54.

Chapman P. M., and Riddle M. J. (2003). Missing and needed: Polar marine ecotoxicology. Marine Pollution Bulletin, 46, 8, 927-928.

Chapman P. M., and Riddle M. L. (2005). Toxic effects of contaminants in polar marine environments. Environmental Science and Technology, 39, 200A-207A.

Chapman, P.M., Riddle, M.L., 2005. Toxic effects of contaminants in polar marine environments. Environ. Sci. Technol. 39, 200A–207A.

Chen W. Y., Liao C. M., Ju Y. R., Singh S., Jou L. J., Chen B. C., and Tsai J. W. (2012). Toxicokinetics/toxicodynamics with damage feedback improves risk assessment for tilapia and freshwater clam exposed to arsenic. Ecotoxicology, 21, 485-495.

Chevalier M., Comte L., Laffaille P., and Grenouillet G. (2018). Interactions between species attributes explain population dynamics in stream fishes under changing climate. Ecosphere, 9, 1.

Christiansen J. S., Dalmo R. A., and Ingebrigtsen K. (1996). Xenobiotic excretion in fish with aglomerular kidneys. Marine Ecology Progress Series, 136, 303-304.

Christiansen J. S., Mecklenburg C. W., and Karamushko O. V. (2014). Arctic marine fishes and their fisheries in light of global change. Blobal Change Biology, 20 (2), 352-359.

Connon R. E., Geist J., and Werner I. (2012). Effect based tools for monitoring and predicting the ecotoxicological effects of chemicals in the aquatic environment. Sensors, 12, 12741-12771.

Cusa M. L. J. (2016). The effect of seasonality on polar cod (Boreogadus saida) dietary habits and temporal feeding strategies in Svalbard waters. Thesis, Faculty of Biosciences, fisheries and economics, Department of Arctic and marine biology, The Arctic University of Norway.

David C., Lange B., Krumpen T., Flores H., Schaafsma F. L., Franeker J. A. V. (2016). Under-ice distribution of polar cod Boreogadus saida in the central Arctic Ocean and their association with sea-ice habitat properties. Polar Biology, 39, 981-994.

De Vries, P., Tamis, J., Nahrgand, J. et al. (2021). Quantifying the consequence of applying conservative assumptions in the assessment of oil spill effects on polar cod (Boreogadus saida) populations. Polar Biology, 44, 575-586.

Devillers J., Ed. (2009). Ecotoxicology Modeling: Emerging Topics in Ecotoxicology. Volume 2, Springer Science, New York.

Dickins, D., et al., 2011. Behavior of oil spills in ice and implications for Arctic spill response. In: OTC Arctic Technology Conference. Offshore Technology Conference.

Dimitriv S. D., Mekenyan O. G., and Schultz T. W. (2000). Interspecies modeling of narcotics toxicity to aquatic animals. Bulletin of Environmental Contamination and Toxicology, 65, 399-406.

Dorne J. L. C. M. (2010). Metabolism, variability and risk assessment. Toxicology, 268, 156-164.

Ducrot V., Ashauer R., Bednarska A. J., Hinarejos S., Thorbek P., and Weyman G. (2016). Using Toxicokinetic-Toxicodynamic modeling as an acute risk assessment refinement approach in vertebrate ecological risk assessment. Intregrated Environmental Assessment and Management, 12, 1, 32-45.

Dyck M. G., and Kebreab E. (2009). Estimating the energetic contribution of polar bear (Ursus Maritimus) summer diets to the total energy budget. Journal of Mammalogy, 90, 3, 585-593.

Dyer S. D., Versteeg D. J., Belanger S. E., Chaney J. G., and Mayer F. L. (2006). Interspecies correlation estimates predict protective environmental concentrations. Environmental Science and Technology, 40, 9, 3102-3111.

ECHA. 2015. Read-Across Assessment Framework (RAAF). European Chemical Agency. http://echa.europa.eu/documents/10162/13628/raaf_en.pdf (Last accessed July-27-2016)

Engraff M., Solere C., Smith K. E. C., Mayer P., and Dahllof I. (2011). Aquatic toxicity of PAHs and PAH mixtures at saturation to benthic amphipods: linking toxic effects to chemical activity. Aquatic Toxicology, 102, 142-149.

Environmental Protection Agency. (1992). Framework for ecological risk assessment. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington DC. EPA/630/R-92/001.

Eriksen E., Huserbraten M., Gjosaeter H., Vikebo F., and Abertsem J. (2019). Polar cod egg and larval drift patterns in the Svalbard archipelago. Polar Biol (2019). https://doi.org/10.1007/s00300-019-02549-6

Escher B. I. (2001). Molecular mechanisms in aquatic ecotoxicology: specific and non-specific membrane toxicity. PhD dissertation, Swiss Federal Institute of Technology (ETH) Zurich and Swiss Federal Institute for Environmental Science and Technology (EAWAG).

Escher B. I., and Hermens J. L. (2004). Internal exposure: linking bioavailability to effects. Environmental Science and Technology, 38, 23, 455A-462A.

Escher B. I., Hackermuller J., Polte T., Scholz S., Aigner A., Altenburger R., Bohme A., Bopp S. K., Brack W., Busch W., Chadeau-Hyam M., Covaci A., Eisentrager A., Galligan J. J., Garcia-Reyero N., Hartung T., Hein M., Herberth G., Jahnke A., Kleinjans J., Kluver N., Krauss M., Lamoree M., Lehmann I., Luckenbach T., Miller G. W., Muller A., Phillips D. H., Reemtsma T., Rolle-Kampczyk U., Schuurmann G., Schwikowski B., Tan Y. M., Trump S., Walter-Rohde S., and Wambaugh J. F. (2017). From the exposome to mechanistic understanding of chemical-induced adverse effects. Environmental International, 99, 97-106.

Euling S. Y. (2013). Toxicogenomics in risk assessment. Toxicology and Applied Pharmacology, 271, 3.

Fahd F., Khan F., Hawboldt K., and Abbassi R. (2014). Developing a novel methodology for ecological risk assessment of thiosalts. Stochastic Environmental Research and Risk Assessment, 28, 2, 383-391.

Fahd F., Khan F., Veitch B. (2019). Arctic marine fish 'biotransformation toxicity' model for ecological risk assessment. Marine Pollution Bulletin, 142, 408-418.

Fahd F., Khan F., Veitch B., and Yang M. (2017). Aquatic ecotoxicological models and their applicability in Arctic regions. Marine Pollution Bulletin, 120, 1-2, 428-437.

Fahd F., Veitch B., and Khan F. (2019). Arctic marine fish 'biotransformation toxicity' model for ecological risk assessment. Marine Pollution Bulletin, 142, 408-418.

Fahd F., Veitch B., and Khan F. (2020). Risk assessment of Arctic aquatic species using ecotoxicological biomarkers and Bayesian network. Marine Pollution Bulletin, 156, 111212.

FAO, Food and Agriculture Organization of the United Nations. (2015). http://www.fao.org/fishery/species/2233/en

Fox D. R. (2010). A Bayesian approach for determining the no effect concentration and hazardous concentration in ecotoxicology. Ecotoxicology and Environmental Safety, 73, 123-131.

Furuhuma A., Hasunuma K., and Aoki Y. (2015). Interspecies Quantitative Structure-Activity-Activity Relationships (QSAARs) for prediction of acute aquatic toxicity of aromatic amines and phenols. SAR and QSAR in Environmental Research, 26, 4, 301-323.

Gallaway B. J., Konkel W. J., and Norcross B. L. (2017). Some thoughts on estimating change to Arctic cod populations from hypothetical oil spills in the eastern Alaska Beaufort Sea. Arctic Science, 3, 716-729.

Gardiner W. W., Word J. Q., Word J. D., Perkins R, A., McFarlin K. M., Hester B. W., Word L. S., and Ray C. M. (2013). The acute toxicity of chemically and physically dispersed crude oil to key Arctic species under Arctic conditions during the open water season. Environmental Toxicology and Chemistry, 32, 10, 2284-2300.

Gaylor D. W. (2000). The use of Haber's law in standard setting and risk assessment. Toxicology, 14, 149, 1, 17-19.

Gergs A., Kulkarni D., Preuss T. G. (2015). Body size dependent toxicokinetics and toxicodynamics could explain intra and interspecies variability in sensitivity. Environmental Pollution, 206, 449-455.

Gergs A., Zenker A., Grimm V., and Preuss T. G. (2013). Chemical and natural stressors combined: from cryptic effects to population extinction. Scientific Reports , 3, 2036-2043.

Gewurtz, S.B., Laposa, R., Gandhi, N., Christensen, G.N., Evenset, A., Gregor, D., Diamond, M.L., 2006. A comparison of contaminant dynamics in arctic and temperate fish: a modeling approach. Chemosphere 63, 1328–1341.

Giulio R. T., and Hinton D.E. (2008). The toxicology of fishes. CRC press, Taylor and Francis Group, Boca Raton.

Gramatica P. (2007). Principles of QSAR models validation: internal and external. QSAR and Combinational Science, 26, 5, 694-701.

Groh K. J., Carvalho R. N., Chipman J. K., Denslow N. D., Halder M., Murphy C. A., Roelofs D., Rolaki A., Schirmer K., and Watanabe K. H. (2015). Development and application of the adverse outcome pathway framework for understanding and predicting chronic toxicity: I. Challenges and research needs in ecotoxicology. Chemosphere, 120, 764-777.

Haber L. T., Maier A., Zhao Q., Dollarhide J. S., Savage R. E., and Dourson M. L. (2001). Toxicological Sciences, 61, 32-39.

