ANNEX C HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

C.1.1 *Components of Risk Assessment*

Risk assessment can be divided into four (4) major steps and each is discussed in the following sections:

- Hazard identification;
- Dose-response evaluation;
- Exposure assessment; and
- Risk characterisation.

C.1.2 *Hazard Identification*

C.1.2.1 Introduction

Hazard identification is the process of determining whether exposure to a chemical could cause an increase in adverse health effects. It involves characterizing the nature and quantity of possible contaminant releases to the environment, selecting a set of Contaminants of Concern (COCs), gathering and evaluating data on the types of health injury or disease that may be produced by a contaminant, and gathering and evaluating data on the conditions of exposure under which injury or disease is produced.

This section presents a framework for the evaluation of the potential human health effects resulting from ingestion of contaminants contained within the edible portion of marine organisms.

C.1.2.2 Contaminants of Concern (COCs)

Some COCs are known carcinogens while others are not considered to be carcinogenic but having other toxic effects (***Table C.1***). There are also COCs that are both toxic and carcinogenic. Assessment criteria have been developed for each type of toxicological effect to be discussed in ***Section C.1.3.1***.

The COCs adopted are those recommended in the Contaminated Spoil Management Study completed in 1991 (1) and the study on Classification of Dredged Material for Marine Disposal (2), and have been identified as COCs in the EM&A programmes for ESC CMPs and SB CMPs. Information on the toxic effects of each COC is presented below in ***Table C.1***.

Table C.1 Contaminants of Concern

COCs	Potential Toxic Effects
Arsenic (inorganic) (As)	Greater toxicity than organic forms. Inorganic arsenic is a known carcinogen. Bioaccumulated by organisms (bioaccumulation occurs more readily in invertebrates than in fish). Teratogenic, fetotoxic and embryotoxic in several animal species. Effects in humans from exposure to high levels include skin and lung cancers, hearing loss, birth defects and liver, kidney and heart damage. Arsenobetaine, the principal arsenic compound in seafood, is not carcinogenic to mammals.
Cadmium (Cd)	Potential carcinogen and teratogen. Bioaccumulated by organisms. Effects in fish include reduced survival, growth and reproduction, decreased oxygen consumption, enzyme disruption, kidney dysfunction and altered blood chemistry. Effects in mammals include reduced haemoglobin levels, decreased growth, immunotoxicity, histopathology, birth defects, and leukemia. Effects in humans include kidney damage, possible increased risk of cancer, and skeletal disorders.

(1) Mott MacDonald (1991) Contaminated Spoil Management Study. Prepared for Civil Engineering Department.
(2) EVS (1996) Classification and Testing of Sediments for Marine Disposal. Prepared for Civil Engineering Department.

COCs	Potential Toxic Effects
Chromium (Cr)	Considered to be mutagenic and teratogenic at elevated concentrations. Effects in fish include reduced growth and survival, altered plasma cortisol metabolism and locomotor activity. Effects in mammals include adverse effects on blood chemistry and morphological changes in liver, teratogenic effects and genotoxicity. Effects in humans include respiratory disease due to inhalation, and possible carcinogenicity (inhalation route for Cr (VI) only). Cr can exist in many chemical forms although it is usually present as either III or VI oxidation states. Cr (III) is an essential element whereas Cr (VI) is a carcinogen with bronchogenic carcinoma (i.e. lung cancer) being its principal deleterious effect reported in mammals.
Copper (Cu)	Can be acutely toxic to animals but is also an essential nutrient at lower doses. Little tendency to bioaccumulate. Effects in fish include mortality and behavioural changes. Effects in mammals include mortality, growth retardation and teratogenicity. Toxic effects to humans are uncommon, however it is a known teratogen.
Lead (Pb)	Organic lead compounds are usually more toxic than inorganic compounds. Invertebrates are more sensitive than fish to elevated levels. Effects in fish include anaemia, enzyme inhibition, paralysis, teratogenicity, growth reduction, and reduced survival. Effects in mammals include mortality, behavioural effects, paralysis, development effects, weight loss and reduced reproduction. Effects in humans include loss of appetite, cramps, headache, fatigue, paralysis, lead encephalopathy and death. It is also a likely mutagen in humans.
Mercury (Hg)	Organic compounds, especially methyl mercury, are more toxic than inorganic forms. Strongly bioaccumulated in aquatic biota and known to biomagnify within the food chain. Effects to fish include mortality, reproductive impairment, behavioural effects, lesions, enzyme disruption and neurotoxicity. Effects in humans include motor and mental impairment, blindness, deafness, microcephaly, intestinal disturbances, tremors and tissue pathology.
Nickel (Ni)	Bioaccumulates in aquatic organisms, although organisms can naturally regulate levels through increased excretion or decreased uptake. Effects in fish include mortality, deformities, and reduced growth and reproduction. Established teratogen and carcinogen in mammals through inhalation of nickel dust, not through ingestion. Also potential mortality, genotoxicity, and immunological, neurological, developmental, and reproductive effects in mammals. High doses in humans result in intoxication and nausea.
Silver (Ag)	Bioaccumulates in invertebrates and vertebrates. Effects in mammals include cardiac enlargement, vascular hypertension, hepatic necrosis, anaemia, lowered immunological activity, enzyme inhibition, growth retardation, and a shortened life span. No evidence of cancer in humans has been reported.
Zinc (Zn)	Strongly bioaccumulated in all organisms. Minor biomagnification through the food chain. Effects in fish include mortality, deformities and reduced growth, teratogenicity and reproductive impairment. In mammals only very high doses are considered to be toxic, with potential immunological, neurological, developmental, genotoxic, and reproductive effects. Effects in humans include digestive disorders, altered immune system, headache, muscular incoordination, renal failure and death.
Polychlorinated biphenyls (PCBs)	Bioaccumulated in fatty tissues. Biomagnification in higher trophic levels. In humans, symptoms include irritation and lacerations of the skin and mucous membranes, neurological disorders, immunosuppression and carcinogenicity. In addition, reproductive impairment, birth defects and development abnormalities are known to occur when women are exposed before or during pregnancy.
Organochlorine pesticides (DDE/ DDT)	Highly persistent and biologically active in the body. They interfere with fertility and reproduction in a variety of wildlife. Bioconcentrate and biomagnify through the food chain. In mammals they are teratogenic and reproductive toxicants, and potent carcinogens. They are also known to cause abnormalities in the central nervous system.
Tributyltins (TBTs)	High bioconcentration potential, especially in fish and molluscs. Major impact on marine organisms, particularly shellfish at very low concentrations. Effects in fish include disruption of enzyme activity, decreased growth, behavioural abnormalities, increased liver weight, histopathological changes to the liver, kidney and gills, thymus

COCs	Potential Toxic Effects
	atrophy, reduced hatchability of eggs, decreased embryo viability and vertebral malfunctions in larvae. Much less is known about the toxic effects to humans; very high levels of exposure have resulted in death, but exposure at very low levels has not yet been correlated with specific health effects. Medium-level exposure may result in disruption of the endocrine system.

