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Risk comparisons relevant to sea disposal of low level radioactive waste

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FOREWORD

The Contracting Parties to the Convention on the Prevention of Marine Pollution by Dumping of Wastes and Other Matter (London Convention 1972, LC 72), formerly referred to as the London Dumping Convention (LDC), have designated the International Atomic Energy Agency as the competent international organization in technical matters relating to radioactive waste. In accordance with its mandate, the IAEA has periodically formulated a definition of high level radioactive waste unsuitable for dumping at sea as specified in Annex I to the Convention, most recently in 1986. The IAEA has also provided recommendations regarding the forms, conditions and methods for dumping of other radioactive matter under the Convention, as well as guidance on the nature and content of environmental assessments of dumping activities.

Dumping of low level radioactive wastes at sea was discontinued in 1983 when the Contracting Parties to the LC 72 agreed on a non-binding moratorium pending a review of the scientific, political, legal, economic and social aspects of this practice. The LC 72 subsequently requested the IAEA, in consultation with other international agencies (UNEP, UNESCO, WHO), to review and summarize available scientific information on estimates of risks, both voluntary and involuntary, that result from various human activities to provide a basis for comparison with risks resulting from the dumping at sea of radioactive wastes.

To fulfil this task, the IAEA convened an Advisory Group Meeting on the Risks from Sea Dumping Compared with Other Risks in July 1989. This meeting considered a working document prepared at a consultants meeting held in December 1988. The report was finalized during further consultants meetings held in the period 1990 to 1992 taking into account the comments received from the Inter-Governmental Panel of Experts on Radioactive Waste Dumped at Sea (IGPRAD) and other experts in the field.

EDITORIAL NOTE

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CONTENTS

SUMMARY

This document contains estimates of, and comparisons among, risks to human health posed by exposures to radionuclides, including those associated with low level radioactive waste dumping at sea, and organic chemical contaminants resulting from seafood consumption. This study was conducted at the request of the Contracting Parties to the Convention on the Prevention of Marine Pollution by Dumping of Wastes and Other Matter (the London Convention 1972, formerly referred to as the London Dumping Convention) as a component of a review of the wider political, legal, economic and social aspects of sea dumping of radioactive wastes.

Section 1 places this study in the context of the request from the LC 72 and previous studies and publications relating to sea disposal of radioactive wastes.

Section 2 explains the concepts of risk, hazard, harm and safety and defines risk in terms of the probability that something unpleasant will occur. It presents a framework for the assessment of risk and illustrates the range of risks to human health to which individuals within society are commonly subjected. This section also provides an explanation of the concept of harm to populations and briefly refers to the basis for assessing acceptability of risk. Finally, Section 2 explains the basis for the estimation of the risks of fatal cancer associated with exposures to radiation and some chemicals suspected to be human carcinogens.

Section 3 provides a more detailed explanation of how exposures to radionuclides and chemical carcinogens are converted into potential risks expressed in terms of probability. Sections 2 and 3 together provide the basis for the estimation and comparison of risks in the remainder of the document. The common basis of comparison is the potential risk of fatal cancer induction in humans through the consumption of marine foodstuffs containing natural radionuclides and radioactive and chemical contaminants.

Section 4 deals with estimates of potential harm, in terms of cancer fatalities, to large populations posed by the presence of radionuclides and suspected chemical carcinogens in seafood.

Section 5 deals with estimates of the risks to individuals associated with the consumption of seafood contaminated by radionuclides and suspected chemical carcinogens. Such risk estimates are first presented for the 'average' individual within large populations. Then, the potential risks to individuals within a critical group of extreme seafood consumers are calculated as a means of obtaining complementary information on the most extreme individual risks.

In Section 6, comparisons are made among the estimates within each of the three representations, that is, harm to entire populations, the average individual risks to members of those populations, and risks to members of the critical group. The magnitudes of potential risks and health detriments associated with seafood consumption in these three categories are discussed.

The highest potential risks associated with seafood consumption are generally those resulting from exposures to naturally occurring radionuclides. In some representations, the potential risks associated with polychlorinated biphenyls (ΣPCB) and dieldrin in seafood are of the same order as those arising from naturally occurring radionuclides. The peak annual risks resulting from low level radioactive waste dumping at sea, assessed on any rational basis, are at least two orders of magnitude lower than those associated with the ingestion of common organic chemical contaminants in seafood.

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1. INTRODUCTION

Ocean disposal of packaged low level radioactive wastes has taken place at various sites in the oceans since 1945. In 1958, the United Nations Conference on the Law of the Sea concluded that "every State shall take measures to prevent pollution of the sea from dumping of radioactive wastes, taking into account any standards and regulations which may be formulated by competent international organizations" [1]. Pursuant to its responsibilities, the IAEA set up successive scientific panels to provide specific guidance and recommendations for ensuring that disposal of radioactive wastes into the sea would not result in unacceptable hazards to man and marine organisms. Since 1957, a series of meetings of experts, and of publications, has reflected progress towards this goal. A first IAEA Experts Meeting on Radioactive Waste Disposal into the Sea, chaired by H. Brynielson, and held in 1957, resulted in the publication of IAEA Safety Series No. 5 "Radioactive Waste Disposal into the Sea" [2]. This was followed in 1960 by a meeting on the Administration, Organization and Legal Measures to Implement Safety Series No. 5. In 1965, the IAEA published "Methods of Surveying and Monitoring Marine Radioactivity" [3] and, in 1970, "Reference Methods in Marine Radioactivity Studies" [4].

Following the United Nations Conference on the Human Environment, held in Stockholm in 1972, the Convention on the Prevention of Marine Pollution by Dumping of Wastes and Other Matter (the London Convention 1972, formerly referred to as the London Dumping Convention) entered into force in 1975 [5]. The London Convention 1972 (LC 72) entrusted the IAEA with specific responsibilities for the definition of high level radioactive wastes unsuitable for dumping at sea, and for making recommendations to national authorities on matters concerning the issuance of special permits for ocean dumping of low level radioactive wastes. To fulfil its mandate, the IAEA in 1974 produced the "Provisional Definition of High Level Radioactive Waste Unsuitable for Dumping at Sea and Recommendations" [6]. Revised versions of the "Definition and Recommendations" were issued by the IAEA in 1978 [7] and in 1986 [8]. The most recent version of the "Definition and Recommendations" is supported by various documents such as "Control of Radioactive Waste Disposal into the Marine Environment" [9], "Environmental Assessment Methodologies for Sea Dumping of Radioactive Wastes" [10], the "Oceanographic and Radiological Basis for the Definition of High Level Wastes Unsuitable for Dumping at Sea" [11], "Sediment K_ds and Concentration Factors for Radionuclides in the Marine Environment" [12], and GESAMP Reports and Studies No. 19 "An Oceanographic Model for the Dispersion of Wastes Disposed of in the Deep Sea" [13].

In 1983, a non-binding resolution of the Contracting Parties to the Convention imposed a voluntary moratorium on dumping radioactive wastes at sea. In order to decide whether the Annexes to the London Dumping Convention warranted amendment to prohibit the dumping of radioactive waste, a Panel of Experts (PRAD) was established. In 1985, this Panel reported to the ninth Consultative Meeting of the Contracting Parties to the London Convention 1972. In its conclusions and recommendations, the Panel of Experts agreed that "no scientific or technical grounds could be found to treat the option of sea dumping differently from other available options when applying internationally accepted principles of radioprotection to radioactive waste disposal" [14]. At the tenth Consultative Meeting in 1986, a further Inter-Governmental Panel of Experts on Radioactive Waste Dumped at Sea (IGPRAD) was established [15] to examine the wider political, legal, economic and social aspects of the sea dumping of radioactive wastes within the terms of reference defined in Resolution 21(9) of the ninth Consultative Meeting [16]. IGPRAD activities and conclusions are periodically reported to the London Convention 1972 and are expected to be completed in 1993.

In connection with IGPRAD work, the Contracting Parties to the London Convention 1972 requested the IAEA to carry out several supporting studies, of which some have been completed, such as "Assessing the Impact of Deep Sea Disposal of Low Level Radioactive Waste on Living Marine Resources" [17], "Estimation of Radiation Risks at Low Dose" [18], "Low Level Radioactive Waste Disposal: An Evaluation of Reports Comparing Ocean and Land Based Disposal Options" [19] and "Inventory of Radioactive Material Entering the Marine Environment: Sea Disposal of Radioactive Waste" [20].