Hallanger I. G., Ruus A., Warner N. A., Herzke D., Evenset A., Schoyen M., Gabrielsen G. W., and Borga K. (2011). Differences between Arctic and Atlantic fjord systems on bioaccumulation of persistent organic pollutants in zooplankton from Svalbard. Science of the Total Environment, 409, 2783-2795.

Hansen B. H., Altin D., Bonaunet K., and Overjordet I. B. (2014). Acute toxicity of eight oil spill response chemicals to temperate, boreal and Arctic species. Journal of Toxicology and Environmental Health, Part A, 77, 495-505.

Hansen B. H., Altin D., Overjordet I. B., Jager T., and Nordtug T. (2013). Acute exposure of water soluble fractions of marine diesel on Arctic Calanus glacialis and boreal Calanus finmarchicus: Effects on survival and biomarker response. Science of Total Environment, 449, 276-284.

Heagler M. G., Newman M. C., Mulvey M., and Dixon P. M. (1993). Allozyme genotype in mosquitofish, Gambusia holbrooki, during mercury exposure: temporal stability, concentration effects and field verification. Environmental Toxicology and Chemistry, 12, 2, 385-395.

Helle I., Lecklin T., Jolma A., and Kuikka S. (2011). Modeling the effectiveness of oil combating from an ecological perspective- A Bayesian network for the Gulf of Finland; the Baltic Sea. Journal of Hazardous Materials, 185, 182-192.

Helle I., Makinen J., Nevalainen M., Afenyo M., and Vanhatalo J. (2020). Impacts of oil spills on Arctic marine ecosystems: A quantitative and probabilistic risk assessment perspective. Environmental Science and Technology, 54, 4, 2112-2121.

Hilderband G. V., Hanley T. A., Robbins C. T., and Schwartz C. C. (1999). Role of brown bears (Ursus arctos) in the flow of marine nitrogen into a terrestrial ecosystem. Oecologia, 121, 546-550.

Hoff D., Lehmann W., Pease A., Raimondo S., Russom C., and Steeger T. (2010). Predicting the toxicities of chemicals to aquatic animal species. U.S. Environmental Protection Agency Report, October 27.

Hook S. E., Gallagher E. P., amd Batley G. E. (2014). The role of biomarkers in the assessment of aquatic ecosystem health. Integrated Environmental Assessment and Management, 10, 3, 327-341.

Hoondert R. P. J., Van den Brink N. W., Van den Heuvel-Greve M. J., Ragas J., Jan Hendriks A. (2020). Implications of trophic variability for modeling biomagnification of POPs in marine food webs in the Svalbard Archipelago. Environmental Science and technology, 54, 7, 4026-4035.

Hoop L. D., Schipper A. M., Leuven R. S. E. W., Huijbregts M. A. J., Olsen G. H., Smit M. G. D., and Hendriks A. J. (2011). Sensitivity of Polar and temperate marine organisms to oil components. Environmental science and Technology, 45, 9017-9023.

Hooper M. J., Ankley G. T., Cristol D. A., Maryoung L. A., Noyes P. D., and Oinkerton K. E. (2012). Interactions between chemical and climate stressors: A role for mechanistic toxicology in assessing climate change risks. Environmental Toxicology and Chemistry, 32, 1, 32-48.

Hop H., and Gjosaeter H. (2013). Polar cod (Boreagadus saida) and capelin (Mallatus villosus) as key species in marine food webs of the Arctic and the Barents Sea. Marine Biology Research, 9, 878-894.

Hoyle S.D., and Maunder M.N. (2004). A Bayesian integrated population dynamics model to analyze data for protected species. Animal Biodiversity and Conservation, 27, 1.

Huesemann M. H., Hausmann T. S., and Fortman T. J. (2002). Microbial factors rather than bioavailability limit the rate and extent of PAH biodegradation in aged crude oil contaminated model soils. Bioremediation Journal, 6,4, 321-336.

Huntington H. P. (2009). A preliminary assessment of threats to arctic marine mammals and their conservation in the coming decades. Marine Policy, 33, 77-82.

Huserbraten M. B. O., Eriksen E., Gjosaeter H., and Vikebo F. (2019). Polar cod in jeopardy under the retreating Arctic sea ice. Communication Biology, 2, 407.

Ingebrigtsen K., Christiansen J. S., Lindhe O., Brandt I. (2000). Disposition and cellular binding of 3H-benzo(a)pyrene at subzero temperatures: studies in an aglomerular arctic teleost fish – the polar cod (Boreogadus saida). Polar Biology, 23, 503–509.

IPCC, 2014: Climate Change 2014: Synthesis Report. Contribution of Working Groups I, II and III to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change [Core Writing Team, R.K. Pachauri and L.A. Meyer (Eds.)]. IPCC, Geneva, Switzerland, 151 pp.

Jager T. (2013). All individuals are not created equal; accounting for interindividual variation in fitting life history responses to toxicants. Environmental Science and Technology, 47, 1664-1669.

Jager T., Albert C., Preuss T. G., and Ashauer R. (2011). General unified threshold model of survival – a toxicokinetic-toxicodynamic framework for ecotoxicology. Environmental Science and Technology, 45, 2529-2540.

Jagielski P. M., Dey C. J., Gilchrist H. G., Richardson E. S., Semeniuk A. D. (2021). Polar bear foraging on common eider eggs: estimating the energetic consequences of a climate mediated behavioural shift. Animal Behaviour, 171, 63-75.

Jensen E. M. (2014). Exposure of juvenile Atlantic cod (Gadus morhua) to water accomodated fractions of Arabian Light crude oil: biotransformation and DNA damage. Master of Science thesis. University of Oslo, Oslo, Norway.

Jensen L. K. (2011). Acute and long-term effects from petroleum discharges on temperate and Arctic Calanus species. PhD dissertation, University of Tromso.

Jensen L. K., and Carroll J. (2010). Experimental studies of reproduction and feeding for two Arctic dwelling Calanus species exposed to crude oil. Aquatic Biology, 10, 261-271.

Jonsson F., and Johanson G. (2003). The Bayesian population approach to physiological toxicokinetic-toxicodynamic models- an example using the MCSim software. Toxicology Letters, 138, 143-150.

Jonsson, H., Sundt, R.C., Aas, E., Sanni, S., 2010. The Arctic is no longer put on ice: evaluation of Polar cod (Boreogadus saida) as a monitoring species of oil pollution in cold waters. Mar. Pollut.

Bull. 60, 390–395.

Kahn I., Maran U., Benfenati E., Netzeva T. I., Schultz W., and Cronin M. T. D. (2007). Comparative Quantitative Structure-Activity-Activity Relationships for toxicity to tetrahymena pyriformis and pimephales promelas. Altenatives to Laboratory Animals, 35,1, 15-24.

Kaikkonen L., Parviainen T., Rahikainen M., Uusitalo L., Lehikoinen A. (2020). Bayesian networks in environmental risk assessment: A review. Integrated Environmental Assessment and Management, 17, 1, 62-78.

Kaiser, M.J., Attrill, M.J., Jennings, S., Thomas, D.N., Barnes, D.K.A., Brierley, A.S., Hiddink, J.G., Kaartokallio, H., Polunin, N.V.C., Raffaelli, D.G., 2011. Marine Ecology: Processes, Systems, and Impacts. Oxford University Press, Oxford.

Kastelein R. A., Ford J., Berghout E., Wiepkema P. R., and Van Boxsel M. (1994). Food consumption, growth and reproduction of Belugas (Delphinapterus leucas) in human care. Aquatic Mammals, 20,2, 87-97.

Khan, B., Khan, F., Veitch, B., and Yang, M. (2018). An operational risk analysis tool to analyze marine transportation in Arctic waters. Reliability Engineering and System Safety 169, 485-502.

Kheriji S., Cafsi E., Masmoudi W., Castell J.D., Romdhane M.S. (2003). Salinity and temperature effects on the lipid composition of mullet sea fry. Aquaculture International, 11, 6, 571-582.

Kim H. J., Koedrith P., and Seo Y. R. (2015). Ecotoxicogenomic approaches for understanding molecular mechanism of environmental chemical toxicity using aquatic invertebrate, Daphnia model organism. International Journal of Molecular Sciences, 16, 12261-12287.

King C. K., and Riddle M. J. (2001). Effects of metal contaminants on the development of the common Antarctic sea urchin Sterechinus neumayeri and comparisons of sensitivity with tropical and temperate echinoids. Marine Ecology Progress Series, 215, 143-154.

Knapen D., Vergauwen L., Villeneuve D. L., and Ankley G. T. (2015). The potential of AOP networks for reproductive and developmental toxicity assay development. Reproductive Toxicology, 56, 52-55.

Kulkarni D., Daniels B., Preuss T. G. (2013). Life-stage-dependent sensitivity of the cyclopoid copepod Mesocyclops leuckarti to triphenyltin. Chemosphere, 92, 1145-1153.

Lacoste M. L. D., Akcha F., Devier M. H., Morin B., Burgeot T., Budzinski H. (2013). Comparative study of different exposure routes on the biotransformation and genotoxicity of

PAHs in the flatfish species, Scophthalmus maximus. Environmental Science Pollution Research International, 20, 2, 690-707.

Laidre K. L., Stern H., Kovacs K. M., Lowry L., Moore S. E., Regehr E. V., Ferguson S. H., Wiig O., Boveng P., Angliss R. P., Born E. W., Litovka D., Quakenbush L., Lydersen C., Vongraven D., and Ugarte F. (2015). Arctic marine mammal population status, sea ice habitat loss, and conservation recommendations for the 21st century. Conservation Biology, 29, 3, 724-737.