Sources:

1. EVS (1996a) Classification and Testing of Sediments for Marine Disposal. Prepared for CED.
2. EVS (1996b) Contaminated Mud Disposal at East of Sha Chau: Comparative Integrated Risk Assessment. Prepared for CED.
3. Aspinwall Clouston Ltd (1998) A Study of Tributyltin Contamination of the Marine Environment of Hong Kong. Prepared for EPD.
4. Irwin RJ, M VanMouwerik, L Stevens, MD Seese & W Basham (1998) Environmental Contaminants Encyclopedia. National Park Service, Water Resources Division, Water Operations Branch, Colorado.
5. Integrated Risk Information System (IRIS), US EPA.

C.1.3 *Dose-response Evaluation*

Dose-response evaluation involves quantifying the relationship between the degree of exposure to a substance and the extent of toxic injury or disease. The majority of data are derived from animal studies in laboratory or, less frequently, from studies in exposed human populations. There may be many different dose-response relationships for a substance if it produces different toxic effects under different conditions of exposure. The risks of a substance cannot be ascertained with any degree of confidence unless dose-response relationships are quantified, even if the substance is known to be "toxic". Such dose-response relationships have been established for various COCs for exposures to humans, but with varying degrees of certainty.

C.1.3.1 Categorisation of Human Health Effects

For the purpose of the assessment, the effects of the substances listed in ***Section C.1.2.2*** have been classified into two categories: non-carcinogenic effects or carcinogenic effects to humans. Substances are included within both categories if they exhibit both types of effect.

C.1.3.1.1 Non-Carcinogenic Health Effects

One of the fundamental principles of toxicology is the *dose-response relationship*. For virtually all toxic substances, there is a direct relationship between the exposure level (and duration) and the severity of the effects produced. As the exposure level (and/or duration period) is lowered, for the great majority of toxic effects, a point is reached at which no detectable effect occurs. This is termed the threshold dose or No Observed Adverse Effects Level (NOAEL).

In laboratory experiments non-carcinogens display NOAELs as the animals under testing can tolerate doses below a certain finite value, with only a limited chance of the expression of toxic effects. NOAEL themselves are not directly used for human health criteria as the NOAELs relate to toxicity observed in animal bioassays and may not adequately protect the most sensitive receivers in human populations (e.g. embryos). In order to develop criteria for human health, Uncertainty Factors (UFs) [3] are applied to the NOAEL data in order to ensure that risks are over-estimated rather than underestimated. For example, extrapolation of animal toxicity response doses to humans utilises two safety factors of ten, the first for animal-to-human extrapolation and the second for variation of sensitivities within the human population.

The human health criteria developed after application of the UFs are referred to as Reference Dose (RfD). The RfD, promulgated by the US Environmental Protection Agency (USEPA), is an estimate of the daily exposure which appears to present a low risk of adverse effects during an exposure to the

([3]) US EPA (1989) Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish. A Guidance Manual. EPA-503/8-89/002

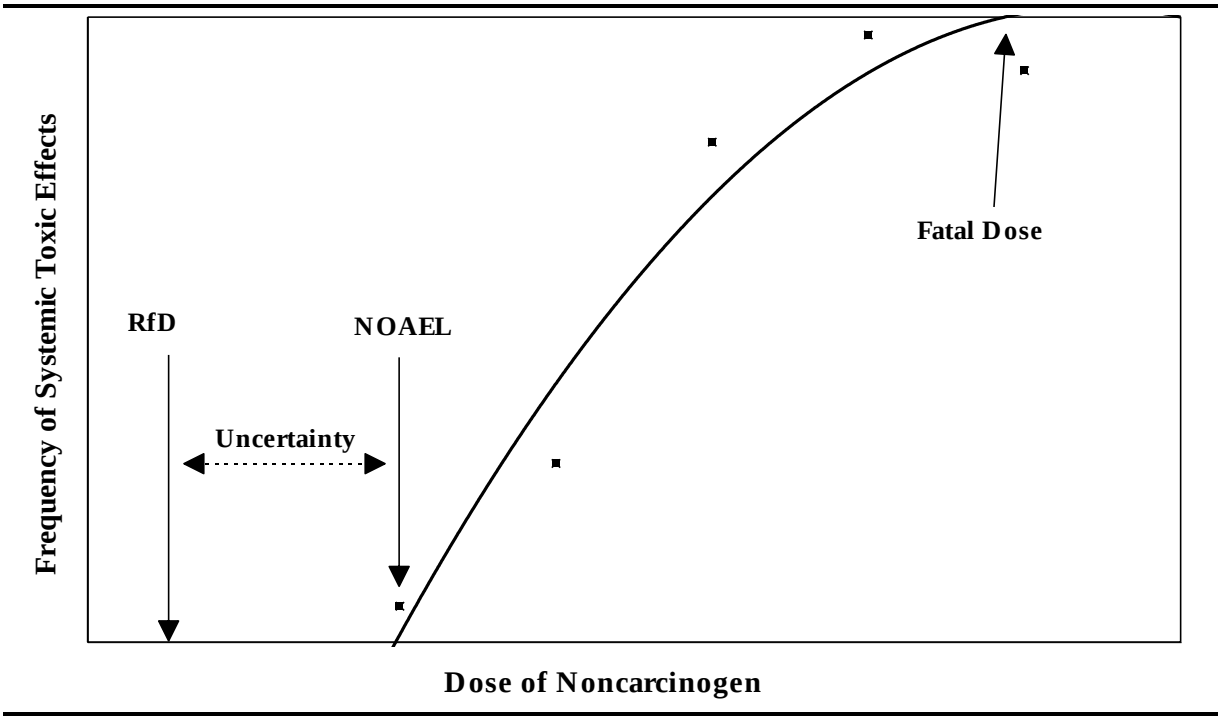
most sensitive members of the receiving population. The purpose of the RfD is to provide a benchmark against which other doses might be compared. Doses which are less than the RfD are not likely to be of concern. Doses which are significantly greater (i.e. at least one order of magnitude) than the RfD may indicate that inadequate margins of safety could exist for exposure to that chemical. The RfD is an approximate number, and while doses higher than the RfD have a higher probability of producing an adverse effect, it should not be inferred that such doses are, by definition, unacceptable (4). For the ingestion route, the RfD is expressed in units of mg kg (body weight)$^{-1}$ day^{-1}.