During its first meeting in 1987 [21], IGPRAD recommended, and the contracting parties to the LC 72 subsequently confirmed the request, that the IAEA, in consultation with UNEP, WHO and UNESCO, "review and summarize available scientific information on estimates of risks, both voluntary and involuntary, to human well-being that result from various human activities to provide a basis for comparison with those risks estimated in the Report of the Expanded Panel [14], to result from the dumping at sea of radioactive wastes carried out under the provisions of the London Dumping Convention and pursuant to the Recommendations of the IAEA. Where possible, risk estimates from other human uses, applications, disposals and disseminations of potentially hazardous substances should be included. All measures of risk should be carefully defined so that uniform bases of comparison can be developed".

At its second meeting in 1988 [22], IGPRAD stated that "when considering risks from a somewhat broader perspective, it was suggested that there may be additional ways of expressing risks to human health than merely probability of fatality. For example, should consideration be given to risk of non-fatal, curable, disease induction and to the decreased marketability of fisheries products due to consumer perceptions of their susceptibility to radioactive contamination? It was agreed that while perceptions of risk warranted consideration and that there were other bases for the expressions of risk, it was important that the compilation of risks from a variety of sources should be presented on a common and comprehensible basis. Furthermore, the basis chosen should be one which would permit the quantification of risks from a broad range of human and industrial practices. It was consequently agreed that, in the first instance, the compilation of risks would probably most appropriately be based on risk of fatality per annum. The IAEA was, nevertheless, requested to include in the report a discussion of other types of risk expression, and perceptions of risk, to the extent warranted in making the study of greater value in the London Dumping Convention forum".

The present document deals specifically with the request to the IAEA to undertake a comparison of risks from sea dumping of radioactive wastes with other risks prevalent in society. It first presents an explanation of risk and harm and outlines numerical values of the risks of fatality commonly experienced by members of the public. It then provides a basis, with definitions and explanation, for estimating and comparing risks that are most similar to those associated with sea dumping of low level radioactive wastes. The report then presents estimates of communal harm and individual risk associated with marine foodstuff pathways of human exposure to natural radionuclides, and to a small number of organic chemical contaminants assumed to be human carcinogens. These estimates of harm and individual risk are compared with the harm and risks associated with fatal cancer induction arising from sea dumping of radioactive waste.

It should be noted that this report discusses only the estimates of risks associated with dumping of radioactive wastes at sea in the North-East Atlantic area under the auspices of the Nuclear Energy Agency (NEA) and the provisions of the London Convention 1972 pursuant to the recommendations of the IAEA. It does not deal with risks associated with contamination of the ocean with radioactive material as a result of accidental losses of equipment and vessels or unnotified dumping operations.

The IAEA points out to readers that nothing in its publications should be construed as encouraging the dumping of radioactive wastes into the ocean. It also emphasizes that the

"Definition and Recommendations" set forth by the IAEA pursuant to the London Convention 1972 should not be interpreted as precluding the adoption of more restrictive requirements by appropriate national authorities. In particular, in the latest IAEA Revised Definition and Recommendations Concerning Radioactive Wastes and other Radioactive Matter [8], cited in Annexes I and II to the LC 72, the IAEA recommends that a general policy of continued isolation and containment of radioactive waste after descent to the sea-bed should be pursued, through the use of suitable packaging, to minimize the release of radionuclides, thereby preventing unnecessary contamination of the marine environment.

2. THE CONCEPT OF RISK

2.1. DEFINITION

There are many definitions of risk. In this report the term risk is used as the **probability** — the likelihood — that something unpleasant will happen. An associated term is that of hazard. A **hazard** is essentially a 'set of circumstances' that may result in harmful consequences. **Harm** is generally taken to include adverse effects on health or the quality of life; it can also be expressed in terms of loss, including loss of life, of working days, or of material things, such as environmental amenities. It is often possible, therefore, to represent losses as costs to society. Because costs are also incurred in reducing risks, the two sets of costs have often been used to estimate the optimum 'value for money' in terms of cost–benefit assessments of measures for reducing risk.

Harm to humans obviously includes death, injury and illness but can also include intangibles such as anxiety about suffering adverse health effects. Because anxiety depends on perceived risk, which may differ substantially from actual risk, quantification of the risk for anxiety is outside the scope of this document. Illustrative discussions of risk perceptions in relation to actual risks have been published (e.g. [23]).

The likelihood of a hazard resulting in harm is the probability of adverse effect, or the risk, associated with it. As a familiar illustration, the risk associated with the hazard of using cars (a set of circumstances) relates to the occurrence of accidents. The associated set of circumstances, or factors, which influence the likelihood of accidents, include engineering and mechanical characteristics, light, road and traffic conditions, and the quality of drivers (e.g., experience, consumption of alcohol or drugs). The qualitative part of risk analysis is termed 'hazard identification' and the quantitative part 'risk assessment'. Risk assessment can provide a predictive estimate of the total number of accidents per year in a population under a specified set of circumstances. It can also quantify the contribution to the overall risk of the various individual elements that comprise a set of circumstances. Thus, risk assessment is the basis for making policy decisions to reduce risk. Risk management is the process of making and implementing structured decisions based on the magnitude of risk. The aim of risk management is to decrease risk and, accordingly, increase safety.

In absolute terms, no set of human circumstances is entirely safe but, obviously, the lower the risk, the higher the degree of safety. The two terms (risk and safety) are therefore inversely related, and what most people perceive as being 'safe' actually corresponds to an acceptable level of risk. The upper limit for the acceptability of risk depends on how the risk is perceived by individuals or the community. Such acceptability can sometimes be judged in comparison with other levels of risk that are generally accepted by individuals or by the community at large.

For the purpose of risk assessment associated with human exposures to substances, different methods are used. When a waste material is released into the environment, hazard identification requires answers to the following questions:

(a) Has the agent the potential to harm humans through either its chemical or radiological properties?

(b) Can people be exposed to the agent and, if so, will there be adverse effects observed in the exposed population that can be linked to the presence of the agent?

For the quantification of risk, two types of data are required:

(a) Dose-response assessment, which is the relationship between the magnitude of exposure and the frequency of incidence of adverse health effects, derived from human epidemiological studies or from animal experiments; and

(b) Exposure assessment, which involves the determination of routes and magnitudes of external and internal exposure to substances.

Based on the dose-response relationship and exposure assessment, the probability of response for current, or predicted, exposures is calculated. Response is a narrower term than effect. Every toxic compound is able to cause effects of varying severity. Each effect changes gradually from mild to severe. For example, low level lead (Pb) exposure results in biochemical changes without clinical signs but, when lead exposure is high, anaemia develops with increasing severity. Unlike effect, response is a quantal change that is either present or not (i.e. it is stochastic). However, its prevalence in the exposed population increases with increasing exposure. In risk assessment for carcinogenesis, the chosen endpoint is most commonly death because: (i) in animal experiments, measurement of lethality (or potentially moribund condition caused by the presence of malignancies) is precise, quantal, and unequivocal; and (ii) epidemiological studies are primarily based on death certificates.

It should be appreciated that risks depend not only on the present level of exposure to a cancer causing agent but also on previous and future exposures. A wide range of time scales is involved in the overall incremental risk of cancer from various types of exposure. These include inter alia the time scales of environmental dissemination of cancer causing agents, the time scales of removal of such agents from the environment to locations to which humans are not exposed, the residence times of these agents in the body, and the time scales of decay and reaction to form new substances having different properties.

Thus, for example, as a result of the intake of a radionuclide into the human body, there is a commitment to radiation dose which is a function of the nature and rate of decay of the nuclide and the rate of its elimination from the body. In radiological protection, this commitment is referred to as the committed equivalent dose, for which the unit is the sievert (Sv).

2.2. RISKS TO INDIVIDUALS

The principal causes of death are cardiovascular disease, cancer, respiratory disease, accidents, violence, infectious disease, and poisoning; the relative ranking of these differs to some extent from country to country (Table I). Premature death is related to hazards in the home, transport, occupation, recreation, medicine and surgery, but can be influenced by personal habits — such as smoking, alcohol consumption and diet [24, 25]. To these causes may be added natural disasters and phenomena, plus the ubiquitous presence of man-made chemicals in the environment. These deaths can be separated into those which

TABLE I. PROBABILITY OF DEATH, PER YEAR, FROM SELECTED CAUSES IN UK AND USA POPULATIONS (the pertinent population and, where appropriate, the relevant sub-population is indicated in parentheses) [24, 25]

Cause of death	Annual probability
Cardiovascular disease [age 35–74] (UK)	9×10^{-3}
Cancer of any type (USA)	3×10^{-3}
All natural causes [age 40] (UK)	1×10^{-3}
Motor vehicle accident (USA)	2×10^{-4}
Air pollution [eastern] (USA)	2×10^{-4}
Influenza (UK)	2×10^{-4}
Accident at home (USA)	1×10^{-4}
Leukaemia (UK)	8×10^{-5}
Motor vehicle accident [pedestrian] (USA)	4×10^{-5}
Accident at home (UK)	4×10^{-5}
Accident at work [working force] (UK)	2×10^{-5}
Alcohol [light drinker] (USA)	2×10^{-5}
Homicide (UK)	1×10^{-5}
Aflatoxin in 4 tablespoons/day of peanut butter (USA)	8×10^{-6}
Electrocution (USA)	5×10^{-6}
Accident on railway (UK)	2×10^{-6}
Lightning (UK)	1×10^{-7}

are instantaneous — as in accidental deaths — and those which are a delayed result of chronic exposure to a hazard, such as exposure to a cancer causing agent.