Langangen O., Stige L. C., Yaragina N. A., Vikebo F. B., Bogstad B., and Gusdal Y. (2014). Egg mortality of northeast Arctic cod (Gadus morhua) and haddock (Melanogrannus aeglefinus). ICES Journal of Marine Science, 71, 5, 1129-1136.

Lee J. H., and Landrum P. F. (2006). Development of a Multi-Component Damage Assessment Model (MDAM) for time-dependent mixture toxicity with toxicokinetic interactions. Environmental Science and Technology, 40, 4, 1341-1349.

Lee J. H., Landrum P. F., and Koh C. H. (2002). Prediction of time-dependent PAH toxicity in Hyalella Azteca using a Damage Assessment Model. Environmental Science and Technology, 36, 3131-3138.

Lee M. C., Park J. C., Kim D. H., Kang S., Shin K. H., Park H. G., Han J., Lee J. S. (2017). Interralationship of salinity shift with oxidative stress and lipid metabolism in the monogonont rotifer Brachionus koreanus. Comparitive Biochemistry and Physiology, Part A, 214, 79-84.

Lee, K. Boufadel, M., Chen, B., Foght, J., Hodson, P., Swanson, S., Venosa, A. (2015). Expert panel report on the behavior and ecological impacts of crude oil released into adqueous environments. Royal Society of Canada, Ottawa, ON. ISBN: 978-1-928140-0203.

Lenihan H. S., and Oliver J. S. (1995). Anthropogenic and natural disturbances to marine benthic communities in Antarctica. Ecological Applications, 5, 2, 311-326.

Liess M., Cahmapeau O., Riddle M., Schulz R., and Duquesne S. (2001). Combined effects of ultraviolet-B radiation and food shortage on the sensitivity of the Antarctic amphipod Parameora walker to copper. Environmental Toxicology and Chemistry, 20, 2088-2092.

Ling N., Hickey C. W., and Burton G. A. (1998). Are Antarctic marine organisms sensitive to anthropogenic pollutants?. New Zealand Natural Sciences Journal, 23, 106.

Liu K. F. R., Kuo J. Y., Yeh K., Chen C. W., Liang H. H., and Sun Y. H. (2015). Using fuzzy logic to generate conditional probabilities in Bayesian belief networks: a case study of ecological assessment. International Journal of Environmental Science and Technology, 12, 871-884.

Lushchak V. I. (2011). Environmentally induced oxidative stress in aquatic animals. Aquatic Toxicology, 101, 13-30.

Madureira D. J., Weiss F. T., Midwoud P. V., Helbling D. E., Sturla S. J., and Schirmer K. (2014). Systems toxicology approach to understand the kinetics of Benzo(a)pyrene uptake, biotransformation and DNA adduct formation in a liver cell model. Chemical Research in Toxicology, 17, 27, 3, 443-453.

Marcot B. G., Holthausen R. S., Raphael M. G., Rowland M. M., and Wisdom M. J. (2001). Using Bayesian belief networks to evaluate fish and wildlife population viability under land management alternatives from an environmental impact statement. Forest Ecology and Management, 153, 29-42.

McDonald K. S., Ryder D. S., and Tighe M. (2015). Developing best-practice Bayesian Belief Networks in ecological risk assessments for freshwater and estuarine ecosystems: A quantitative review. Journal of Environmental Management, 154, 190-200.

Miller F. J., Schlosser P. M., and Janszen D. B. (2000). Haber's rule: a special case in a family of curves relating concentration and suration of exposure to a fixed level of response for a given endpoint. Toxicology, 149, 21-34.

Moore M. N. (1992). Pollutant induced cell injury in fish liver: use of fluorescent molecular probes in live hepatocytes. Marine Environmental Research, 34, 25-31.

MOSJ, Environmental Monitoring of Svalbard and Jan Mayen. (2019). http://www.mosj.no/en/fauna/marine/polar-cod.html

Nahrgang J., Camus L., Broms F., Christiansen J. S., and Hop H. (2010). Seasonal baseline levels of physiological and biochemical parameters in polar cod (Boreogadus saida): Implications for environmental monitoring. Marine Pollution Bulletin, 60, 1336-1345.

Nahrgang J., Camus L., Gonzalez P., Christiansen J. S., and Hop H. (2009). PAH biomarker responses in polar cod (Boreogadus saida) exposed to benzo(a)pyrene. Aquatic Toxicology, 94, 4, 309-319.

Nahrgang J., Dubourg P., Frantzen M., Storch D., Dahlke F., and Meador J. P. (2016). Early life stages of an arctic keystone species (Boreogadus saida) show high sensitivity to a water-soluble

fraction of crude oil. Environmental Pollution, 218, 605-614.

Nahrgang J., Jonsson M., and Camus L. (2010a). EROD activity in liver and gills of polar cod (Boreogadus saida) exposed to waterborne and dietary crude oil. Marine Environmental Research, 70, 120-123.

Nendza M., Muller M., and Wenzer A. (2014). Discriminating toxicant classes by mode of action: baseline and excess toxicity. SAR and QSAR in Environmental Research, 25, 5, 393-405.

Netzeva T. I., Manuela P., and Worth A. P. (2007). Review of (Quantitative) Structure-Activity Relationships for acute aquatic toxicity. QSAR and Combinatorial Science. 27, 1, 77-90.

Nevalainen M., Helle I., and Vanhatalo J. (2017). Preparing for the unprecedented- Towards quantitative oil risk assessment in the Arctic marine areas. Marine Pollution Bulletin, 114, 90-101.

Newman M. C., and McCloskey J. T. (2000). The individual tolerance concept is not the sole explanation for the probit dose-effect model. Environmental Toxicology and chemistry, 19, 520-526.

Nichols J. W., McKim J. M., Andersen M. E., Gargas M. L., Clewell III H. J., and Erickson R. J. (1990). A physiologically based toxicokinetic model for the uptake and disposition of waterborne organic chemicals in fish. Toxicology and Applied Pharmacology, 106, 433-447.

NSIDC, National Snow Ice Data Centre. (2020) https://nsidc.org/cryosphere/seaice/environment/mammals_seals.html

Nyman A. M., Schirmer K., and Ashauer R. (2014). Importance of toxicokinetics for interspecies variation in sensitivity to chemicals. Environmental Science and Technology, 48, 5946-5954.

Olsen G. H., Carroll M. L., Renaud P. E., Ambrose Jr. W. G., Olsson R., and Carroll J. (2007). Benthic community response to petroleum associated components in Arctic versus temperate marine sediments. Marine Biology, 151, 2167-2176.

Olsen G. H., Smit M. G. D., Carroll J., Jaeger I., Smith T., and Camus L. (2011). Arctic versus temperate comparison of risk assessment metrics for 2-methyl-naphthalene. Marine Environmental Research, 72, 179-187.

Organisation for Economic Co-operation and Development (OECD). (2004). OECD principles of corporate governance. OECD Publication service, Paris.

Otitoloju A., and Olagoke O. (2011). Lipid peroxidation and antioxidant defense enzymes in Clarias gariepinus as useful biomarkers for monitoring exposure to polycyclic aromatic hydrocarbons. Environmental Monitoring and Assessment, 182, issue 1-4, 205-213.

Pagano A. M., Durner G. M., Rode K. D., Atwood T. C., Atkinson S. N., Peacock E., Costa D. P., Owen M. A., Williams T. M. (2018). High energy, high fat lifestyle challenges an Arctic apec predator, the polar bear. Science, 359, 6375, 568-572.

Parker-Stetter S. L., Horne J. K., and Weingartner T. J. (2011). Distribution of polar cod and age-0 fish in the U.S. Beaufort Sea. Polar Biology, 34, 1543-1557.

Patlewicz G., and Fitzpatrick J. M. (2016). Current and future perspectives on the development, evaluation, and application of in silico approaches for predicting toxicity. Chemical Research in Toxicology, 29,438-451.

Pecuchet L., Blanchet M. A., Frainer A., Hussin B., Jorgensen L. L., Kortsch S., Primicerio R. (2020). Novel feeding interactions amplify the impact of species redistribution on an Arctic food web. Global Change Biology, 26, 9, 4894-4906.

Pery A. R. R., Devillers J., Brochot C., Mombelli E., Palluel O., Piccini B., Brion F., and Beaudouin R. (2014). A physiologically based toxicokinetic model for zebrafish danio rerio. Environmental Science and Technology, 48, 781-790.

Pitchforth J., and Mengersen K. (2013). A proposed validation framework for expert elicited Bayesian networks. Expert Systems with Applications, 40, 1, 162-167.

Piva F., Ciaprini F., Onorati F., Benedetti M., Fattorini D., Ausili A., and Regoli F. (2011). Assessing sediment hazard through a weight of evidence approach with bioindicator organism: A practical model to elaborate data from sediment chemistry, bioavailability, biomarkers and ecotoxicological bioassays. Chemosphere, 83, 475-485.

Pollino C. A., and Hart B. T. (2005). Bayesian approaches can help make better sense of ecotoxicological information in risk assessments. Australian Journal Of Ecotoxicology, 11, 57-58.

Priante G., Gianesello L., Ceol M., Prete D. D., Anglani F. (2019). Cell death in the kidney. International Journal of Molecular Science. 20, 14, 3598.

Raies A. B., and Bajic V. B. (2016). In silico toxicology: computational methods for the prediction of chemical toxicity. Wiley Interdisciplinary Review Computational Molecular Science, 6, 2, 147-172.

Raimondo S., Mineau P., and Barron M. G. (2007). Estimation of chemical toxicity to wildlife species using interspecies correlation models. Environmental Science and Technology, 41, 16, 5888-5894.

Ramos E. U., Vaal M. A., and Hermens J. L. M. (2002). Interspecies sensitivity in the aquatic toxicity of aromatic amines. Environmental Toxicology and Pharmacology. 11, 3-4, 149-158.