A summary of RfDs for the COCs is presented in ***Table C.2***. ***Table C.2*** also indicates the carcinogenic class of each COC according to the USEPA classification system which comprises the following categories:

- Class A Human carcinogen
- Class B1 Probable human carcinogen with limited human evidence
- Class B2 Probable human carcinogen with sufficient evidence in animals but inadequate/no evidence in humans
- Class C Possible human carcinogen
- Class D Not classified as a human carcinogen
- Class E Evidence of non-carcinogenicity for humans

Figure C.1 illustrates how RfDs and NOAELs differ from each other.

Figure C.1 Hypothetical Example of a Dose-response Curve for a Non-carcinogen



(4) USEPA Background Document 1A dated March 15, 1993. Reference Dose (RfD): Description and Use in Health Risk Assessments (http://www.epa.gov/iris/rfd.htm).

Table C.2 Toxicity Information taken from the US EPA's Integrated Risk Information System (IRIS)

COC	Oral RfD (mg kg^{-1} day^{-1})	Oral Slope Factor (mg kg^{-1} $day^{-1})^{-1}$	US EPA Carcinogenic Class	Last Revised
Arsenic (inorganic)	0.0003	1.5	A	1/6/1995
Cadmium	0.001 (a)	0.38 (b)	B1	1/2/1999
Chromium III	1.5	-	D	3/9/199
Chromium VI	0.003 (c)	-	D	3/9/1998
Copper	0.04 (d)	-	D	1/7/1997
Lead	0.00143	0.0085	B2	8/7/2004
Mercury	0.00022 (e)	-	D	1/6/1995
Methylmercury	0.0001	-	C	27/7/2001
Nickel	0.02 (f)	no data	A(g)/ B2(h)	1/12/1991
Silver	0.005	-	D	1/12/1991
Zinc	0.3	-	D	3/8/2005
4,4'-DDT	0.0005	0.34	B2	1/2/1996
4,4'-DDE	no data	0.34	B2	22/8/1988
PCBs	0.00002 (i)	0.04 - 2.0 (j)	B2	1/10/1996
Tributyltin (TBT) (k)	0.0003	-	D	1/9/1997

Source: Integrated Risk Information System, USEPA (www.epa.gov/iris).

Notes:

(a) specific RfD for food intake

(b) Office of Environmental Health Hazard Assessment, California Environmental Protection Agency , Public Health Goal for Cadmium In Drinking Water, 1999

(c) used throughout this risk assessment

(d) value derived from Health Effects Assessment Summary Tables (HEAST) reported water quality criteria

(e) no IRIS or HEAST for Hg, converted 0.0003 for $HgCl_2$ by * 0.739

(f) as soluble salts

(g) as inhaled nickel refinery dust and nickel subsulphide

(h) as nickel carbonyl

(i) RfD for Aroclor 1254

(j) 2.0 used throughout the risk assessment

(k) as Tributyltin Oxide (TBTO)

C.1.3.1.2 Carcinogenic Health Effects

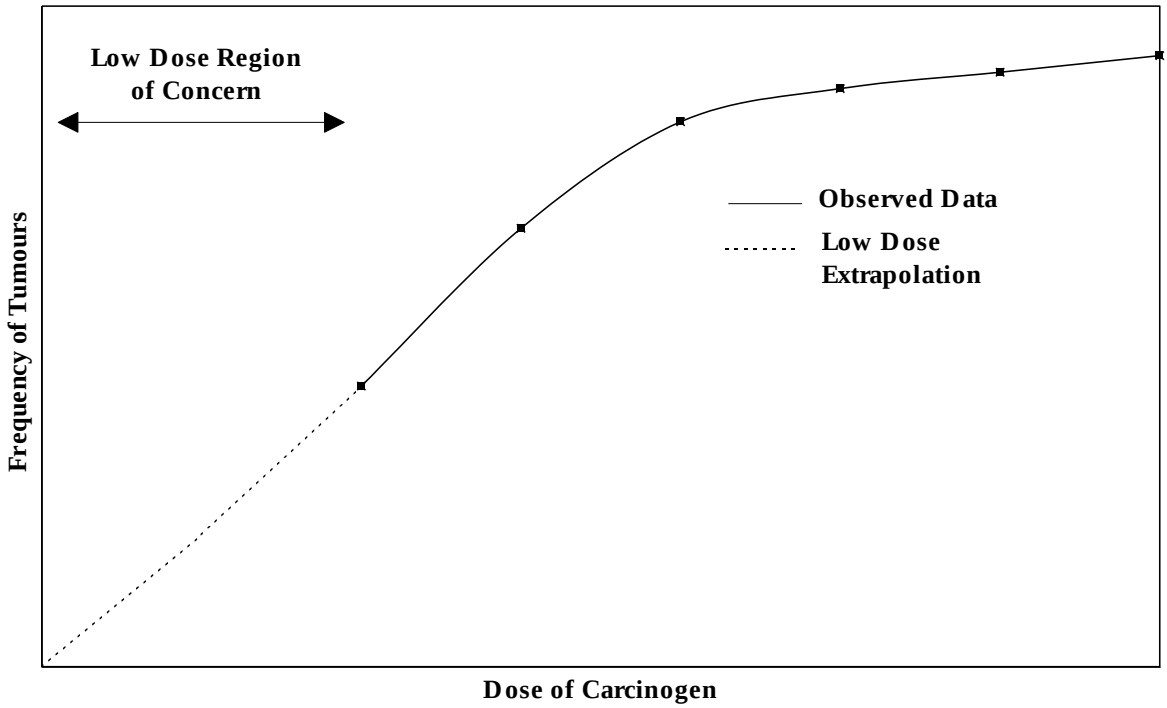
For carcinogenic contaminants there are theoretical grounds for presuming that there may not be a true NOAEL. A carcinogenic health effect can be produced through the mechanisms of initiation or promotion. Genotoxic substances induce cancers by causing mutations in DNA, whereas non-genotoxic substances cause initiated cells to proliferate or differentiate. The two mechanisms differ in that their modes of action lead to fundamentally different techniques of risk assessment. On one hand, genotoxic substances are generally treated as carcinogens for which there is no threshold below which carcinogenic effects are not manifested; in other words, zero risk is only associated with zero exposure. However, non-genotoxic substances are treated as substances which can be tolerated by the receptor up to some finite concentration or dose, beyond which toxic effects are then manifested. In this assessment, a non-threshold approach for all carcinogens is assumed, i.e., all carcinogens are considered to be genotoxic. This is a conservative assumption.