2.2.1. Probabilities of fatality from various causes

There have been many tabulations of risk to individuals in different countries. These usually comprise estimates of risk associated with a variety of common hazards. Exposures to some hazards may be voluntary, such as those accepted by sports enthusiasts involved in relatively high risk recreational activities (e.g. hang-gliding and scuba-diving). Equally, activities such as smoking involve risks which are largely voluntary. The level of exposure to such hazards are controllable by the individual, e.g. the smoker can choose the amount and quality of cigarettes smoked. Similarly, an individual may wish to limit his exposure to natural hazards by choosing, for example, not to live in an earthquake prone area. Exposure to many other hazards is however involuntary, in the sense that an individual is neither able to appreciate nor avoid exposure to the hazard concerned.

Comparisons can be made among different occupational risks. The probabilities of fatality in this category range from about 2×10^{-3} per year for those engaged in sea fishing to less than 1×10^{-5} per year for those engaged in light industrial or office work.

TABLE II. LIFE EXPECTANCY, NUMBER OF SURVIVORS, AND CHANCES PER 1000 OF EVENTUALLY DYING FROM SPECIFIED CAUSES, AT SELECTED AGES, MALES, FOR YEAR STATED FOR SELECTED COUNTRIES [27]

COUNTRY	AGE	LIFE EXPECTANCY AT AGE X	NUMBER OF SURVIVORS TO AGE X OUT OF 100 000 AT BIRTH	CHANCES (PER 1000) OF EVENTUALLY DYING FROM					
				INFECTIOUS AND PARASITIC DISEASES	MALIGNANT NEOPLASMS	DISEASES OF THE CIRCULATORY SYSTEM	DISEASES OF THE RESPIRATORY SYSTEM	INJURY AND POISONING	MOTOR VEHICLE TRAFFIC ACCIDENTS
ARGENTINA 1986	0	68.5	100 000	27.3	181.2	496.3	62.9	65.3	12.2
	1	69.6	97 021	26.0	186.7	510.4	62.6	66.4	12.5
	15	56.2	96 128	25.4	187.5	514.1	62.5	63.5	11.8
	45	28.6	90 365	24.3	191.9	532.1	64.2	41.8	7.3
	65	13.9	68 054	22.9	176.3	559.9	71.2	29.8	4.2
CHILE 1987	0	70.0	100 000	31.5	182.7	310.0	128.8	94.0	9.4
	1	70.4	97 952	30.9	186.5	316.4	127.8	93.3	9.5
	15	57.0	97 153	30.6	187.3	318.9	127.8	90.2	9.1
	45	29.3	91 536	30.5	192.8	333.8	133.2	59.9	5.0
	65	13.7	72 664	28.3	181.9	357.0	147.8	36.2	2.1
CUBA 1988	0	72.0	100 000	11.2	210.0	469.9	95.9	96.2	
	1	72.0	98 658	10.3	212.8	476.1	95.9	97.1	
	15	58.5	97 839	9.6	213.8	479.9	96.2	93.7	
	45	31.1	91 986	9.4	221.3	500.6	100.5	63.2	
	65	15.3	75 434	9.3	216.1	517.6	110.8	47.3	
DENMARK 1988	0	72.2	100 000	4.4	255.0	452.4	76.1	69.8	14.0
	1	71.8	99 179	4.3	257.1	456.0	76.6	70.3	14.1
	15	58.1	98 793	4.1	257.6	457.7	76.8	68.8	13.2
	45	29.9	94 439	3.0	263.0	473.9	79.8	47.7	6.9
	65	14.2	75 580	2.5	249.6	501.0	88.1	37.2	5.2
GUATEMALA 1984	0	61.1	100 000	164.9	54.1	164.9	161.1	75.8	1.3
	1	64.8	92 756	163.8	58.3	177.7	160.1	81.4	1.4
	15	54.9	86 943	144.0	61.9	189.5	156.7	84.5	1.5
	45	29.5	77 469	139.0	66.1	206.6	163.4	50.4	0.4
	65	14.4	60 739	119.7	66.3	225.3	172.9	32.9	0.0
ICELAND 1989	0	73.2	100 000	2.4	266.2	506.9	99.6	53.7	6.5
	1	72.6	99 478	2.4	267.6	509.5	100.1	54.0	6.6
	15	61.7	99 295	2.5	267.6	510.5	100.3	53.2	6.1
	45	33.1	96 386	2.5	271.9	522.6	103.3	36.7	2.9
	65	16.0	84 230	2.9	251.5	538.7	116.1	24.9	2.3
IRELAND 1988	0	71.6	100 000	5.2	228.5	482.8	136.4	43.6	14.4
	1	71.3	99 049	5.2	230.6	487.4	137.5	43.9	14.6
	15	57.6	98 574	5.1	231.0	489.6	137.8	42.2	13.6
	45	29.0	95 261	5.0	233.1	501.2	141.4	25.7	6.5
	65	13.0	76 022	4.6	216.8	505.6	160.1	16.0	13.7
JAPAN 1989	0	76.2	100 000	14.8	262.2	368.1	148.6	57.3	14.6
	1	75.6	99 517	14.7	263.4	369.7	149.2	57.2	14.7
	15	61.8	99 119	14.7	264.0	370.9	149.5	55.7	14.2
	45	33.2	96 147	14.8	266.6	376.7	153.1	42.8	8.9
	65	16.5	82 718	14.5	242.7	394.8	170.5	31.7	6.5
KUWAIT 1987	0	72.5	100 000	33.2	118.7	463.9	91.7	60.3	44.6
	1	72.8	98 125	33.4	120.8	472.6	91.8	61.3	45.5
	15	59.4	97 328	33.5	121.1	476.3	92.3	57.4	42.7
	45	30.7	94 452	33.7	121.5	483.0	93.7	44.8	36.3
	65	14.5	76 997	37.3	112.3	467.3	100.4	34.1	27.7
MALTA 1989	0	73.8	100 000	6.1	212.5	497.6	97.4	39.9	5.9
	1	73.7	98 786	5.8	215.1	503.7	97.9	40.1	6.0
	15	59.8	98 612	5.8	215.5	504.6	97.7	39.8	6.0
	45	31.1	95 823	5.3	217.2	515.8	99.1	28.9	3.5
	65	14.7	79 440	4.9	189.0	533.8	110.8	24.9	2.0
MAURITIUS 1987	0	65.0	100 000	18.4	68.3	507.9	105.2	54.4	11.9
	1	66.0	97 052	16.8	70.4	523.1	107.3	55.1	12.2
	15	52.6	96 116	16.1	70.2	527.4	106.9	53.7	11.6
	45	25.3	88 575	14.6	71.6	549.1	110.4	33.3	6.8
	65	11.7	60 001	15.3	65.2	574.9	133.7	18.8	1.8
NEW ZEALAND 1987	0	71.4	100 000	4.5	230.8	472.9	122.6	63.7	22.5
	1	71.2	98 915	4.4	233.2	478.0	123.1	64.1	22.7
	15	57.6	98 302	4.2	233.4	480.8	123.7	61.2	21.4
	45	29.9	93 308	3.8	239.2	498.7	129.1	32.2	7.1
	65	14.0	75 280	4.0	225.0	503.2	145.9	23.1	4.7
SPAIN 1986	0	73.4	100 000	10.2	234.1	414.2	111.3	52.5	19.1
	1	73.2	98 983	10.0	236.4	417.8	111.8	52.6	19.2
	15	59.5	98 485	9.6	236.9	419.5	112.1	51.0	18.6
	45	31.4	94 213	9.2	240.5	430.7	115.4	33.2	10.2
	65	15.3	77 870	8.5	217.7	455.7	126.7	22.6	6.4
SRI LANKA 1985	0	66.2	100 000	44.9	38.1	190.6	55.3	97.3	5.7
	1	67.0	97 349	44.3	39.1	195.2	53.6	99.6	5.8
	15	54.1	95 714	41.6	39.4	197.6	52.3	97.8	5.7
	45	27.9	86 510	39.9	39.8	202.7	53.1	56.3	3.9
	65	13.5	64 227	31.4	27.5	165.9	45.8	35.0	3.1
SURINAME 1985	0	63.6	100 000	45.3	79.8	322.2	86.2	123.9	36.9
	1	64.8	96 690	41.1	82.5	332.9	87.4	128.2	38.2
	15	51.6	95 504	40.3	83.1	336.5	87.0	125.2	37.2
	45	26.4	82 835	41.9	93.3	361.7	91.4	74.8	26.7
	65	13.6	55 252	39.5	96.7	346.9	111.2	51.0	22.5
TRINIDAD AND TOBAGO 1986	0	68.1	100 000	17.6	134.8	443.6	69.9	74.1	19.3
	1	67.9	98 851	17.5	136.4	448.7	69.2	74.7	19.5
	15	54.4	98 143	17.0	136.7	451.8	68.6	71.9	18.3
	45	27.1	91 029	16.4	142.4	474.1	68.6	43.8	9.3
	65	12.9	65 310	16.0	151.1	487.0	76.8	28.3	5.5
UK 1989	0	72.7	100 000	4.2	257.2	459.5	127.7	35.1	9.9
	1	72.4	99 048	4.0	259.6	463.8	128.5	35.3	10.0
	15	58.6	98 648	3.8	260.1	465.5	128.7	33.9	9.2
	45	30.1	95 293	3.5	263.5	475.4	132.0	19.6	4.2
	65	13.9	77 749	3.2	248.0	476.9	147.9	13.5	2.8
UNITED STATES OF AMERICA 1988	0	71.6	100 000	13.3	227.8	452.4	98.3	68.8	19.8
	1	71.4	98 895	13.3	230.3	457.1	99.1	69.2	19.9
	15	57.7	98 367	13.2	231.0	459.3	99.4	66.7	19.0
	45	30.5	92 155	13.0	240.6	481.7	104.5	36.8	8.1
	65	15.0	73 529	13.1	223.3	501.5	116.9	26.7	5.2