Regoli F., Giuliani M. E., Benedetti M., and Arukwe A. (2011). Molecular and biochemical biomarkers in environmental monitoring: A comparison of biotransformation and antioxidant defense systems in multiple tissues. Aquatic Toxicology, 105S, 56-66.

Riget F., Vorkamp K., Eulaers I., Dietz R. (2020). Influence of climate and biological variables on temporal trends of persistent organic pollutants in Arctic char and ringed seals from Greenland. Environmental Science: Processes and Impacts, 22, 4, 993-1005.

Rodd A. L., Messier N. J., Vaslet C. A., and Kane A. B. (2017). A 3D fish liver model for aquatic toxicology: Morphological changes and Cyp1a induction in PLHC-1 microtissues after repeated benzo(a)pyrene exposures. Aquatic Toxicology, 186, 134-144.

Rozman K. K., Doull J. (2000 a). Using Haber's law to define the margin of exposure. Toxicology, 149,1-2.

Rozman K. K., Doull J. (2000 b). Dose and time as variables of toxicity. Toxicology, 144, 1-3, 169-178.

Rubach M. N., Baird D. J., Boerwinkel M. C., Maund S. J., Roessink I., and Van den Brink P. J. (2012). Species traits as predictors for intrinsic sensitivity of aquatic invertebrates to the insecticide chlorpyrifos. Ecotoxicology, 21, 2088-2101.

Ryg M., and Øritsland N. A. (1991). Estimates of energy expenditure and energy consumption of ringed seals (Phoca hispida) throughout the year. Polar Research, 10:2, 595-602.

Santana M. S., Sandrini-Neto L., Filipak Neto F., Ribeiro C. A. O., Domenico M. D., and Prodocimo M. M. (2018). Biomarker responses in fish exposed to polycyclic aromatic hydrocarbons (PAHs): Systematic review and meta-analysis. Environmental Pollution, 242, 449-461.

Schultz T. W., Przybylak K. R., Richarz A. N., Mellor C. L., Bradbury S.P., Cronin M. T. D. (2017). Read-across of 90-day rat oral repeated-dose toxicity: a case study for selected 2-alkyl-1-alkanols. Computational Toxicology (In press).

Singsaas, I., Brandvik, P.J., Daling, P.S., Reed, M., Lewis, A., 1994. Fate and behaviour of oils spilled in the presence of ice — a comparison of the results from recent laboratory, meso-scale flume and field tests. In: Proceedings of the 17th AMOP Technical Seminar. vol. 1. Environment Canada, pp. 355–370.

Sobek A., McLachlan M. S., Borga K., Asplund L., Lundstedt-Enkel K., Polder A., and Gustafsson O. (2010). A comparison of PCB bioaccumulation factors between an Arctic and temperate marine food web. Science of the Total Environment, 408, 2753-2760.

Song Y., Nahrgang J., Tollefsen K. E. (2019). Transcriptomic analysis reveals dose-dependent modes of action of benzo(a)pyrene in polar cod (Boreogadus saida). Science of the Total Environment, 653, 176-189.

Sorhus E., Donald C. E., Da Silva D., Thorsen A., Karlsen O., Meier S. (2021). Untangling mechanisms of crude oil toxicity: Linking gene expression, morphology and PAHs at two developmental stages in a cold-water fish. Science of the Total Environment, 757, 143896.

Sorstrom, S.E., Brandvik, P.J., Buist, I., Daling, P., Dickins, D., Faksness, L.-G., Potter, S.,Fritt-Rasmussen, J., Singsaas, I., 2010. Joint industry program on oil spill contingency for Arctic and ice-covered waters: summary report. In: Tech. rep. SINTEF.

Stadnicka J., Schirmer K., and Ashauer R. (2012). Predicting concentrations of organic chemicals in fish by using toxicokinetic models. Environmental Science and Technology, 46, 3273-3280.

Stark J. D., Banks J. E., and Acheamponga S. (2004). Estimating susceptibility of biological control agents to pesticide: influence of life history strategies and population structure. Journal of Biological Control, 29, 392-398.

Steiner N. S., Cheung W. W. L., Cisneros-Montemayor A. M., Drost H., Hayashida H., Hoover C., Lam J., Sou T., Rashid Sumaila U., Suprenand P., Tai T., C., and VanderZwaag D. L. (2019). Impacts of the changing ocean-sea ice system on the key forage fish Arctic cod (Boreogadus Saida) and subsistence fisheries in the western Canadian Arctic – Evaluating linked climate, ecosystem and economic (CEE) models. Frontiers in Marine Science, 6, 179.

Strobel A., Burkhardt-Holm P., Schmid P., and Segner H. (2015). Benzo(a)pyrene metabolism and EROD and GST biotransformation activity in the liver of red and white-blooded Antarctic fish. Environmental Science and Technology, 49, 13, 8022-8032

Suprenand P.M., Hoover C., Ainsworth C.H., Dornberger L.N., Johnson C.J. (2020) Preparing for the Inevitable: Ecological and Indigenous Community Impacts of Oil Spill-Related Mortality in the United States' Arctic Marine Ecosystem. In: Murawski S. et al. (eds) Scenarios and Responses to Future Deep Oil Spills. Springer, Cham. https://doi.org/10.1007/978-3-030-12963-7_27.

Suter G. W. (2007). Ecological Risk Assessment, second edition. CRC press, Taylor and Francis group, Boca Raton.

Tantipisanuh N., Gale G. A., and Pollino C. (2014). Bayesian networks for habitat suitability modeling: a potential tool for conservation planning with scarce resources. Ecological Applications, 24, 7, 1705-1718.

Tarsi K., and Tuff T. (2012). Introduction to population demographics. Nature Education Knowledge, 3, 11, 3.

Tomy G. T., Halldorson T., Chernomas G., Bestvater L., Danegerfield K., Ward T., Pleskach K., Stern G., Atchison S., Majewski A., Reist J. D., and Palace V. P. (2014). Polycyclic aromatic hydrocarbon metabolites in Arctic cod (Boreogadus saida) from the Beaufort Sea and associated fish health effects. Environmental Science and Technology, 48, 11629-11636.

Turcotte D. (2008). Toxicity and metabolism of alkyl-polycyclic aromatic hydrocarbons in fish. Department of Chemistry, Queen's University Canada.

Underwood F. M., Parkes G., and Swasey J. H. (2015). Building Bayesian Belief Networks to investigate how fishery performance responds to management interventions. Fisheries Research, 182, 28-38.

Van Tiem L. A. (2011). Molecular mechanisms of polycyclic aromatic hydrocarbon-induced teratogenesis in Zebrafish (Danio rerio). PhD dissertation, Department of Environment, Duke

University.

Varnasi U. (1989). Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. CRC press

Veltman K., Hendriks A. J., Huijbregts M. A., Wannaz C., Jolliet O. (2014). Toxicokinetic toxicodynamic (TK TD) modeling of Ag toxicity in freshwater organisms: whole-body sodium loss predicts acute mortality across acquatic species. Environmental Science and Technology, 48, 24, 14481 – 14489.

Verhaar H. J. M., Leeuwen C. J. V., and Hermens J. L. M. (1992). Classifying environmental pollutants: Structure-Activity Relationships for prediction of aquatic toxicity. Chemosphere, 25, 4, 471-491.

Verhaar H. J. M., Morroni J. R., Reardon K. F., Hays S. M., Gaver Jr. D. P., Carpenter R. L., and Yang R. S. H. (1997). A proposed approach to study the toxicology of complex mixtures of petroleum products: The integrated use of QSAR, lumping analysis and PBPK/PD modeling. Environmental Health Perspectives, 105, 1, 179-195.

Vieweg et al. 2017. Antioxidant defenses in polar cod (Boreogadus saida) and responsiveness toward dietary crude oil exposure. Marine Environmental Reasearch, 130, 48-59.

Vieweg I., Benedetti M., Lanzoni I., Regoli F., and Nahrgang J. (2017). Antioxidant defenses in polar cod (Boreagadus saida) and responsiveness toward dietary crude oil exposure. Marine Environmental Research, 130, 48-59.

Vink S. R., Mikkers J., Bouwman T., Marquart H., and Kroese E. D. (2010). Use of read-across and tiered exposure assessment in risk assessment under REACH – a case study on a phase-in substance. Regulatory Toxicology and Pharmacology, 58, 64-71.

Wade H. P., Loseto L. L., Majewski A., Hussey N. E. (2021). Cod movement ecology in a warming world: Circumpolar arctic gadids. Fish and Fisheries, 22, 3, 562-591.

Werner I. (2006). Seasonal dynamics, cryo-pelagic interactions and metabolic rates of Arctic pack-ice and under-ice fauna – A review. Polarforschung, 75, 1, 1-19.

Willis A. M., and Oris J. T. (2014). Acute photo-induced toxicity and toxicokinetics of single compounds and mixtures of polycyclic aromatic hydrocarbons in zebrafish. Environmental Toxicology and Chemistry, 33, 9, 2028-2037.

Zhang X. J., Qin H. W., Su L. M., Qin W. C., Zou M. Y., and Sheng L. X. (2010). Interspecies correlations of toxicity to eight aquatic organisms: theoretical considerations. Science of Total Environment, 408, 4549-4555.

Supplementary Material

7.1 Determining the CPTs for polar cod risk BN model

Winter season in Table S1 was for seven months, Summer was for three months and Autumn for two months. The CPTs for the 'Season' node are presented in Table S1 and were calculated as follows: Winter = 7/12; Summer = 3/12; and Autumn = 2/12.