Where a NOAEL cannot be demonstrated experimentally, mathematical models have been developed, particularly in the US, to enable a worst-case extrapolation from high doses to much lower

exposures to be made. Using such calculations, the USEPA has also ranked substances causing cancer in animals using Slope Factors (SFs) (formerly known as Cancer Potency Factors).

The SFs can be used to estimate the excess lifetime cancer risks associated with various levels of exposure to potential human carcinogens. The SF is a number which, when multiplied by the lifetime average daily dose per kg body weight of a potential carcinogen, yields the lifetime cancer risk resulting from exposure at that dose. In practice, SFs are derived from the results of human epidemiological studies or chronic animal bioassays. The data from animal studies are fitted to linearized multistage models and a dose-response curve is obtained. The slope in the low-dose range is subjected to various adjustments, and an interspecies scaling factor is applied to derive the slope factor for humans. ***Figure C.2*** illustrates a hypothetical dose-response curve for a carcinogen. The SF is used to determine the number of tumours likely to occur at low doses below which experimental data do not exist. The extrapolation is forced through the origin since for carcinogens NOAELs are not predicted to occur; i.e. only zero exposure equals zero risk.

Figure C.2 Hypothetical Example of a Dose-response Curve for a Carcinogen



Amongst the potential COCs are several substances that exhibit route-specific toxicity. Inhalation of Cadmium, Chromium VI and Nickel has been associated with increased incidence of cancer in animals and/or humans. However, no adequate evidence exists for systematic carcinogenic effects following oral exposure to these compounds, either because the substances may not be available for absorption through the gastrointestinal tract, or because they may cause lung cancer by a mechanism which has no parallel in the gastrointestinal tract. In this assessment the main purpose is to evaluate risks associated with the ingestion of seafood and hence only the oral SFs are of interest. Oral SFs have been summarised in ***Table C.2***.

C.1.3.2 Selection of Assessment Endpoints and Measures of Effect (Measurement Endpoints) for Human Health

Measurement endpoints for the human health risk assessment will include:

- Incidence of cancer in humans (for carcinogenic substances); and

- Incidence of chronic conditions in humans (for non-carcinogenic substances).

For the purpose of this assessment, exposure parameters representing the "typical" or "average" individual were selected. It is assumed that values protective of this individual will be protective of the majority of the exposed population. Measurement endpoints can be evaluated either through direct or indirect measurements. These measurements are referred to as measures of effect. Measures of effect are measurable responses to stressors that may affect the characteristic component of the measurement endpoints (5) (6). While some contaminants may influence only one characteristic, other contaminants may affect more than one characteristics (see ***Table C.1***). Therefore, the risks are assessed as a whole, and are not specified by receiving system.

C.1.4 *Exposure Assessment*

C.1.4.1 Introduction

The purpose of an exposure assessment is to determine the intake of each COC by potentially exposed individuals. In this Study, this will involve characterisation of the major pathways for contaminant transport leading from the CMPs to the points of exposure. Exposure evaluation considers various routes of contaminant release and migration from the CMPs to targeted populations by:

- Evaluating fate and transport processes for the contaminants;
- Establishing likely exposure scenarios for each medium (e.g. water, diet, etc);
- Determining the concentrations of the contaminants in each medium;
- Determining exposures to potentially affected populations; and
- Calculating maximum short-term or average lifetime doses and resultant intakes.

The resultant doses to and intakes by potentially exposed populations are calculated once exposure concentrations in all relevant media have been determined. Dose is defined as the amount of chemical contacting body boundaries (skin, lungs or gastrointestinal tract) and intake is the amount of chemical absorbed by the body. When the extent of intake from a given dose is unknown, or cannot be estimated defensibly, dose and intake are taken to be the same (i.e. 100% absorption from contact). This is a highly conservative approach and there are very few instances in which 100% of a chemical is absorbed in this manner.

Figure C.3 provides a conceptual model to aid the assessment of contaminant exposures to humans. The model is used to illustrate the relationship between the stressors (COCs) and the receptors of concern (i.e. humans). The conceptual model integrates the available information to identify exposure pathways. Each exposure pathway will include the stressor source (dredged material disposal activities), the stressor of concern (COCs), the exposure route (ingestion), and the receptor of concern (i.e. humans). The basic premise of the model is to evaluate the toxicological effects of the contaminants of concern associated with disposal activities at the CMPs.

Substances potentially migrating from the CMPs into the marine environment will be dispersed into the ambient environment and may potentially impact human populations through ingestion of contaminated sediment, ingestion of dissolved and suspended contaminants in water, ingestion of organisms with contaminant residues in their edible portions and through contact with water. Of these four (4) pathways the primary pathway of concern is considered to be that of the ingestion of contaminants contained within the edible portion of marine organisms.