Comparisons can also be made among risks associated with different recreational activities and pleasures. Such exposures are, however, controllable by the individual and, although interesting, the risks are of little direct relevance to this report. Nevertheless, in order to place the risk estimates provided later in this report into perspective, a list of selected risks of fatality for individuals in UK and USA populations is given in Table I [24, 25]. These values provide an order of magnitude appreciation of the annual probability of death associated with common causes. As shown in Table II, it should be noted that the probability of death associated with these hazards varies with age and location.

2.2.2. Risk of death from cancer

In the USA the annual probability of death from cancer is second only to that of heart disease. About 35% of cancer deaths are attributable to diet, 30% to smoking, 7% to reproductive and sexual behaviour, 4% to occupational hazards, 3% to geophysical factors, 3% to alcohol and 2% to pollution [26]. Pollution appears therefore to be responsible only for a small proportion of cancer deaths. Most of the cancer deaths in the geophysical factor category are associated with exposure to ultraviolet radiation. Only a small proportion of cancer deaths in this category could be attributed to radon.

2.2.2.1. Cancer risks associated with radiation exposures

There are two categories of health effects potentially associated with exposures to ionizing radiation. In the first category are those effects that occur above a certain level of threshold exposure. Their severity depends on the level and rate of exposure. Such effects are called **deterministic** but, in previous reports, they were commonly referred to as non-stochastic. They are not relevant to sea dumping of low level radioactive waste, because such high radiation doses are not associated with this practice. The other type of adverse effect is cancer, a **stochastic** response. The probability of cancer being induced by radiation increases with both the total integrated exposure and the rate of exposure. The relationship is assumed to increase linearly with dose without threshold [18]. Thus, risk factors for cancer induction are expressed as a probability per unit of annual or total dose. The International Commission on Radiological Protection (ICRP) refers to these as 'probability coefficients'. Other stochastic responses, which are not discussed here, are hereditary effects of radiation exposure. For a given dose, the ensuing number of cancers in the exposed individuals is believed to far outnumber future cases of serious hereditary defects [18, 28].

Estimates of risk per unit dose have frequently been revised. The value used in this report, as an average lifetime risk for fatal cancers arising from whole-body irradiation, for all ages and both sexes, is 5×10^{-2} per Sv [28]. (For further discussion of this conversion coefficient see Section 3.1). Thus, an increment of dose of 2 mSv implies an increment of risk of subsequently dying from cancer of 1×10^{-4}.

2.2.2.2. Cancer risks associated with chemical exposures

There exist some analogies between the effects of radiation and chemicals. Chemicals, like radiation, are able to induce cancer and are also able to cause increasingly severe effects with increasing exposure. Whether cancer induction by chemicals has a threshold is, however, a matter of some debate.

WHO Guidelines for Drinking Water Quality [29] adopted the linearized multi-stage model for low dose extrapolation and selected 10^{-5} as an acceptable level of lifetime risk. In 1987, a meeting on Potentially Toxic Microorganic Substances in Drinking Water [30] concluded that the earlier guidelines did not explain exactly how low dose risk calculations are carried out. The report of that meeting did not specify low dose extrapolation methods but recommended that a sufficiently comprehensive set of analyses be conducted with different models to enable the selection of an extrapolation method. This left the extrapolation method unspecified but confirmed the 10^{-5} lifetime risk level for carcinogens as a default value. Confirmation of a risk level, rather than the adoption of safety factors, also implied the acceptance of a no-threshold dose-response relationship for carcinogens. Accordingly, intakes corresponding to the 10^{-5} lifetime risk level using published slopes based on the multi-stage model were subsequently published [31]. The same approach for six organic chemicals assumed to be human carcinogens has been used here.

A more recent WHO environmental health criteria document [32], dealing with principles for toxicological assessment of pesticide residues in food, advocates that carcinogenic risk assessment be based on the concept of threshold and the use of safety factors. This document, however, only provides very general guidance on the use of safety factors. It argues that safety factors must be considered on a case-by-case basis because they depend on:

(a) The quality of data supporting No Effect Levels (NOELS) determined in animal experiments,
(b) The quality of the total database,
(c) The type and significance of the initial toxic response,
(d) The shape of the dose–response curve,
(e) Metabolic considerations,
(f) Knowledge of the comparative mechanism of toxic action in experimental animals and humans.

It also states that "where there is a need for a very high safety factor due to concern about the safety of a pesticide, it may be prudent to recommend that the pesticide should not be used where residues in food may occur".

The selection of safety factors for the calculation of acceptable or tolerable daily intakes is clearly not a simple procedure. The case of hexachlorobenzene (HCB) provides an example of how problematic this subject is. In 1975, WHO recommended an acceptable daily intake (ADI) of 0.6 mg/kg body weight. This was based on a safety factor of 2000. In 1978, this ADI was withdrawn because of the demonstration of HCB carcinogenicity. Since that time no new ADI has been proposed. In the absence of recommended ADI values for potential human carcinogens, the most suitable procedure for the calculation of carcinogenic risks associated with chemical exposures is to use no-threshold dose-response slopes derived from multi-stage models.

2.3. HARM TO POPULATIONS

The utility of risk assessment would be limited if it could only be applied to the risks to individuals with known exposures. It is equally useful to estimate the magnitude of expected harm within populations from estimated, extrapolated or predicted exposures. A probability of 10^{-3} of fatality to an individual due to a specific cause means one chance in one thousand of dying from this cause. For a population with the same average exposure, it means that, statistically, one should expect one fatality in each thousand individuals due to the same cause. The product of the average individual lifetime probability of dying from a specific cause and population size divided by mean life expectancy gives the average number of deaths from that cause per year.

Where populations are reasonably uniformly exposed to a hazard — such as the risk of being struck by lightning — then the number of fatalities expected per year is simply the product of the annual individual risk and the size of the population. Indeed, for such risks, the individual risk is based on the number of occurrences in the population as a whole. Exposure to chemical carcinogens and radiation will however vary within a given population or occupational group. This means that there is less uniformity in the distribution of risks to individuals posed by chemicals and radiation. Nevertheless, an appreciation of the potential harm to large populations provides a useful perspective on the levels of risk to 'average' individuals within those populations.