The trends of seasonal variation in 'Sea ice thickness', 'Feeding activity', 'Baseline Phase I activity' and 'Baseline Phase II activity' were established in section 2.0 of the manuscript. Based on these trends, assumptions were utilized to develop the CPTs for these nodes in the network. The CPTs are presented in Table S2 to S5. All the environmental and geophysical factors are analyzed and quantitatively tied to variation in enzymic activity using conditional dependencies in the Bayesian model. Changes in the Phase I and Phase II activities are evaluated based on the EROD and GST biomarkers activity from various experiments on polar cod as mentioned in Nahrgang et al. (2009, 2010). For exposure concentration in low range, the biomarker activity observed was also in the lower range. However, for exposure concentration in medium range, the biomarker activity was reported highest, and for exposure concentration in high range, the biomarker activity dipped down. Based in this information and assumptions from the authors, the CPTs for 'Phase I activity' and 'Phase II activity' were determined and presented in Table S7 and S8. The CPTs for 'Liver microsomes', 'PAH spill concentration', 'PAH spill location', and 'Leads, polynyas and brine channels' are based on assumptions and presented in Tables S6, S11, S12, and S13. The CPTs for other nodes in the model are presented in Tables S14 to S21.

The advantage with the BBN is that when new probabilistic dependent data is available, the model can be immediately updated.

Table S 1: CPTs for 'Season' node

Season	
Winter	0.59
Summer	0.25
Autumn	0.16

Table S 2: CPTs for 'Sea ice thickness'

Season	**Winter**	**Autumn**	**Summer**
Low	0	0.7	0.45
Medium	0.05	0.25	0.5
High	0.95	0.05	0.05

Table S 3: CPTs for 'feeding activity' node.

Season	**Winter**	**Autumn**	**Summer**
Low	0.75	0.01	0.25
High	0.25	0.99	0.75

Table S 4: CPT for 'Baseline phase I activity' node.

Season	**Winter**	**Autumn**	**Summer**
Normal	0.95	0.75	0.15
High	0.05	0.25	0.85

Table S 5: CPT for 'Baseline phase II activity' node.

Season	**Winter**	**Autumn**	**Summer**
Normal	0.95	0.05	0.3
High	0.05	0.95	0.7

Table S 6: CPT for 'liver microsomes' node.

Low	0.1
Medium	0.6
High	0.3

Table S 7: CPT for 'Phase I activity' node

Baseline phase I activity	**Normal**									**High**								
Concentration in liver	'Concentration in liver' Low			'Concentration in liver' Med			'Concentration in liver' High			'Concentration in liver' Low			'Concentration in liver' Med			'Concentration in liver' High		
Liver microsomes	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.85	0.8	0.8	0.65	0.35	0.15	0.25	0	0	0.99	0.6	0.55	0.8	0.35	0.25	0.6	0.4	0.2
Med	0.15	0.2	0.2	0.35	0.65	0.85	0.25	0.35	0.2	0.01	0.4	0.45	0.2	0.65	0.75	0.4	0.6	0.8
High	0	0	0	0	0	0	0.5	0.65	0.8	0	0	0	0	0	0	0	0	0

Table S 8: CPT for 'Phase II activity' node.

Baseline phase II activity	**Normal**																										
Concentration in liver	Low									Med									High								
Liver microsomes	Low			Med			High			Low			Med			High			Low			Med			High		
Phase I activity	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.9	0.85	0.65	0.9	0.85	0.6	0.9	0.85	0.55	0.55	0.45	0.4	0.55	0.3	0.15	0.8	0	0	0.85	0.65	0.45	0.1	0	0	0.6	0.3	0
Med	0.1	0.15	0.35	0.1	0.15	0.4	0.1	0.15	0.45	0.45	0.55	0.6	0.45	0.5	0.65	0.2	0.65	0.3	0.15	0.35	0.55	0.6	0.7	0.6	0.4	0.7	0.1
High	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0.2	0	0.35	0.7	0	0	0	0.3	0.3	0.4	0	0	0.9

Baseline phase II activity	**High**																												
Concentration in liver	Low									Med									High										
Liver microsomes	Low			Med			High			Low			Med			High			Low			Med			High				
Phase I activity	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High

Low	0.9	0.85	0.35	0.9	0.6	0.55	0.75	0.2	0.25	0.65	0.55	0.4	0.85	0.3	0.35	0.9	0.2	0.3	0.85	0.8	0.5	0.85	0.1	0.05	0.75	0.2	0.1
Med	0.1	0.15	0.65	0.1	0.4	0.45	0.25	0.8	0.75	0.35	0.45	0.6	0.15	0.7	0.65	0.1	0.8	0.7	0.15	0.2	0.5	0.15	0.8	0.85	0.25	0.8	0.8
High	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.1	0.1	0	0	0.1

Table S 9: CPT for 'Bioavailable concentration' node.

PAH spill concentration	**Low**

Leads, polynyas and brine channels	Low						Med						High					
Spill location	Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice	
Feeding activity	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
Low	0.99	0.9	0.85	0.95	0.75	0.7	0.4	0.35	0.85	0.9	0.3	0.2	0.25	0.2	0.9	0.99	0.2	0.1
Med	0.01	0.1	0.15	0.05	0.25	0.3	0.6	0.65	0.15	0.1	0.7	0.8	0.75	0.8	0.1	0.01	0.8	0.9
High	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

PAH spill concentration	**Med**																	
Leads, polynyas and brine channels	Low						Med						High					
Spill location	Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice	
Feeding activity	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
Low	0.75	0.45	0	0	0.25	0.1	0.1	0	0.25	0.15	0	0	0	0	0.4	0.3	0	0

Med	0.25	0.55	0.8	0.9	0.55	0.6	0.4	0.45	0.75	0.85	0.35	0.3	0.35	0.3	0.6	0.7	0.25	0.2
High	0	0	0.2	0.1	0.2	0.3	0.5	0.55	0	0	0.65	0.7	0.65	0.7	0	0	0.75	0.8

PAH spill concentration	**High**																	
Leads, polynyas and brine channels	Low						Med						High					
Spill location	Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice		Over ice		Under ice		Over thin ice	
Feeding activity	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
Low	0.25	0	0	0	0.05	0	0.1	0	0	0	0	0	0	0	0	0	0	0
Med	0.2	0.4	0.2	0.1	0.3	0.3	0.2	0.25	0.225	0.15	0.2	0.15	0.2	0.05	0.4	0.3	0.15	0.01
High	0.55	0.6	0.8	0.9	0.65	0.7	0.7	0.75	0.775	0.85	0.8	0.85	0.8	0.95	0.6	0.7	0.85	0.99

Table S 10: CPT for 'Concentration reaching liver'

Bioavailable concentration	**Low**			**Med**			**High**		
Accumulation in lipid	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.9	0.9	0.95	0.15	0.3	0.35	0	0	0
Med	0.1	0.1	0.05	0.85	0.7	0.65	0.1	0.3	0.35
High	0	0	0	0	0	0	0.9	0.7	0.65

Table S 11: CPT for 'PAH spill concentration' node

Low	0.2
Med	0.45
High	0.35

Table S 12: CPT for 'PAH spill location' node.

Over_ice	0.4
Under_ice	0.4
Over_thin_ice	0.2

Table S 13: CPT for 'Leads, polynyas and brine channels' node.

Season	Winter			Autumn			Summer		
Sea ice thickness	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.6	0.85	0.9	0	0	0.05	0	0	0.15
Med	0.4	0.15	0.1	0.01	0.2	0.8	0.1	0.25	0.7
High	0	0	0	0.99	0.8	0.15	0.9	0.75	0.15

Table S 14: CPT for 'Total cell death' node

Cell death from DNA damage	Low			Med			High		
Cell death from peroxidation	Low	Med	High	Low	Med	High	Low	Med	High
Low	1	0.4	0.1	0.4	0	0	0.05	0	0
Med	0	0.6	0.9	0.6	0.99	0.6	0.75	0.4	0.01
High	0	0	0	0	0.01	0.4	0.2	0.6	0.99

Table S 15: CPT for 'Cell death from DNA damage' node.

Reactive metabolites (ROS)	Low										Med										High								
TOSCOH	Low			Med			High				Low			Med			High				Low			Med			High			
Catalase (CAT)	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.45	0.55	0.65	0.65	0.65	0.7	0.7	0.75	0.9	0.2	0.225	0.32	0.4	0.45	0.45	0.35	0.25	0.25	0	0	0	0	0	0.1	0.3	0.35	0.35		
Med	0.55	0.45	0.35	0.3	0.3	0.3	0.3	0.25	0.1	0.45	0.45	0.38	0.35	0.35	0.35	0.65	0.75	0.75	0.01	0.15	0.15	0.3	0.4	0.4	0.65	0.65	0.65		
High	0	0	0	0.05	0.05	0	0	0	0	0.35	0.325	0.3	0.25	0.2	0.2	0	0	0	0.99	0.85	0.85	0.7	0.6	0.5	0.05	0	0		

Table S 16: CPT for 'Catalase (CAT)' node

Low	0.2
Med	0.5
High	0.3

Table S 17: CPT for 'TOSC ROO' node.

Low	0.15
Med	0.15
High	0.7

Table S 18: CPT for 'Lipid peroxidation' node

TOSC ROO	Low			Med			High		
Reactive metabolites (ROS)	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.85	0.15	0.05	0.85	0.25	0	0.85	0.4	0.75
Med	0.15	0.7	0.05	0.15	0.65	0.15	0.15	0.55	0.15
High	0	0.15	0.9	0	0.1	0.85	0	0.05	0.1

Table S 19: CPT for 'Cell death from peroxidation' node.