The impact hypotheses for the assessment of human health risks are thus defined as follows:

(5) Suter GW (1990) Endpoints for regional ecological risk assessments. Environmental Management 14:19-23
(6) Suter GW (1993) Ecological Risk Assessment. Lewis Publishers, Boca Raton, Florida, USA

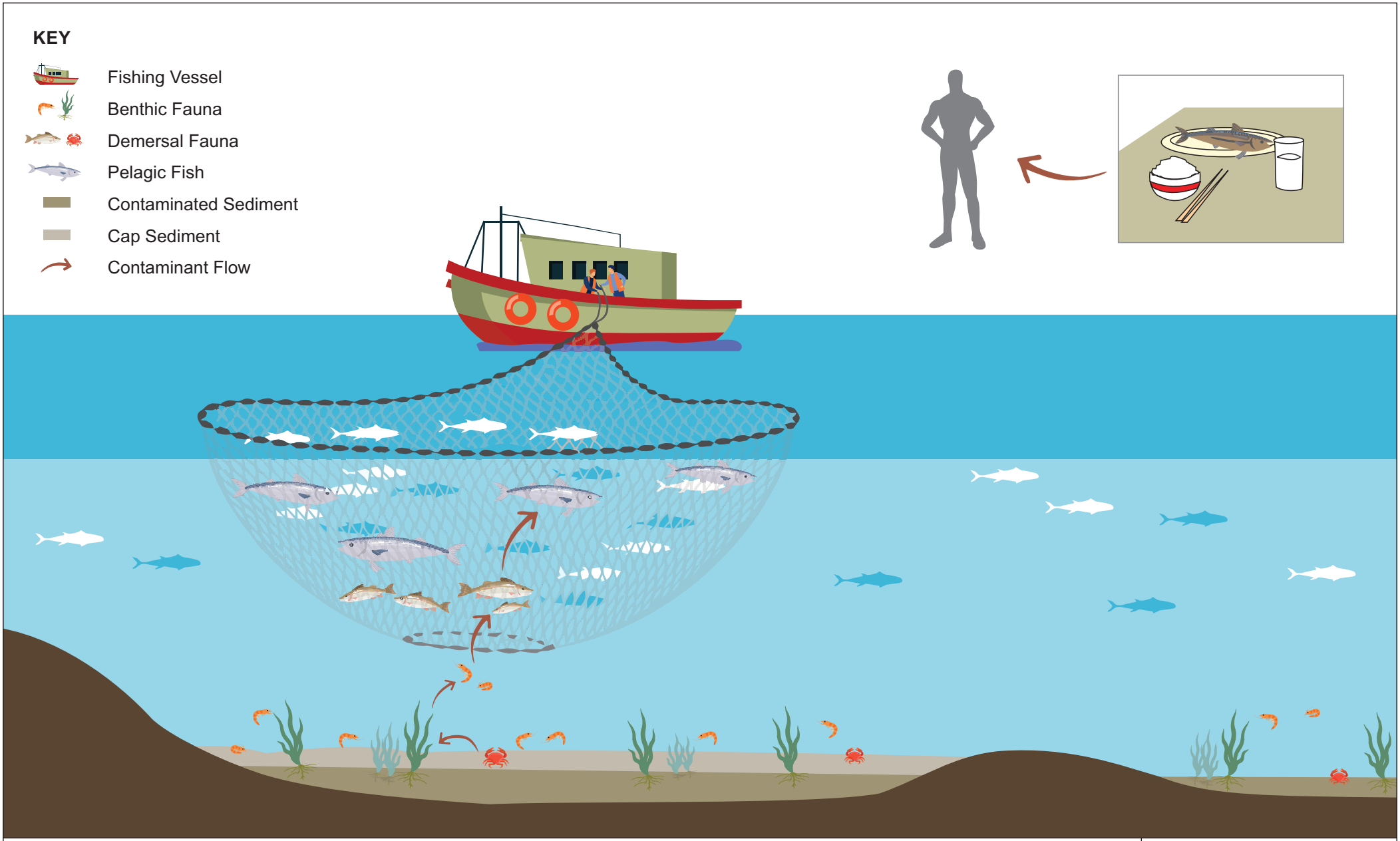


Figure C.3

Indicative Pathways to Potential Contaminant Release

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Environmental Resources Management

ERM

IH_1: Risks to human health from consumption of commercial species captured adjacent to the WL CMPs are no greater than risks associated with consumption of species remote from the WL CMPs; and

IH_2: Risks to human health from consumption of commercial species captured adjacent to the WL CMPs are below the detectable levels defined by the screening risk criterion.

C.1.4.2 Human Health Risk Assessment

The general equation used to estimate exposure is presented below.

$$Intake\ (mg\ kg^{-1}day^{-1}) = \frac{CF\ x\ IR\ x\ FI\ x\ EF\ x\ ED}{BW\ x\ AT}$$

where,

CF	=	Contaminant Concentration in Seafood (mg kg^{-1} wet weight)
IR	=	Seafood Net Ingestion Rate from WL Area (kg day^{-1})
FI	=	Fraction Ingested from Contaminated Source (unitless)
EF	=	Exposure Frequency (day $year^{-1}$)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged in days)

The relative contributions of each dietary item to the total intake are then included in the calculation to give an indication of overall exposure via seafood ingestion. Input values have been calculated to reflect local conditions and are discussed below.

Although AFCD's most recent Port Survey was conducted in 2016-2017, the data published do not contain some of the necessary details for this assessment. In particular, no current information on the percentage catch of the target species for Hong Kong or the local study area was available. However, more recent information (i.e. 2018-2019) is available from AFCD's Annual Departmental Report such as the annual marine fish consumption in Hong Kong.

C.1.4.2.1 Contaminant Concentrations

The data used in this assessment are the 95th percentile values obtained during the monitoring of tissue contaminant concentrations at ESC reference area between January 2016 and February 2021. These values represent the high end of the range and are likely to result in high estimates of risk [(7)]. The 95th percentile values are typically outlying high values which have lower opportunities to be encountered by the receptors and are thus considered highly conservative for the risk assessment. For comparison purposes the assessment also summarises the risks associated with consumption of seafood using 50th percentile data on contaminant concentration, which is considered as better representing the average situation (i.e. in a normal distribution the arithmetic mean equals to the 50th percentile value) that is more likely to be encountered by the receptors. Using both percentile values provide estimates of risks from both a highly conservative and more realistic point of view.