2.4. ACCEPTABILITY OF RISKS

Risks are differently perceived for a variety of reasons. There is a general tendency for the public to overestimate what are actually low risks of death, such as those associated with major catastrophes, and to underestimate the effects of common diseases such as diabetes. Factors which affect the perception of risk include: personal experience of a specific hazard, publicity associated with hazards, and the degree to which an individual is able to control the level of exposure to a hazard. In some cases, risks can be avoided completely, and this becomes a matter of personal choice. There are other risks, however, that are ubiquitous in modern society. It is virtually impossible to define a universally acceptable level of risk for any hazard. However, a probability of serious harm of 10^{-5} is often selected as an acceptable level of lifetime risk (essentially a default risk value) for individuals in fairly large populations [29]. National jurisdictions may adopt a range of values for acceptable risk depending on the size of the exposed population. The United States Environmental Protection Agency (EPA) has, for example, suggested a '*de minimis*[1] lifetime risk level' of 10^{-5} to 10^{-4} for the protection of individuals in small populations and 10^{-7} to 10^{-6} for the protection of individuals in large populations [33]. By and large, what is regarded as being 'safe enough' for members of the public is directly dependent upon the confidence they have in the management of the hazard associated with the risk and whether or not it is under their direct control.

2.5. METHODS AND UNCERTAINTIES IN RISK ASSESSMENT

The best estimates of risk are based on actual, well-documented, epidemiological information. The risks associated with a specific hazard can only be calculated by determining the increment in risk relative to that arising from an otherwise identical situation in which that hazard was not present at all. Accordingly, for such information to be obtained from epidemiological studies, it is necessary to have data on two populations. At low levels of exposure, where the differences in effect between the two populations are expected to be small, large numbers of people must be included to detect differences which are statistically significant. Thus, epidemiological studies often reveal excess disease that is manifest in a few per cent of the exposed population. However, it is extremely rare for excess disease affecting less than 0.1% of the population to be reliably detected. The statistical proof of carcinogenicity at low levels of exposure is further hindered by the high background rate of cancer deaths. For example, the probability of death from cancer in the USA (Table I) is about 3×10^{-3} per year. Prediction of risk at lower levels can be based on experimentally derived data involving laboratory animal exposures. These experiments involve subjecting animals to sufficiently large exposures to produce an effect at a frequency which attains statistical significance. The extrapolation of such data to humans requires two conceptual steps: an assumed similarity between the toxicity of the chemical to different species, and an extrapolation from high levels of exposure to low ones.

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[1] *De minimis* risk signifies a risk level that is below regulatory concern and stems from the Roman legal expression 'de minimis non curat praetor' (the prosecutor is not concerned with trivialities).

Extrapolation from high dose to low dose forms an essential part of the assessment of carcinogenic risk. The reason for this is that, in both epidemiological studies and animal experiments, statistically significant increases in incidence rates, or in the prevalence of injury, are observed only at relatively high dose levels. Null, or insignificant, response rates do not exclude the possibility of carcinogenicity and uncertainty is inversely related to group size. Thus, when none of, say, ten animals given a certain dose develops cancer, the upper confidence limit of the probability of cancer induction with 99% assurance is 37%. When the number of animals is increased to 100, and there is still no response, the 99% upper confidence limit becomes 4.5%.

2.5.1. Risks associated with radiation exposures

The effects of radiation at high dose and dose rates are reasonably well documented. However, there is still considerable uncertainty about the effects of exposure to radiation at low dose and dose rates. This is because the expression of effects is masked by the normal occurrence of disorders which may or may not be due to radiation exposure. To make a statistically valid study of the effects of radiation on man at low dose levels requires the observation of a population of millions over several generations. Such an analysis is further complicated by the fact that it is not possible to isolate an otherwise identical 'control' population that is not exposed to radiation.

It is generally agreed by the radiological protection community that the only observable effect of the exposure of a large number of people to low level radiation may be the induction of a few cancers, in addition to the thousands which occur naturally, years or even decades after the exposure has been incurred. It is often forgotten that most forms of cancer are diseases of old age. In countries where life expectancy is in the range of 60–70 years, 20% or more of all deaths are due to cancer but the average victim is about 65 years old. In addition to ionizing radiation, individuals are commonly exposed in their everyday lives to many factors that can cause cancer. The list of such carcinogens includes nitrosamines, chimney soot, arsenic, paraffin oil, coal tar, tobacco smoke, ultraviolet light, asbestos, some chemical dye intermediates, fungal toxins, viruses and even heat. Only in exceptional circumstances is it possible to identify conclusively the cause of a particular cancer. There is also experimental evidence from work with animals and plants that exposure to radiation can have genetic effects, though none directly attributable to the effects of radiation exposure have so far been detected with certainty in humans.

It is presumed that exposures to radiation, even at the levels associated with natural background, can affect human health. It is further assumed as a practical hypothesis that the probability of an effect is strictly proportional to exposure. No evidence to contradict this assumption has been found. Considerable debate has, however, taken place regarding the nature of the dose–response (risk–dose) relationship at extremely low doses and dose rates. Because of the ubiquitous nature of natural radiation, however, this debate must be based on a recognition that zero dose does not exist and that the important element of the debate relates to whether an increment in dose results in a proportional increment in risk. Whether or not the risk is directly proportional to incremental exposure in the region near zero on the dose–response curve is irrelevant. Thus, in view of the finite level of natural radiation and knowledge about the effects of higher doses and dose rates of radiation, an assumption of linearity between risk and dose appears to be wholly reasonable [18]. It is also entirely consistent with evaluations of the frequency of natural radiation damage particle tracks intercepting individual human cells if it is assumed that damage and repair of cells are not influenced by simultaneous damage to remotely situated cells.

The uncertainties of applying the linear, no threshold, dose–response relationship for low level radiation exposure relate primarily to the selection of the coefficient for converting dose to risk. Evidence i.e. from medical radiation exposures suggests that the risks from photon radiation at low doses and dose rates are lower than the values assessed at high dose rates. Accordingly, the coefficient for dose to risk conversion is probably an overestimate by a factor of between 2 and 10. Also, the difference between the application of an absolute risk model or relative risk model for estimating the conversion coefficient suggests that there exists about a factor of 2 uncertainty in the chosen value. In the simple 'absolute' model, the excess cancer death probability rate is dose dependent but independent of age. In the simplest version of the 'relative' model, the excess rate increases with age at the same rate as the background cancer rate.

2.5.2. Risks associated with the exposure to chemical carcinogens

Mathematical models are used for extrapolation from high to low doses. These models have inherent weaknesses, especially when dose extrapolation is combined with interspecies extrapolation. Extrapolation on the basis of exposure where the active contaminant is inhaled, ingested, or even parenterally administered assumes that this exposure correlates linearly with the dose to the target tissue. This is rarely the case, because enzymes acting on the administered agent may become saturated resulting in either the inactivation of a carcinogen or the inhibition of carcinogen formation from a pre-carcinogen. Further assumptions concern the presence or absence of threshold and the shape of the dose-response relationship below the observational range.

The assumption of low dose linearity in a no-threshold linearized multi-stage model enables the risk associated with low doses for a large number of compounds to be evaluated. The slopes of the dose–response relationship derived from such models can be converted to risk at any low dose level and used for the estimation of risks. The slopes, and therefore the risks calculated for an actual or projected exposure, are upper bounds corresponding to the 95% upper confidence limit. This means that, at the 95% confidence level, the range of lifetime risk is between zero and the upper bound. Accordingly, estimates of incremental risks based upon this approach cannot be legitimately summed. However, despite these inherent limitations, upper bound risk estimates do at least provide some quantitative basis for determining public health protection policy.

3. ESTIMATION AND CALCULATION OF RISKS OF DEVELOPING CANCER

If exposure to hazardous substances and radiation can be estimated, the calculation of the associated risk requires a knowledge of the slopes of the relevant dose-response relationships. These slopes are referred to here as risk-coefficients.

3.1. RISK COEFFICIENTS IN RADIOLOGICAL PROTECTION

It has long been assumed that, following exposure to radiation, there is an increased risk of developing cancer, irrespective of whether or not the dose was received from sources external or internal to the body. Indeed, for the latter, the dose itself (the committed effective dose) is received over an extended period of time. An increase in the probability of dying from cancer can therefore be assessed from a single exposure to radiation or, for example, from a single annual intake of food contaminated with a known quantity of radionuclides. For general application, at a population level, for low doses of radiation, this risk coefficient for a 'lifetime cancer death probability' is now taken to be 5×10^{-2} per Sv [28]. It should be stressed that the increased risk may not result in effects during the increased exposure period. Most cancers do not develop for some considerable time, ranging from a few years in the case of leukaemia to tens of years for other cancers. Thus, the result of the increased risk, in terms of an increase in the 'age specific death probability rate', would also not occur for many years. During that time, however, the risk of death from other causes — including the risk of dying from cancer of different aetiology — will also have increased.