GPx	Low			Med			High		
Lipid peroxidation	Low	Med	High	Low	Med	High	Low	Med	High
Low	0.75	0.15	0	0.85	0.75	0	0.999	0.75	0.05
Med	0.25	0.65	0.1	0.15	0.25	0.5	0.001	0.25	0.2
High	0	0.2	0.9	0	0	0.5	0	0	0.75

Table S 20: CPT for 'TOSC OH' node.

Low	0.5
Med	0.3
High	0.2

Table S 21: CPT for ' GPx' node.

Liver microsomes and cytosol	Low	Med	High
Low	0.9	0.2	0
Med	0.1	0.8	0.3
High	0	0	0.7

7. 2 CPTs for apex marine species risk model

The polar bear population was unavailable for Svalbard region or of the east Greenland region. Therefore, the bear population of Barents Sea is used for this study. The population of bears in this region is the highest when compared to other regions. The population of polar bears, whales and seals is obtained from Laidre et al. (2015). The population of polar bears is classed into three states

of low, med and high based on the assumptions made by this study. The probabilities for each state are also assumed.

Table S 22: CPT for 'Baseline polar bear quantity' node.

State of the node	Probability
Low (1900-2400)	0.25
Med (2400-2900)	0.5
High (2900-3500)	0.25

The population of beluga whale and narwhal are considered in this node as both are toothed whales and their diet consists of fish such as Arctic cod and Arctic charr. The population of both these whales were not available for Svalbard region. However, the population of these whales was estimated for the East Greenland region and is used in this study.

Table S 23: CPT for 'Baseline whale population' node.

States of the node	Probability
Low (7000-15000)	0.25
Med (15000-30000)	0.5
High (30000-45000)	0.25

Table S 24: CPT for 'Baseline seal population' node.

State of the node	Probability
Low (545K-645K)	0.2
Med (645K-745K)	0.6
High (>745K)	0.2

The biomass of the polar cod in Barents Sea is used in the model. The biomass of the polar cod varied from 500 million tonnes to 2000 million tonnes. The states of this node were classified in

300 million tonnes intervals. Based on the frequency of occurrence of biomass of polar cod in the last 20 years as obtained from MOSJ (2019).

Table S 25: CPT for 'Baseline polar cod population' before spill.

State of the node	Probability
Biomass 500	0.393
Biomass 800	0.191
Biomass 1100	0.111
Biomass 1400	0.141
Biomass 1700	0.08
Biomass 2000	0.08

Table S 26: CPT for 'Spill size'

State of the node	Probability
Low	0.333
Med	0.333
High	0.333

Table S 27: CPT for 'spill location'.

State of the node	Probability
Over_ice	0.12
Under_ice	0.22
Over_thin_ice	0.66

Table S 28: CPT for 'Season'

Season	Probability
Winter	0.333
Summer	0.333
Autumn	0.333

Table S 29: CPT for 'Adult whale oil spill impact' node.

Location of spill	Over thick ice									Under thick ice									Over thin ice								
Season	Winter			Summer			Autumn			Winter			Summer			Autumn			Winter			Summer			Autumn		
Spill size	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Impact_10	0.75	0.65	0.45	0.65	0.55	0.455	0.5	0.53	0.55	0.55	0.3	0.25	0.4	0.3	0.15	0.5	0.3	0.13	0.65	0.4	0.38	0.55	0.35	0.25	0.6	0.4	0.25
Impact_20	0.15	0.25	0.45	0.25	0.35	0.49	0.4	0.4	0.35	0.2	0.45	0.35	0.35	0.55	0.6	0.3	0.4	0.4	0.35	0.4	0.35	0.35	0.5	0.5	0.4	0.55	0.28
Impact_40	0.1	0.05	0.05	0.1	0.05	0.05	0.09	0.01	0.09	0.15	0.1	0.25	0.1	0.15	0.15	0.1	0.25	0.35	0	0.1	0.15	0.1	0.15	0.15	0	0.05	0.35
Impact_60	0	0.05	0.05	0	0.05	0.005	0.005	0.06	0.005	0.1	0.1	0.1	0.1	0	0.05	0.1	0.05	0.12	0	0.1	0.1	0	0	0.05	0	0	0.12
Impact_80	0	0	0	0	0	0	0.005	0	0.0025	0	0.05	0.05	0.05	0	0.05	0	0	0	0	0	0.02	0	0	0.05	0	0	0
Impact_100	0	0	0	0	0	0	0	0	0.0025	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Table S 30: CPT for 'Whale offspring oil impact' node.

Location of spill	Over thick ice									Under thick ice									Over thin ice								
Season	Winter			Summer			Autumn			Winter			Summer			Autumn			Winter			Summer			Autumn		
Spill size	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Impact_10	0.99	0.99	0.99	0.9	0.82	0.7	0.85	0.7	0.55	0.9	0.63	0.59	0.75	0.54	0.4526322	0.65	0.59	0.45	0.9	0.76923	0.76923	0.7	0.52293	0.52293	0.68	0.57843	0.57843
Impact_20	0.01	0.01	0.01	0.1	0.12	0.22	0.1	0.2	0.2	0.05	0.36	0.4	0.2	0.44	0.3368422	0.25	0.39	0.3	0.05	0.21978	0.21978	0.15	0.29357	0.29357	0.15	0.17647	0.17647
Impact_40	0	0	0	0	0.06	0.05	0.05	0.09	0.15	0.05	0.005	0.005	0.03	0.01	0.1263166	0.06	0.01	0.15	0.05	0.00549	0.00549	0.15	0.11009	0.11009	0.17	0.12745	0.12745
Impact	0	0	0	0	0	0.	0	0.	0.	0	0.0	0.0	0.	0.	0.0	0.	0.	0.	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0

ct_60						03		01	05		05	05	02	01	526320	04	01	08		0549495	0549495		4587272	4587272		8823535	8823535
Impact_80	0	0	0	0	0	0	0	0	0.05	0	0	0	0	0	0.0315779	0	0	0.02	0	0	0	0	0.0275233	0.0275233	0	0.0294122	0.0294122
Impact_100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1.11E-16	0	0	0	0	0	0	0	3.33E-16	3.33E-16	0	0	0

Table S 31: CPT for 'Adult polar cod mortality' node

Location of spill	**Over thick ice**									**Under thick ice**										**Over thin ice**								
Season	**Winter**			**Summer**			**Autumn**			**Winter**			**Summer**			**Autumn**			**Winter**			**Summer**			**Autumn**		
Spill size	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**
P (0 to 5)	0.7	0.7	0.7	0.9	0	0	0.7	0	0	0.7	0	0	1	0	0	0.7	0	0	0.7	0.7	0	1	0.1	0	0.7	0	0
P (5 to 10)	0.3	0.3	0.3	0.1	0.9	0	0.3	0.7	0.1	0.25	0.7	0.7	0	1	0	0.28	0.7	0.1	0.3	0.25	0.7	0	0.9	0	0.2	0.7	0

																								95		
P (10 to 15)	0	0	0	0	0.1	1	0	0.3	0.7	0.05	0.18	0.1	0	0	1	0.02	0.3	0.7	0	0.05	0.2	0	0	1	0.005	0.3	0.3
P (15 to 20)	0	0	0	0	0	0	0	0	0.2	0	0.05	0.1	0	0	0	0	0	0.2	0	0	0.1	0	0	0	0	0	0.7
P (20 to 25)	0	0	0	0	0	0	0	0	0	0	0.05	0.05	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
P (50)	0	0	0	0	0	0	0	0	0	0	0.02	0.05	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Table S 32: CPT for 'Adult polar bear oil spill impact' node.

Location of spill	**Over thick ice**									**Under thick ice**										**Over thin ice**								
Season	**Winter**			**Summer**			**Autumn**			**Winter**			**Summer**			**Autumn**			**Winter**			**Summer**			**Autumn**		
Spill size	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**
Impact_10	0.95	0.95	0.9	0.8	0.78	0.78	0.98	0.78	0.78	0.9	0.9	0.85	0.74	0.59	0.3	0.88	0.7	0.2	0.87	0.85	0.7	0.78	0.45	0.05	0.86	0.65	0.02
Impac	0.	0.	0.1	0.	0.2	0.2	0.	0.2	0.2	0.1	0.1	0.	0.	0.4	0.	0.1	0.	0.7	0.	0.	0.	0.	0.	0.7	0.	0.	0.

t_20	05	05		19			02					15	23		65		28		13	13	28	19	41		12	25	65
Impact_40	0	0	0	0.01	0.02	0.02	0	0.02	0.02	0	0	0	0.03	0.01	0.03	0.02	0.02	0.08	0	0.02	0.02	0.03	0.1	0.2	0.02	0.1	0.25
Impact_60	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.01	0	0	0.01	0	0	0	0	0.03	0.03	0	0	0.03
Impact_80	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.01	0	0	0.01	0	0	0	0	0.005	0.01	0	0	0.04
Impact_100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.005	0.01	0	0	0.01

Table S 33: CPT for 'Polar bear offspring oil spill impact' node.