C.1.4.2.2 Ingestion Rate (Seafood Consumption Rate)

The rate of seafood consumption is a key exposure variable for use in this risk assessment. Seafood is known to be an important component of the diet of Hong Kong residents and it is estimated that the amount consumed daily is an order of magnitude higher than that consumed in other countries, such as the US [(8)]. The seafood consumed in Hong Kong is derived from a wide variety of sources:

- Imported from overseas as live, fresh, chilled, frozen, canned, preserved, salted, smoked or dried forms;

(7) USEPA (1992) Guidelines for Exposure Assessment. http://www.epa.gov/raf/publications/pdfs/GUIDELINES_EXPOSURE_ASSESSMENT.PDF
(8) Per Capita Consumption 2011 http://www.st.nmfs.noaa.gov/st1/fus/fus11/08_percapita2011.pdf

- Landed by the Hong Kong fishing fleet but caught outside of Hong Kong waters; and
- Landed by the Hong Kong fishing fleet and caught within Hong Kong waters.

According to the latest available information contained in the AFCD Annual Report 2018-2019 (9), the annual consumption of seafood in Hong Kong was 193,611 tonnes in 2018 (10). The population of Hong Kong was approximately 7,451,000 in the same year (11), as such 25.98 kg yr^{-1} or 74.23 g day^{-1} (based on 350 days) of seafood were consumed per person in 2018/19. The above per capita seafood consumption rate is assumed for the purpose of this assessment.

For sectors of the population that consume comparatively more fisheries products, e.g. fishermen, the USEPA recommends using a gross consumption rate of 300 g day^{-1}. This rate is considered to be an upper bound and rarely expected to occur in reality. Consequently the maximum consumption rate has been applied to the fishermen populations, i.e. Hong Kong Fishermen and WL Fishermen.

The values above are likely to be an overestimate as the amount actually ingested will be lower due to molluscs, crustaceans and fish having shells, viscera and skeletal structures. Conversion factors that can be used to convert gross seafood ingestion rates into tissue-specific ingestion rates as presented in Shaw (1995) (12). These values were higher than those suggested for use by the US National Marine Fisheries Service (NMFS) because it was considered that more of the seafood product is eaten in eastern cultures, such as internal organs (e.g. swim bladder or crab hepatopancreas) which do not usually form part of the western diet. For the purposes of this risk assessment the following factors have been applied to calculate net consumption rates for each dietary item:

- Shrimps / Prawns = 0.88 (maximum value from NMFS 1987 (13))
- Swimming Crabs = 0.22 (14)
- Fish = 0.5 (15)
- Molluscs / Bivalve = 1.0

The risk assessment calculations for ingestion rate were proportioned into the different dietary items. It was assumed that the proportion of each dietary item in catches in Hong Kong would reflect the proportion in the diet of Hong Kong people and the fishermen working around the Study Area. The composition of the catch in Hong Kong was identified using data from AFCD's Fisheries Study (16) presented below in ***Table C.3***. For the purpose of the risk assessment, all individuals were assumed to have a seafood dietary composition consistent with the WL catch composition.

Table C.3 Composition of Catches (%) in Hong Kong (ERM 1998)

Type	Hong Kong Catch
Pelagic Fish	41.7
Predatory Fish	46.8
Predatory Crab	3.0

(9) AFCD (2019) Departmental Annual Report 2018-2019. Accessed via <https://www.afcd.gov.hk/misc/download/annualreport2019/en>
(10) The value was derived from dividing the sum of local consumption of local production in capture fisheries (34,000 tonnes) and mariculture sector (850 tonnes) in 2018 by the percentage of all seafood consumed in Hong Kong being accounted by the local production (18%).
(11) Census and Statistics Department (2019) Population and Household Statistics Analysed by District Council District. Accessed via <https://www.statistics.gov.hk/pub/B11303012019AN19B0100.pdf>
(12) Shaw BJ (1995) Evaluation of Risks to Human Health in Hong Kong from Consumption of Chemically Contaminated Seafood: A Risk Assessment Approach. MSc Thesis. The University of Hong Kong.
(13) NMFS (National Marine Fisheries Service) (1987) Fisheries of the United States, 1987. Current Fisheries Statistics. No. 8700. US Government Printing Office, Washington DC
(14) NMFS (1987) *Op cit.*
(15) Shaw BJ (1995) *Op cit*.
(16) ERM (1998) Fisheries Resources and Fishing Operations in Hong Kong Waters. Prepared for the Agriculture and Fisheries Department

Type	Hong Kong Catch
Predatory Shrimp	6.1
Molluscs	2.4

After application of the conversion factor data and the catch composition/ dietary fraction information presented above to the gross seafood consumption rate (74.24 g day^{-1} for general public / 300.0 g day^{-1} for fishermen), consumption rates were then calculated for each dietary item in g day^{-1}. The resultant total net seafood consumption rates after application of the conversion factors are 39.11 g day^{-1} and 158.03 g day^{-1}, respectively, for a Hong Kong person and for a Hong Kong Fishermen (***Table C.4***).

Table C.4 Daily Net Consumption (i.e. Edible Tissues Only) of Each Dietary Item

Dietary Item	Net Consumption (g day^{-1}) for a Hong Kong person's diet	Net Consumption (g day^{-1}) for a fisherman's diet
Pelagic Fish	15.48	62.55
Predatory Fish	17.37	70.20
Predatory Crab	0.49	1.98
Predatory Shrimp	3.99	16.10
Molluscs	1.78	7.20
Total	**39.11**	**158.03**

C.1.4.2.3 Fraction Ingested from Contaminated Source

It is unlikely that 100% of the seafood consumed by an individual will be from the same source. For this risk assessment, the Fraction Ingested (FI) value estimated represents the fraction of total seafood ingested from the WL area.