3.2. RISK COEFFICIENTS FOR CHEMICAL CARCINOGENESIS

In contrast to radiation, the majority of the data relating to an increased probability of dying from cancer as a result of exposure to chemicals has been derived from experiments in which exposures have been continuous. Comparisons have then been drawn between populations chronically exposed to different concentrations of the assumed carcinogen. Thus, there are differences between radiological and chemical exposures, particularly with regard to a commitment of risk. By and large, risks are only calculated on the basis of continuous lifetime exposures. Such lifetime risks are affected differently if the level of exposure, or the length of exposure time, is varied.

3.3. METHODS OF CALCULATION

In view of the above, drawing simple and direct comparisons between the probability of death by cancer as a result of the ingestion of radioactive and non-radioactive contaminants in food is not straightforward. For radionuclides it is possible to calculate a rate of intake (Bq/a) which can be converted to a dose rate using the conversion factor (Sv/Bq) appropriate to each radionuclide. Then, using the risk-dose conversion factor of 5×10^{-2} per Sv, the resulting probability of death by cancer for annual ingestion of radionuclides in foods can be estimated.

For carcinogenic chemicals, on the other hand, estimates of increased probability of death by cancer are based on prolonged, most frequently lifetime, exposures. Chronic exposure over long periods can be converted to a cumulative exposure and linear extrapolation methods used to express the associated risks on the basis of unit daily exposure. Thus, the unit risk given by the multi-stage model corresponds to the lifetime incremental risk for a continuous daily intake of 1 mg per kg body weight per day derived from the upper bound slope of the linear part of the dose-response curve. The unit risk is a reference value for low dose extrapolation and can exceed unity (i.e. can exceed 100% risk for 1 mg/kg/day intake).

It is commonly assumed that initiators, which are DNA reactive, have no threshold. It is further assumed that promoters, which are not DNA reactive and only transform initiated [i.e. altered/induced] cells to tumour cells, have a threshold for action. In this document however, it is assumed that upper bound slopes can be applied to the estimation of risks associated with low exposures to both initiators and promoters, that the incremental risk of mortality (attributable to individual carcinogens) remains constant after the termination of exposure, and that the dose–response relationship is linear in time. These as sumptions are similar to those previously adopted elsewhere [34, 35] and permit conservative estimates of the risks of cancer induction resulting from exposures to chemical carcinogens to be calculated on a basis which is reasonably comparable to that adopted for the assessment of risks from radiation exposure.

Due to the assumption of a linear relationship between dose and the probability of cancer induction, the above approaches can also be applied to entire populations. In radiological protection, an assessment of harm can be made by calculating a collective (effective) dose, which is usually expressed in terms of man-sieverts. If, for example, the collective dose in a given year was 200 man·Sv then, using a risk-dose conversion value of 5×10^{-2} per Sv, 10 excess cancer deaths would be expected. This would apply equally to a population of 10 000 people each receiving 0.02 Sv, and to a population of 200 000 people each receiving 0.001 Sv, even though the risk to **individuals** in the two populations would differ by a factor of 20. This assumption holds true irrespective of whether the population is exposed continuously or not. Thus, if the intake of a radionuclide by a population is assumed to be relatively constant over a prolonged period, an estimate of harm (i.e. expected excess cancer deaths) can be made and compared with the excess cancer deaths expected as a result of continuous exposure to a carcinogenic chemical.

For this study, comparisons of risk associated with the consumption of seafood are particularly appropriate. The disposal of radioactive waste in the deep sea results in the slow release and dispersion of radionuclides over large areas and during long timescales. As a result, there is a possibility of small quantities of radionuclides entering the human food web — primarily seafood derived from surface waters. Seafood also contains naturally occurring radionuclides and is contaminated with several chemicals which are believed to have carcinogenic properties and are known to be disseminated by anthropogenic activities. Accordingly, estimates of the carcinogenic risks posed by exposures to all three categories of substance in seafood provides a suitable basis for equitable comparison.

It should be emphasized that the actual exposures and associated risks relating to sources and practices involve consideration of exposures received by individuals through all pathways. Previous assessments of the impact on human health of practices involving radiation exposure take account of all routes of exposure to individuals. In the case of sea dumping these pathways include inhalation of suspended sediment and marine aerosols, consumption of sea salt and desalinated seawater, and external exposures, in addition to those involving seafood consumption. Similarly, assessments of the actual risks posed by chemical exposures would have to consider all potential routes of human exposure. However, for the specific requirements of this task, it was necessary to select a basis for equitable comparison of the risks posed by radiation and chemical exposures. Because of the limitations in information on the incidence of chemicals in the environment, the specifics of possible exposure pathways, and associated effects for chemicals, the basis chosen for comparative purposes is that of seafood consumption. For the case of radionuclide exposures associated with sea dumping of radioactive waste, with one exception, seafood consumption pathways are the dominant pathways of exposure. This may not be the case with exposures and risks associated with chemicals in the environment. The one exception for radiation exposures from sea dumping of radioactive waste involves the dominant (95%) contribution of ^{14}C to the **collective** dose. The collective doses associated with sea dumping of low level radioactive waste to large populations estimated in this document do not include the contribution from ^{14}C for which the exposure pathway is primarily through the consumption of terrestrial foodstuffs. It has shown to be extremely difficult to reduce the collective dose commitment from ^{14}C because of its long half-life (5730 years). In all other respects, the doses and risks estimated for sea dumping of radioactive waste correspond to the integrated exposures from all pathways although seafood consumption is the most important of these.

4. CALCULATIONS OF COMMUNAL HARM ASSOCIATED WITH RADIONUCLIDE AND CHEMICAL EXPOSURES THROUGH MARINE PATHWAYS

In this section, estimates of the communal harm, or the expected cancer fatalities in large populations, resulting from exposures to both radionuclides and chemicals in seafood are calculated. This approach to risk estimation is dealt with first because it provides a convenient basis for comparison, it requires fewer assumptions than comparable estimates of risk to individuals, and existing collective dose estimates for large populations can be used to calculate harm associated with radiation exposures from several practices.

4.1. HARM ASSOCIATED WITH RADIONUCLIDES IN SEAFOOD

As discussed in Section 3, for the purposes of radiological protection, it is useful to calculate the dose received by a population in units of man-sieverts. Of particular interest is the fraction of dose received from specific pathways, such as that from different foodstuffs. The annual collective dose from a particular radionuclide, widely dispersed in the marine environment and actually present in seafood, can therefore be estimated for a given population as follows:

$$S = AFD \qquad (1)$$

where:

S is the collective dose rate (man-sievert/a),
A is the average concentration of the radionuclide (Bq/kg) in a particular type of seafood from a given sea area,
F is the annual aggregate rate of seafood consumption by the population (kg/a),
D is the committed effective dose per unit intake of radionuclide (Sv/Bq).

A summation over all sea areas for each seafood therefore gives an annual collective dose (in man-sieverts) for that radionuclide when ingested. When repeated for all such radionuclides, a total collective dose value is obtained.

Equation (1) can then be used to estimate the expected number of fatal cancers (G) in a given year in a population, by application of the risk coefficient (R), which is taken to be 5×10^{-2} per Sv. Thus,

$$G = AFDR \qquad (2)$$

An important feature of such a calculation is the fact that data on individual consumption (and, thus, individual exposure) rates are not required. Calculation can also be made, using models appropriate to the distribution of the radionuclide in space and time, of the collective dose commitment over different periods of time. Such calculations are used when comparing options for future waste disposal. (Note however, that for such applications, the dose rates associated with other exposure pathways, including external exposures, have to be considered as well).

Equation (1) has been used previously to calculate the collective dose rates arising from the disposal of low level radioactive wastes in the deep sea via seafood consumption [36]. These results are given in Table III for both global and regional population. Estimates of excess fatal cancers estimated on the basis of equation (2) are also provided in that table. On their own, such values are not very informative and it is therefore useful to draw comparisons with collective dose rates associated with radionuclides derived from other

TABLE III. ESTIMATES OF COLLECTIVE DOSE AND FATAL CANCERS PER YEAR TO DIFFERENT POPULATIONS AS A RESULT OF RADIATION EXPOSURE VIA SEAFOOD PATHWAYS

SOURCE	**COLLECTIVE DOSE RATE (man·Sv/a)**	**FATAL CANCER EXPECTED PER YEAR**
Naturally occurring radionuclides		
– Global	7.4×10^4	3.7×10^3
– EC	3.4×10^3	1.7×10^2
– UK	5.0×10^2	2.5×10^1
SOURCE	**COLLECTIVE DOSE RATE (man·Sv/a)**	**EXCESS FATAL CANCER EXPECTED PER YEAR**
Past sea dumping of radioactive waste (peak, ≈ 2950)		
– Global	1.5×10^{-1}	7.5×10^{-3}
– EC	1.0×10^{-1}	5.0×10^{-3}
Civil nuclear power discharges		
– Global	7.6×10^2	3.8×10^1
– EC (peak, 1979)	3.3×10^2	1.7×10^1
– UK (peak, 1979)	1.3×10^2	6.5
Weapons test fallout		
– EC (peak, 1979)	1.0×10^2	5.0
Chernobyl accident		
– EC (peak, 1986)	1.4×10^2	7.0

sources that arise in aquatic foodstuffs. The collective dose rate resulting from the presence of naturally occurring radionuclides has recently been estimated with similar methods [37]. The principal contribution is from ^{210}Po.