Location of spill	**Over thick ice**									**Under thick ice**										**Over thin ice**									
Season	**Winter**			**Summer**			**Autumn**			**Winter**			**Summer**			**Autumn**				**Winter**			**Summer**			**Autumn**			
Spill size	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**		**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	
Impact_10	0.5	0.55	0.326	0.69	0.6	0.432	0.65	0.55	0.456	0.95	0.9	0.845	0.99	0.99	0.9	0.99	0.95	0.86		0.8	0.66	0.563	0.82	0.75	0.623	0.82	0.73	0.613	

						731			667													001			966			331
Impact_20	0.5	0.45	0.237	0.29	0.2	0.191235	0.3	0.2	0.156667	0.04	0.08	0.121	0.01	0.01	0.069	0.01	0.04	0.1	0.2	0.3	0.282666	0.15	0.2	0.22893	0.13	0.2	0.255669
Impact_40	0	0	0.282666	0.02	0.2	0.22893	0.05	0.25	0.255667	0.01	0.02	0.03	0	0	0.031	0	0.01	0.025	0	0.04	0.121667	0.03	0.05	0.147104	0.03	0.05	0.098667
Impact_60	0	0	0.121667	0	0	0.147104	0	0	0.098667	0	0	0.003	0	0	0	0	0	0.01	0	0	0.031333	0	0	0	0.02	0.02	0.0025
Impact_80	0	0	0.031333	0	0	0	0	0	0.025	0	0	0.001	0	0	0	0	0	0.005	0	0	0.001333	0	0	0	0	0	0.000733
Impact_100	0	0	0.001333	0	0	0	0	0	0.000733	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Table S 34:: CPT for 'Whale food abundance' node

Alternate food for whale	Available																														
PAH impacted polar cod quantity	Biomass 300			Biomass 500			Biomass 700			Biomass 900			Biomass 1100			Biomass 1300			Biomass 1500			Biomass 1700			Biomass 1900			Biomass 2100		
Baseline whale quantity	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K	Low_100K	Med_200K	High_400K
Below	0.	0.	0.	0.	0.	0.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

	05	09	13	04	05	07																								
Maintenance	0.1	0.15	0.25	0.09	0.1	0.1	0.07	0.07	0.03	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Excess	0.6	0.5	0.42	0.61	0.7	0.65	0.5	0.513	0.52	0.4	0.43	0.45	0.3	0.25	0.23	0.2	0.22	0.25	0.15	0.18	0.19	0.12	0.15	0.18	0.05	0.07	0.08	0.001	0.001	0.001
Abundance	0.25	0.26	0.2	0.26	0.15	0.18	0.43	0.417	0.45	0.6	0.57	0.55	0.7	0.75	0.77	0.8	0.78	0.75	0.85	0.82	0.81	0.88	0.85	0.82	0.95	0.93	0.92	0.999	0.999	0.999

Limited																														
Biomass 300			Biomass 500			Biomass 700			Biomass 900			Biomass 1100			Biomass 1300			Biomass 1500			Biomass 1700			Biomass 1900			Biomass 2100		
Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K	100K	200K	400K
0	0	0	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25
0	0	0	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25	0.5	0.5	0.25
0.0	0.0	0.0	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25	0	0	0.25

0 1	0 1	0 1																											
0 . 9 9 9	0 . 9 9 9	0 . 9 9 9	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5	0	0	0 . 2 5

Table S 35: CPT for 'Polar bear food abundance' node.

Seal prey availability	**Highly unlikely**																												
Baseline polar bear quantity	**Low_1900**									**Med 2650**									**High 3600**										
Alternate food for polar bear	**Berries**			**River fish**			**Whales**			**Berries**			**River fish**			**Whales**			**Berries**			**River fish**			**Whales**				
Baseline seal quantity	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**	**Low**	**Med**	**High**		
Below	0.65	0.6	0.45	0.4	0.3	0.1	0.05	0.05	0	0.75	0.67	0.45	0.55	0.5	0.3	0.05	0.05	0	0.8	0.67	0.4	0.55	0.5	0.3	0.05	0.05	0		
Maintenance	0.25	0.3	0.35	0.3	0.3	0.3	0.35	0.3	0.25	0.25	0.27	0.35	0.4	0.48	0.5	0.4	0.3	0.25	0.2	0.27	0.38	0.4	0.48	0.5	0.4	0.3	0.25		

Excess	0.1	0.1	0.195	0.3	0.4	0.4	0.4	0.45	0.65	0	0.06	0.2	0.05	0.02	0.2	0.35	0.45	0.6	0	0.06	0.22	0.05	0.02	0.2	0.35	0.45	0.6
Abundance	0	0	0.005	0	0	0.2	0.2	0.2	0.1	0	0	0	0	0	0	0.2	0.2	0.15	0	0	0	0	0	0	0.2	0.2	0.15

Unlikely																													
Low_1900									Med 2650									High 3600											
Berries			River fish			Whales			Berries			River fish			Whales			Berries			River fish			Whales					
Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
0.6	0.55	0.4	0.3	0.2	0	0	0	0	0.65	0.6	0.4	0.45	0.4	0.2	0	0	0	0.8	0.6	0.35	0.45	0.45	0.2	0	0	0			
0.3	0.35	0.4	0.4	0.4	0.4	0.35	0.3	0.1	0.35	0.34	0.4	0.45	0.55	0.6	0.4	0.3	0.15	0.2	0.34	0.43	0.45	0.5	0.55	0.4	0.3	0.2			
0.1	0.1	0.195	0.3	0.4	0.4	0.45	0.5	0.75	0	0.06	0.2	0.1	0.05	0.2	0.4	0.45	0.65	0	0.06	0.22	0.1	0.05	0.25	0.4	0.5	0.6			
0	0	0.005	0	0	0.2	0.2	0.2	0.15	0	0	0	0	0	0	0.2	0.25	0.2	0	0	0	0	0	0	0.2	0.2	0.2			

Likely		
Low_1900	Med 2650	High 3600

Berries			River fish			Whales			Berries			River fish			Whales			Berries			River fish			Whales		
Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
0.1	0.1	0.005	0.05	0	0	0	0	0	0	0.06	0.05	0.05	0	0	0	0	0	0.1	0	0	0.05	0.3	0	0	0	0
0.3	0.35	0.4	0.4	0.4	0.3	0.2	0.15	0	0.35	0.34	0.5	0.45	0.5	0.5	0.2	0.1	0	0.3	0.34	0.4	0.4	0.5	0.5	0.1	0	0
0.6	0.55	0.59	0.45	0.5	0.4	0.5	0.55	0.7	0.65	0.5	0.4	0.4	0.45	0.4	0.5	0.55	0.65	0.6	0.65	0.52	0.55	0.1	0.4	0.5	0.55	0.5
0	0	0.005	0.1	0.1	0.3	0.3	0.3	0.3	0	0.1	0.05	0.1	0.05	0.1	0.3	0.35	0.35	0	0.01	0.08	0	0.1	0.1	0.4	0.45	0.5

Highly likely																										
Low_1900									Med 2650									High 3600								
Berries			River fish			Whales			Berries			River fish			Whales			Berries			River fish			Whales		
Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0.4	0.35	0.3	0.36	0.25	0.24	0.2	0.15	0.15	0.5	0.4	0.35	0.4	0.28	0.26	0.2	0.15	0.15	0.5	0.4	0.35	0.4	0.28	0.26	0.2	0.15	0.15
0.6	0.65	0.7	0.64	0.75	0.76	0.8	0.85	0.85	0.5	0.6	0.65	0.6	0.72	0.74	0.8	0.85	0.85	0.5	0.6	0.65	0.6	0.72	0.74	0.8	0.85	0.85

Table S 36: CPT for 'Seal prey availability' node.

Alternate food for seal	No																																			
Baseline seal quantity	Low											Med											High													
PAH impacted polar	Biomass3000	Biomass5000	Biomass7000	Biomass9000	Biomass11000	Biomass13000	Biomass15000	Biomass17000	Biomass19000	Biomass21000	Biomass3000	Biomass5000	Biomass7000	Biomass9000	Biomass11000	Biomass13000	Biomass15000	Biomass17000	Biomass19000	Biomass21000	Biomass3000	Biomass5000	Biomass7000	Biomass9000	Biomass11000	Biomass13000	Biomass15000	Biomass17000	Biomass19000	Biomass21000

codquantity																														
Highly unlikely	0.9	0.3	0.02	0	0	0	0	0	0	0	0	0.95	0.93	0.5	0.18	0.01	0	0	0	0	0	0.97	0.92	0.6	0.31	0.2	0.11	0.05	0	0	0
Unlikely	0.05	0.65	0.75	0.37	0.1	0.01	0	0	0	0	0	0.05	0.07	0.5	0.7	0.79	0.42	0.05	0	0	0	0.03	0.08	0.4	0.69	0.8	0.89	0.95	0.35	0.05	0
likely	0.05	0.05	0.23	0.6	0.8	0.9	0.25	0.2	0.11	0.01	0	0	0	0.12	0.2	0.58	0.7	0.85	0.9	0.6	0	0	0	0	0	0	0	0	0.65	0.85	0.9
Highly likely	0	0	0	0.03	0.1	0.09	0.75	0.8	0.89	0.99	0	0	0	0	0	0	0.25	0.15	0.1	0.4	0	0	0	0	0	0	0	0	0	0.1	0.1

Yes																														
Low												**Med**												**High**								
Biomass	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**	**Biomass**

s3000	s5000	s7000	s9000	s11000	s13000	s15000	s17000	s19000	s21000	s3000	s5000	s7000	s9000	s11000	s13000	s15000	s17000	s19000	s21000	s3000	s5000	s7000	s9000	s11000	s13000	s15000	s17000	s19000	s21000
0.15	0	0	0	0	0	0	0	0	0	0.15	0.1	0.1	0	0	0	0	0	0	0	0.8	0.75	0.15	0.05	0.05	0	0	0	0	0
0.8	0.3	0.02	0.1	0	0	0	0	0	0	0.85	0.8	0.7	0.55	0.45	0.25	0	0	0	0	0.2	0.2	0.5	0.75	0.6	0.7	0.3	0.1	0	0
0.05	0.65	0.75	0.75	0.6	0.55	0.15	0.05	0.01	0	0	0.1	0.2	0.45	0.55	0.35	0.6	0.35	0.1	0.01	0	0.05	0.35	0.2	0.35	0.25	0.7	0.7	0.15	0.05
0	0.05	0.23	0.15	0.4	0.45	0.85	0.95	0.99	1	0	0	0	0	0	0.4	0.4	0.65	0.9	0.99	0	0	0	0	0	0.05	0	0.2	0.85	0.95

Table S 37: CPT for 'PAH impacted polar cod population' node.