The catch from the old AFD fishing zones in the WL area (0029, 0030, 0096, 0097, 0098, 0109) amounts to a total of 1,873 tonnes per year [17]. The total amount of seafood products consumed in Hong Kong was 243,440 tonnes per year in 1999 [18]. The fraction of this amount obtained from the WL area is therefore 1,873 ÷ 243,440 = **0.008.**

Estimates of the FI have been prepared for three exposure populations of concern, which are as follows:

- **Hong Kong People**: It is assumed that this population experiences the average exposure to COC in seafood. The FI for this population is represented by the value derived above, *i.e.* **0.008**. This indicates that 0.8% of the seafood consumed by Hong Kong people is obtained in the WL area. Information on the contribution of seafood to the total diet of Hong Kong People is not needed in this risk assessment as the methodology is concerned with the effects of contaminants in the edible portion of seafood on human health.
- **Hong Kong Fishermen:** Calculating the values for this population is more speculative due to uncertainties over the amount of a fisherman's diet that is composed of seafood. The USEPA estimate that 75% of a fishermen's diet will originate from within local waters (defined as the whole of Hong Kong). The AFCD's 1996-1997 Port Survey Report [19] indicated that the total catch landed in Hong Kong is 186,000 tonnes per year of which 17,681 tonnes per year has been

([17]) Agriculture & Fisheries Department (1998) *Op. Cit.*
([18]) ERM (2007) *Op. cit.*
(19) Agriculture & Fisheries Department (1998) *Op. cit.*

estimated to have been caught in Hong Kong waters [20]. This indicates that 10.6% (1,873 tonnes in WL / 17,681 tonnes in Hong Kong waters) of the Hong Kong catch comes from WL and the FI is thus set at **0.08** (10.6% × 75%). This indicates that eight percent (8%) of the seafood consumed by Hong Kong Fishermen is obtained in the WL area. This population is comparable to the Reasonable Maximum Exposure used in previous risk assessments [21] [22].

- **WL Fishermen**: For this population it is assumed again that 75% of the diet is obtained in local waters, but this time local refers to catches landed at the home port within the WL area (Lamma). The fishing fleet that operate from Lamma obtain 65% of their catch within the WL area. Hence the FI for these fishermen is estimated at **0.49** (65% x 75%). This indicates that 49% of the seafood consumed by WL Fishermen is obtained in the WL area. This population is comparable to the Sensitive Subpopulation used in previous risk assessments [23] [24].

Multiplying the FI for each population of concern with the daily net consumption of each dietary item (***Table C.4***) provides an estimate of net Ingestion Rate (IR) which represents the net consumption rate of a particular dietary item sourced from the WL area. These net IR for WL-sourced dietary items are presented below in ***Table C.5***.

Table C.5 Net Ingestion Rate (IR) of Individual Dietary Items from the WL area for the Three Populations of Concern

Type	HK people (g day^{-1}) FI = 0.008	HK Fishermen (g day^{-1}) FI = 0.08	WL Fishermen (g day^{-1}) FI = 0.49
Pelagic Fish	0.12	5.00	30.65
Predatory Fish	0.14	5.62	34.40
Predatory Crab	0.00	0.16	0.97
Predatory Shrimp	0.03	1.29	7.89
Molluscs	0.01	0.58	3.53
Total	**0.31**	**12.64**	**77.44**

C.1.4.2.4 Exposure Frequency

The exposure frequency is the average number of days per year over which an individual is exposed to one or more COC via ingestion of seafood. A value of 350 days, as specified by the US EPA [25] for long term average contact, has been assumed for this assessment.

C.1.4.2.5 Exposure Duration

The exposure duration is the time period in years over which an individual is exposed to one or more contaminants in seafood from WL. For the purposes of this assessment we have adopted the lifetime of the WL Facility, i.e. twenty (20) years.

(20) ERM (1998) *Op. cit.*
(21) Shaw BJ (1995) *Op. cit.*
(22) EVS (1996) *Ibid*
(23) Shaw BJ (1995) *Ibid*
(24) EVS (1996) *Ibid*
(25) US EPA (1991) Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors. Office of Solid Waste and Emergency Response. OSWER Directive 9285.6^{-3-3}. Washington, DC

C.1.4.2.6 Body Weight

US EPA guidelines for risk assessment [26] indicate that the default value recommended for body weight (BW) is 70 kg. However, Asians are in general smaller in stature than Caucasians, so the US EPA default value is not representative of the Hong Kong population. Instead, a value of 60 kg was used to represent the local Hong Kong population as determined by Shaw (1995) [27].

C.1.4.2.7 Averaging Time

The averaging time (AT) is another important parameter of the intake equation. The AT selected will depend on the type of constituent being evaluated, for example, to assess long term or chronic effects associated with exposure to non-carcinogens, the intake is averaged over the exposure duration (expressed in days). Exposure to carcinogens, however, is averaged over a lifetime in order to be consistent with the approach used to develop SFs. The mean life expectancy for Hong Kong people is 82 years for men and 88 years for women [28]. Averaging time of 82 years was adopted in this risk assessment for the carcinogenic risk assessment which would result in a higher intake than assuming averaging time of 88 years and is thus considered more conservative. For non-carcinogenic risk assessment, the averaging time adopted was twenty (20) years (i.e. the lifetime of the WL Facility).

C.1.4.2.8 Summary

A summary of the values incorporated into the human health risk assessment are presented below in ***Table C.6***.

Table C.6 Summary of Input Parameters for Intake Equation for Human Health Risk Assessment

Variable	Values
Contaminant Concentration in Seafood (mg kg^{-1} ww) (CF)	To be determined from the data of biomonitoring programme
Net Ingestion Rates (IRs)	Presented in ***Table C.5***
Exposure Frequency (EF)	350 days yr^{-1}
Exposure Duration (ED)	20 years
Body Weight (BW)	60 kg
Averaging Time (AT)	Non-carcinogen: (20 years x 365 days = 7,300 days) Carcinogen: 29,930 days (based on a 82 year life expectancy)

C.1.5 Risk Characterisation (Risk Calculation)

C.1.5.1 Introduction

Risk characterisation generally involves the integration of the information and analysis of the first three components of the assessment, as discussed in ***Sections C.1.2***, ***C.1.3*** and ***C.1.4***. Risk is generally characterised as follows:

- For non-carcinogens, and for the non-carcinogenic effects of carcinogens, the margin of exposure is calculated by dividing an estimated daily dose by a derived "safe" dose to form a ratio. This ratio is referred to as a Hazard Quotient and if it is greater than one (1) there is sufficient concern for further analysis.

([26]) US EPA (1989) Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish. A Guidance Manual. EPA-503/8-89/002

([27]) Shaw BJ (1995) *Op cit*.

([28]) http://www.censtatd.gov.hk/statistical_literacy/educational_materials/statistics_and_you/index.jsp

- For carcinogens, risk is calculated by multiplying the estimated daily dose by the risk per unit of dose. A range of risks might be produced, using different models and assumptions about dose-response curves and the relative susceptibilities of humans and animals.