A number of calculations have also been made with respect to other sources for the European Community (EC) in the MARINA programme [38]. A selection of these — civil nuclear power discharges, weapons fallout, and the Chernobyl accident — are also shown in Table III. As can be seen, the collective doses for the world population resulting from the presence of naturally occurring radionuclides are 5×10^5 times greater than those for sea dumping. For the EC population, the estimated rate of excess cancer (5×10^{-3}/a) associated with potential exposures from sea dumping of low level radioactive waste results in one expected fatality every 200 years. This is clearly a relatively small number in a population of some 320 million with an average life expectancy of about 70 years. Furthermore, the most recent UNSCEAR report [39] states: "the product of risk coefficients appropriate for individual risk and the relevant collective dose will give the expected number of cancer deaths in the exposed population, provided that the collective dose is at least of the order of 100 man·Sv. If the collective dose is only a few man-sieverts, the most likely outcome is zero deaths".

4.2. HARM ASSOCIATED WITH CHEMICAL CONTAMINATION OF SEAFOOD

An analogous approach to that used previously for radiation exposure can be developed for the risks associated with carcinogenic chemical intakes based on an assumption that risk is proportional to exposure without threshold. The upper bound slopes, expressed as 'potencies' in (mg per day per kg body weight)$^{-1}$ [34, 35, 40, 41] can be converted to the corresponding lifetime risk per unit annual intake of the chemical

TABLE IV. CONVERSION OF POTENCY VALUES FOR CERTAIN SUSPECTED HUMAN CARCINOGENS TO INCREMENTAL LIFETIME RISKS PER UNIT INTAKE RATE (mg/a)$^{-1}$

Chemical	**Potency**	**Incremental lifetime risk R_c**
ΣPCB	4.43	1.7×10^{-4}
ΣDDT	0.34	1.3×10^{-5}
Dieldrin	30.4	1.2×10^{-3}
Chlordane	1.61	6.3×10^{-5}
HCB	1.67	6.5×10^{-5}
Benzo-a-pyrene	11.5	4.5×10^{-4}

carcinogens, (R_c) to an individual of average 70 kg body weight (Table IV). These, in turn can be used to calculate the number of fatal cancer cases during the life expectancy of individuals (Z_c) and the number of fatal cancers in an exposed population per year (G_c) by the following formulations.

If:

Z_c is the number of fatal cancers during life expectancy of individuals within the exposed population caused by exposure to a chemical carcinogen in seafood,
C_c is the average concentration of a chemical carcinogen in seafood (mg/kg),
F is the annual aggregate rate of seafood consumption by the population (kg/a),
R_c is the lifetime risk per unit annual intake of the chemical carcinogen (mg/a)$^{-1}$, and
L is the life expectancy (years) within the population,

then:

$$Z_c = C_c F R_c \qquad (3)$$

and

$$G_c = C_c F R_c/L \qquad (4)$$

Thus the term [R_c/L] is analogous to the [risk/Sv] term in the equation for risks from radionuclide ingestion. The risk/Sv term in equation (2) has units of Sv^{-1} (i.e. (dose)$^{-1}$) and the R_c/L term in equation (5) has units of mg^{-1} (that is mass^{-1}, equivalent to dose^{-1}). Both relate risk to unit intake.

The seafood consumption rates and chemical concentrations in seafood shown in Table V were assumed for global and European Community populations.

Using the values in Tables IV and V it is possible to calculate the number of cancer fatalities expected annually in the global and EC populations associated with ingestion of chemicals through seafood consumption using equation (4). The results are shown in Table VI.

TABLE V. SEAFOOD CONSUMPTION AND CHEMICAL CONCENTRATIONS IN SEAFOOD ASSUMED FOR THE GLOBAL AND EUROPEAN COMMUNITY POPULATIONS

Community	Aggregate seafood consumption rate (kg/a)	Concentrations in seafood (mg/kg)				
		ΣPCB	ΣDDT	Dieldrin	Chlordane	HCB
Global	3.4×10^{10} [a]	0.002	0.001	–	–	–
EC[b]	2.2×10^{9} [c]	0.025	0.005	0.003	0.002	0.002

[a] Based on the total global fish catch in 1988 of 8.5×10^{10} kg [42] assuming consumption of 40% by weight.
[b] Concentrations for contaminants in fish for the EC [43].
[c] Based on a total EC fish catch in 1984 of 5.6×10^{9} kg [44] assuming consumption of 40% by weight.

TABLE VI. ESTIMATES OF FATAL CANCER INDUCTION ASSOCIATED WITH ORGANIC CHEMICAL CONTAMINANTS IN SEAFOOD

Population	Chemical	Total ingestion in seafood (mg/a)	Number of fatal cancers during lifetime (Z_c)	Number of fatal cancers per year in population[a] (G_c)
Global[b]	ΣPCB	6.8×10^{7}	1.2×10^{4}	1.7×10^{2}
	ΣDDT	3.4×10^{7}	4.4×10^{2}	6.3
EC[c]	ΣPCB	5.5×10^{7}	9.3×10^{3}	1.3×10^{2}
	ΣDDT	1.1×10^{7}	1.4×10^{2}	2.0
	Dieldrin	6.6×10^{6}	7.9×10^{3}	1.1×10^{2}
	Chlordane	4.4×10^{6}	2.8×10^{2}	4.0
	HCB	4.4×10^{6}	2.9×10^{2}	4.1

[a] Annual cancer fatalities in population estimated on the basis of an assumed mean life expectancy of 70 years.
[b] Population 5×10^{9}.
[c] Population 3.2×10^{8}.

4.3. DISCUSSION

The larger number of fatal cancers for ΣPCB exposure for the EC population compared to the global population (Table VI) predominantly reflects uncertainties in the estimates of the average concentrations of ΣPCB in seafood. It must be emphasized that, although such exposure estimates involve uncertainties, these are probably smaller than the uncertainties associated with the extrapolation method. The data presented in Tables III and VI are therefore primarily of comparative value only.

At a global level, naturally occurring radionuclides contribute a far greater collective dose than any marine source of artificial radionuclides, including sea disposal of packaged low level radioactive waste which has contributed the least of the exposures associated with seafood consumption considered here. Converting such data to expected numbers of excess cancer deaths per year is somewhat more equivocal. The estimated 'excess' cancers arising from the dose received from naturally occurring radionuclides can be considered to be part of the background cancer rate from 'natural' causes. Nevertheless, in principle, they indicate the number of cancers that could in principle be avoided if seafood was not consumed. The excess cancer fatality associated with sea disposal of radioactive waste is much smaller than the range of cancer fatalities associated with variations in background radiation.

The number of expected cancer deaths resulting from the ingestion of organic contaminants in seafood, in particular ΣPCB and dieldrin, are similar to those associated with naturally occurring radionuclides in seafood and considerably greater than those associated with ingestion of radionuclides derived from sea disposal of radioactive waste.

It should be noted that the estimates of expected cancer deaths associated with chemicals in seafood are based on actual contemporary measurements of their concentrations in marine fish. Although the use and disposal of these chemicals has been increasingly restricted, they are currently present in marine fish at measurable levels. In contrast, the estimates of harm associated with exposures to radionuclides dumped at sea are based on predictions of peak future concentrations in seafood based on conservative assumptions.

It is useful to consider the extent to which such estimates can be extrapolated, or integrated, over time. In radiological protection, use is made of the term 'collective dose commitment' to integrate dose over different periods of time, or to infinity. The collective dose commitment arising from sea dumping has already been discussed in the previous scientific review conducted for the London Convention 1972 [14].