% baseline fish haffect	100%

ed																																				
Baseline polar cod quantity	**Biomass 500**						**Biomass 800**						**Biomass 1100**						**Biomass 1400**						**Biomass 1700**						**Biomass 2000**					
Adult cod mortality	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**
Bio	0	0	0.	0.1	0.3	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

mass300			01																																			
Biomass500	1	1	0.99	0.9	0.7	0	0	0	0	0	0	1	0	0	0	0	0	0.15	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Biomass700	0	0	0	0	0	0	0	1	1	1	1	1	0	0	0	0	0	0.85	0	0	0	0	0	0	0.55	0	0	0	0	0	0	0	0	0	0	0	0	0
Biomass900	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2	1	1	0	0	0	0	0	0.9	0.45	0	0	0	0	0	1	0	0	0	0	0	0	0
Biomass11	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0.8	0	0	0	0	0	0	0.35	0.95	0.1	0	0	0	0	0	0	0	0	0	0	0	1

00																																	
Biomass1300	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0.65	0.05	0	0	0	0	0	0.4	1	0	0	0	0	0	0	0	0
Biomass1500	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.6	1	0.6	0	0	0	0	0	0	0	0.35	0
Biomass1700	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0.4	0	0	0	0	0.2	0.55	0.6	1	0.65	0		
Biomass1900	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.8	0.45	0.4	0	0	0		

Biomass2100																																				
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

10%																																				
Biomass 500						**Biomass 800**						**Biomass 1100**						**Biomass 1400**						**Biomass 1700**						**Biomass 2000**					
P(0to5)	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**	**P(0to5)**	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**	**P(0to5)**	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**	**P(0to5)**	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**	**P(0to5)**	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**	**P(0to5)**	**P(5to10)**	**P(10to15)**	**P(15to20)**	**P(20to25)**	**P(50)**
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.55	0.55	0.55	0.55	0.55	0.55	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.45	0.45	0.45	0.45	0.45	0.45	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

20%																																			
Biomass 500						Biomass 800						Biomass 1100						Biomass 1400						Biomass 1700						Biomass 2000					
P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 15)	P(15 to 20)	P(20 to 25)	P(50)
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0.9	0.9	0.9	0.9	0.9	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0.1	0.1	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0.2	0.2	0.2	0.2	0.9	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.8	0.8	0.8	0.8	0.8	0.1	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

30%																																			
Biomass 500						Biomass 800						Biomass 1100						Biomass 1400						Biomass 1700						Biomass 2000					
P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)	P(0 to 5)	P(5 to 10)	P(10 to 1)	P(1 to 2)	P(2 to 2)	P(50)

		5)	0)	5)				5)	0)	5)				5)	0)	5)				5)	0)	5)				5)	0)	5)				5)	0)	5)	
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0.2	0.25	0.25	0.25	1	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.8	0.8	0.75	0.75	0.75	0	0	0	0	0	0.15	0.8	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0.85	0.2	0	0	0	0.2	0.3	0.8
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.6	1	1	0.8	0.7	0.2
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0

40%																																			
Biomass 500						**Biomass 800**						**Biomass 1100**						**Biomass 1400**						**Biomass 1700**						**Biomass 2000**					
P(0 to 5)	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**	**P(0 to 5)**	**P(5 to 10)**	**P(10 to 15)**	**P(15 to 20)**	**P(20 to 25)**	**P(50)**
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	0.1	0	0	0	0	0	0.1	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.1	0.1	0.9	0.9	0.9	0.9	0	0	0	0	0	0.55	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.9	0.9	0.1	0.1	0.1	0	0	0	0	0.2	0.35	0.45	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0.8	0.65	0	0	0	0	0	0	1
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.55	0.6	1	1	1	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.45	0.4	0	0	0	0

Table S 38: Whale food requirement

Beluga whale size	**Kg fish diet annually**
Low (200 kg)	3200
Med (600-700 kg)	4900
High (1400 kg)	5300

The conversion of Kj energy to diet in Kg of fish is based on digestible energy of 757 Kj/100 g of Arctic charr (Table S 39).

Table S 39: Seal food requirement

Seal size distribution	Daily energy requirement in Kj/seal	Annual energy requirement on Kj/seal	Annual diet Kg/seal
Low (<40 kg)	9500	$3.47*10^6$	459
Med (40-70 kg)	12950	$4.73*10^6$	625
High (>70 kg)	19400	$7.10*10^6$	938

Table S 40: CPT for 'Adult whale impact' final outcome node.

Whale food abundance	**Below**						**Maintenance**					
Adult whale spill impact	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**
Impact_10	0	0	0	0	0	0	0.5	0	0	0	0	0
Impact_20	0.5	0	0	0	0	0	0.5	0.5	0	0	0	0
Impact_40	0.5	0.5	0	0	0	0	0	0.5	0.5	0	0	0
Impact_60	0	0.5	0.5	0	0	0	0	0	0.5	0.5	0	0
Impact_80	0	0	0.5	0.5	0.15	0	0	0	0	0.5	0.5	0
Impact_100	0	0	0	0.5	0.85	1	0	0	0	0	0.5	1

Excess						**Abundance**					
Impact_10	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**

0.95	0	0	0	0	0	0.99	0	0	0	0	0
0.05	0.95	0	0	0	0	0.01	0.99	0	0	0	0
0	0.05	0.95	0	0	0	0	0.01	0.99	0	0	0
0	0	0.05	0.95	0	0	0	0	0.01	0.99	0	0
0	0	0	0.05	0.95	0	0	0	0	0.01	0.99	0
0	0	0	0	0.05	1	0	0	0	0	0.01	1

Table S 41: CPT for 'whale offspring impact' final outcome node.

Whale food abundance	**Below**						**Maintenance**					
Adult whale spill impact	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**
Impact_10	0	0	0	0	0	0	0.5	0	0	0	0	0
Impact_20	0.5	0	0	0	0	0	0.5	0.5	0	0	0	0
Impact_40	0.5	0.5	0	0	0	0	0	0.5	0.5	0	0	0
Impact_60	0	0.5	0.5	0	0	0	0	0	0.5	0.5	0	0
Impact_80	0	0	0.5	0.5	0.15	0	0	0	0	0.5	0.5	0
Impact_100	0	0	0	0.5	0.85	1	0	0	0	0	0.5	1

Excess						**Abundance**					
Impact_10	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**
0.95	0	0	0	0	0	0.99	0	0	0	0	0
0.05	0.95	0	0	0	0	0.01	0.99	0	0	0	0
0	0.05	0.95	0	0	0	0	0.01	0.99	0	0	0
0	0	0.05	0.95	0	0	0	0	0.01	0.99	0	0
0	0	0	0.05	0.95	0	0	0	0	0.01	0.99	0
0	0	0	0	0.05	1	0	0	0	0	0.01	1

Table S 42: CPT for 'Adult polar bear impact' final outcome node

Whale food abundance	Below						Maintenance					
Adult whale spill impact	Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100	Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100
Impact_10	0.5	0	0	0	0	0	0.65	0	0	0	0	0
Impact_20	0.5	0.5	0	0	0	0	0.35	0.65	0	0	0	0
Impact_40	0	0.5	0.5	0	0	0	0	0.35	0.65	0	0	0
Impact_60	0	0	0.5	0.5	0	0	0	0	0.35	0.65	0	0
Impact_80	0	0	0	0.5	0.5	0	0	0	0	0.35	0.65	0
Impact_100	0	0	0	0	0.5	1	0	0	0	0	0.35	1

Excess						Abundance					
Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100	Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100
0.9	0	0	0	0	0	0.99	0	0	0	0	0
0.1	0.9	0	0	0	0	0.01	0.99	0	0	0	0
0	0.1	0.9	0	0	0	0	0.01	0.99	0	0	0
0	0	0.1	0.9	0	0	0	0	0.01	0.99	0	0
0	0	0	0.1	0.9	0	0	0	0	0.01	0.99	0
0	0	0	0	0.1	1	0	0	0	0	0.01	1

Table S 43: CPT for 'Polar bear offspring impact' final outcome node.

Whale food abundance	Below						Maintenance					
Adult whale spill impact	Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100	Impact_10	Impact_20	Impact_40	Impact_60	Impact_80	Impact_100
Impact_10	0.4	0	0	0	0	0	0.65	0	0	0	0	0

Impact_20	0.3	0.4	0	0	0	0	0.35	0.65	0	0	0	0
Impact_40	0.3	0.3	0.4	0	0	0	0	0.35	0.65	0	0	0
Impact_60	0	0.3	0.3	0.4	0	0	0	0	0.35	0.65	0	0
Impact_80	0	0	0.3	0.3	0.5	0	0	0	0	0.35	0.65	0
Impact_100	0	0	0	0.3	0.5	1	0	0	0	0	0.35	1

Excess						**Abundance**					
Impact_10	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**	**Impact_10**	**Impact_20**	**Impact_40**	**Impact_60**	**Impact_80**	**Impact_100**
0.9	0	0	0	0	0	0.99	0	0	0	0	0
0.1	0.9	0	0	0	0	0.01	0.99	0	0	0	0
0	0.1	0.9	0	0	0	0	0.01	0.99	0	0	0
0	0	0.1	0.9	0	0	0	0	0.01	0.99	0	0
0	0	0	0.1	0.9	0	0	0	0	0.01	0.99	0
0	0	0	0	0.1	1	0	0	0	0	0.01	1