Although this step can be more complex than is indicated above, especially if issues of the timing and duration of exposure are introduced, the hazard quotient and the carcinogenic risk are the ultimate measures of the likelihood of injury or disease from a given exposure or range of exposures. This sub-section describes the approach used to assess the overall risks of fish and shellfish ingestion to humans. The approaches used are independent of each other to a large degree, and are presented separately.

C.1.5.2 Human Exposure

C.1.5.2.1 Non-carcinogens

The intakes, calculated using the data presented in ***Table C.6*** and the equation in ***Section C.1.4.2***, will be compared with the Reference Doses (RfDs) (see ***Table C.2***) as a means of calculating non-carcinogenic hazards, which are expressed as the Hazard Quotient (HQ).

$$Hazard\ Quotient = \frac{Intake}{Reference\ Dose\ (RfDs)}$$

HQs can be summed to provide an estimate of the cumulative non-carcinogenic hazard which is known as the Hazard Index (HI). This is a conservative approach and assumes that all of the COCs exert an effect on the same target organ.

C.1.5.2.2 Carcinogens

Carcinogenic risks were calculated using the following equation:

$$Risk = Intake\ x\ Slope\ Factor$$

This equation will provide an estimate of the lifetime carcinogenic risk associated with the estimated intake.

C.1.5.2.3 Additive Effects

Concern is often expressed about the hazard to health from exposure to mixtures of substances, rather than individual substances. There is no agreed procedure among toxicologists for estimating such a hazard. The toxic effects of two substances in combination may be the sum of the individual toxicities (i.e. additive), more than the sum (i.e. synergistic), or less than the sum (i.e. antagonistic). The available literature on antagonistic or synergistic effects is very limited and, where it does exist, is largely restricted to the behaviour of metals in experimental animals. The application of such data to human studies is, at best, questionable. In the absence of any well-established scientific basis for predicting antagonistic or synergistic reactions in complex mixtures, only examination of an additive model of toxicity is considered.

There are two related methods of making some quantitative assessment of the toxic impact of a mixture. The first method, recommended by the UK Health and Safety Executive (HSE), is to use the following equation for non-carcinogens:

$$\frac{C_1}{L_1} + \frac{C_2}{L_2} + \frac{C_3}{L_3} \ldots + \frac{C_n}{L_n} = X$$

where C_1, C_2, C_3...C_n are the concentrations of each contaminant in food and L_1, L_2, L_3...L_n = the "safe levels" of each, i.e. the reference dose RfD. If the total X is less than one (1), the mixture is considered not to represent a health hazard, whereas, if X is greater than one (1), steps should be taken to reduce the concentrations of one or more of the contaminants.

The second method details risk calculation for carcinogens, in which a conservative approach is achieved using the "response-addition" process. This process simply sums the individual lifetime risks linearly to reflect the combined potential of cancer should a person be exposed to all of the substances over a lifetime.

Total Excess Cancer Risk = Risk 1 + Risk 2 + Risk 3 + ... Risk "n"

where, Risk 1 = Individual excess cancer risk [29] from a lifetime exposure from the first substance;

Risk "n" = Individual risk of additional substances.

While the "response-addition" process is encouraged as a "first-cut" or screen to indicate that a cancer may occur from the exposure to multiple substances, it should be remembered that the conservative nature of risk assessments for individual substances can be exaggerated by this additive approach.

C.1.6 *Assumptions and Uncertainties*

The risk estimates generated in this investigation are based on a considerable number of assumptions, uncertainties and variability associated with each step in the risk assessment process. According to USEPA guidelines these assumptions and uncertainties should be presented along with the results so that a fully informed picture is given to decision makers [30] [31].

The approach presented here relies on conservative, upper-bound estimates, such as the 95th percentile contaminant concentration, and results in a very conservative estimate of risks. The uncertainties associated with each step of the risk assessment are detailed below:

- **Hazard Identification**: This stage is based on data for which detection, identification and quantification limits could introduce errors. The selection of COCs in this assessment was made according to the list from the EM&A Manual of ESC CMP [32] which, though not an exhaustive list, was the best available reference for the purposes of this assessment. Other chemicals may pose a threat to human health and exclusion from this investigation does not infer that they are not of concern.
- **Dose-response Evaluation**: The toxicity assessment stage has a very high degree of uncertainty associated with the slope factors and reference doses. In future assessments the toxicological information should be revisited and updated using the latest available information.
- **Exposure Assessment**: This stage depends heavily on the assumptions made about the pathways, frequency and duration of exposure to COC. It should be noted that this risk assessment is focussing only on the exposure pathway concerned with consumption of seafood from within a specific area and seafood from other sources and exposures from foods other than seafood have not been taken into account. Although this is not the complete exposure pathway, it is, for the most sensitive sub-population (Fishermen at WL), likely to be the major pathway for exposure to the COC of interest to this Study. Exposure to the COC via other pathways, such as via air (inhalation), water (drinking) and dermal contact are expected to be minor.
- **Risk Characterisation**: The computation of screening-level risk is an exercise in applied probability of extremely rare events (for example acceptable lifetime risk for the purpose of this assessment is set at 1 x 10^{-3} year^{-1}), therefore not every conceivable outcome can be evaluated. This introduces an inherent conservatism which often results in assessing a scenario that will likely never be experienced.

([29]) Excess cancer risk refers to the excess risk of cancer from exposure to a chemical which is described in terms of the probability that an exposed individual will develop cancer because of that exposure.

([30]) US EPA (1989) Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish. A Guidance Manual. EPA-503/8-89/002

([31]) LaGrega MD, Buckingham PL, Evans JC, ERM Group (1994) Hazardous Waste Management. McGraw-Hill Inc 1146pp

([32]) ERM (2017) Updated EM&A Manual for ESC CMP V. Prepared under Agreement No. CE 63/2016 (EP).

In summary, risk assessment by design is very conservative and incorporates features such as Uncertainty Factors so that potential exposures and risks are unlikely to be understated. Despite varying degrees of uncertainty surrounding risk assessments, they represent the most useful tools that can be used to determine and protectively manage the risk to human health under the situation of limited available information [33].

([33]) Institute for Environment and Health (2003) Uncertainty factors: their use in human health risk assessment.