Three sea dumping scenarios have been evaluated by the NEA [36]. For past sea dumping of low level radioactive waste (Scenario A) the overall collective dose commitment amounts to some 3.8×10^4 man·Sv, of which some 3.6×10^4 man·Sv arises from ^{14}C. It is again stressed that the primary exposure pathway for ^{14}C is incorporation into the global CO_2 cycle, uptake into terrestrial foodstuffs and their subsequent ingestion. For continued sea dumping of radioactive waste for a further 5 years, at either the same rates as in the recent past (Scenario B), or at ten times that rate (Scenario C), the total collective dose commitment increases to about 5.9×10^4 and 2.8×10^5 man·Sv respectively. Thus, using a risk factor of 5×10^{-2} per Sv, past dumping may be considered to result in a total of 1900 cases of severe harm, distributed throughout the world's population (assumed to be 10^{10} persons), over a period of 10^4 years (i.e. 400 generations or 140 life expectancies). Approximately 95% of these fatalities are associated with ingestion of ^{14}C in terrestrial foodstuffs. Again, it should be stressed that reduction of the collective dose commitment from ^{14}C in wastes is very difficult irrespective of which waste management option is selected. This figure can be compared with the estimate of 3700 fatalities **per year** (or 37 million fatalities over 10^4 years) associated with the ingestion of naturally occurring radionuclides in seafood in the global population.

In conclusion, of the exposures considered here, the greatest harm to the global population arises from exposure to naturally occurring radionuclides in seafood. Harm associated with ΣPCB exposures through seafood consumption is a little over an order of magnitude lower. That caused by exposures to radionuclides arising from sea dumping of radioactive wastes is over four orders of magnitude lower. Using estimates of harm in the European Community population, for which more reliable chemical concentration data are available, reinforces the view that the harm associated with exposure to organic chemicals ranges from values close to that associated with naturally occurring radionuclides to about two orders of magnitude lower. The harm to the European Community population associated with sea dumping of radioactive waste is at least a further three orders of magnitude lower than those associated with exposures to chemicals in seafood.

5. RISKS TO INDIVIDUALS

Although the estimated number of fatal cancers in a population arising from the consumption of contaminated seafood is low, individuals will still remain concerned about the level of risk posed to them. Differences in individual consumption rates, and geographical variations in contaminant concentrations, will result in individual risks varying considerably.

To take account of this variability, two different approaches have been adopted. One deals with the risk to an 'average' member of a population and is expressed as per caput risk. The second deals with the risks to individuals in a specific group (a critical group) which, by virtue of the large quantities of food eaten and the higher concentration of contaminants assumed in such foodstuffs, will be exposed to higher doses than any other group of individuals within the population. Such a critical group can either be categorically identified within a specific population for exposures concurrent with the practice, or, for practices where the greatest dose to individuals is expected to occur sometime in the future, postulated on the basis of a number of reasonably conservative assumptions. A critical group of members of the public defined on the basis of radiation exposure from one radionuclide and one environmental pathway may be exposed through other pathways and other nuclides released from the same (or other) sources. In order to ensure that the appropriate dose limit is not exceeded, account must be taken of exposure from all pathways and nuclides that contribute to the total dose of an average member of the critical group.

In view of the long time periods of concern in a radiological assessment of sea disposal, it is appropriate to consider not only those exposure pathways that currently exist (actual pathways) but also pathways which might conceivably become important in the future (hypothetical pathways) and which have been agreed for consideration in international discussions [8]. Of the three primary modes of exposure, internal irradiation through ingestion and inhalation and external irradiation, this section deals only with ingestion pathways for marine foodstuffs. The same pathways have been considered here for calculating the risk associated with ingestion of seafood contaminated with carcinogenic chemicals. Due to possible changes in population distribution, societal conditions, fishing practices, dietary and other habits that cannot be characterized in detail far into the future, it is convenient to define the critical group of seafood consumers in terms of hypothetical individuals who consume large amounts of surface and mid-depth fish, crustacea, molluscs, seaweed (actual pathways) and plankton. The consumption of plankton constitutes a hypothetical pathway.

5.1. ESTIMATES OF PER CAPUT RISKS TO INDIVIDUALS IN LARGE POPULATIONS

The calculation of per caput risks is easily performed by dividing the previous estimates of harm to large communities by their respective populations. These representations of average individual risks (probabilities of harm to individuals) are shown in Table VII. It must again be stressed that these are average values and take no account of the heterogeneity of habits, including the rates of fish consumption, within the population. Such data are only useful for comparative purposes in that they indicate the approximate order of magnitude of risk to which individuals in a population may be exposed. As an average, each value will undoubtedly be an underestimate for the actual consumers of seafood because not all members of a population will eat seafood on a regular basis. However, the comparative risks among those posed by different substances in seafood will be unaffected by such heterogeneity.

Table VII also provides the range of risks of fatal cancer associated with radiation and chemical exposures via seafood pathways. The individual risks of fatal cancer induction

TABLE VII. 'PER CAPUT' RISKS OF DYING OF CANCER EACH YEAR FROM RADIATION AND CHEMICAL CARCINOGEN EXPOSURES ASSOCIATED WITH SEAFOOD CONSUMPTION

Population	Source	Per caput risk per year
Global[a]	Naturally occurring radionuclides	7.4×10^{-7}
	Civil nuclear site discharges	7.6×10^{-9}
	ΣPCB in seafood	3.4×10^{-8}
	ΣDDT in seafood	1.3×10^{-9}
	Past sea dumping of radioactive waste (peak, ≈ 2950)	1.5×10^{-12}
EC[b]	Naturally occurring radionuclides	5.3×10^{-7}
	Civil nuclear site discharges (peak, 1979)	5.3×10^{-8}
	Chernobyl fallout (peak, 1986)	2.1×10^{-8}
	Weapons test fallout (peak, 1979)	1.6×10^{-8}
	ΣPCB in seafood	4.1×10^{-7}
	ΣDDT in seafood	6.2×10^{-9}
	Dieldrin in seafood	3.4×10^{-7}
	Chlordane in seafood	1.2×10^{-8}
	HCB in seafood	1.3×10^{-8}
	Past sea dumping of radioactive waste (peak, ≈ 2950)	1.4×10^{-11}
UK[c]	Naturally occurring radionuclides	4.5×10^{-7}
	Civil nuclear site discharges (peak, 1979)	1.2×10^{-7}

[a] Population 5×10^{9}.
[b] Population 3.2×10^{8}.
[c] Population 5.5×10^{7}.

from naturally occurring radionuclides constitute the dominant risk from seafood consumption in all populations and, as expected, are similar at about 5×10^{-7}/a. The more intense use of civil nuclear power in the European Community, compared with the global average, results in the individual risks in the EC from this practice being about an order of magnitude greater than the global average. However, the individual per caput risks associated with ΣPCB ingestion in seafood are larger than those associated with civil nuclear discharges for both global and EC populations.

The conclusion that can be immediately drawn from this comparison is simply that the risks associated with chemical ingestion via seafood are of similar magnitude to those posed by civil nuclear discharges and nuclear weapons fallout. Furthermore, they are about three orders of magnitude greater than the peak risks to average members of both the global and EC populations likely to arise in the future from sea dumping of radioactive wastes.

5.2. RISKS TO INDIVIDUALS IN MOST EXPOSED GROUPS

The doses received by individual members of the critical group through seafood consumption are estimated using consumption rates defined on the basis of actual contemporary types and extreme rates of seafood consumption (Table VIII). The selection of the consumption rate for the only hypothetical pathway (plankton consumption) was made on a less conservative basis to reflect the uncertainty about whether this form of seafood consumption would constitute a realistic pathway in the future. The consumption rates are appropriate to adult members of the public and lead to dose overestimates for children because their consumption rates are lower although the dose per unit intake is higher. No account was taken of variations in diet, habit, or doses per unit intake with age.

TABLE VIII. SEAFOOD CONSUMPTION RATES ASSUMED FOR THE MOST EXPOSED INDIVIDUAL

Seafood type	**Consumption rate (kg/a)**
Surface and intermediate depth fish	220.0
Crustaceans	36.5
Molluscs	36.5
Plankton	1.1
Seaweed	36.5

It should be stressed that the seafood consumption rates attributed to the members of the critical group are relatively high. This emphasizes the pessimistic or conservative nature of these calculations. The total individual consumption rate corresponds to 5 seafood meals per day. The global per caput consumption rate of seafood is approximately 6.7 kg/a based on a world fish catch of 8.5×10^{10} kg/a [42] divided by the global population assuming that only 40% of the catch by weight is consumed. According to WHO [45] only 5% of seafood consumers eat more than 9 times the population average (i.e. more than 60 kg/a). The assumed average consumption rate by members of the critical group exceeds this latter figure by about a factor of five and corresponds to anecdotal information about extraordinary rates of seafood consumption by some people. Nevertheless, the critical group consumption rates were chosen to reflect the highest potential exposures to a small group of individuals for the assessment of the impact of sea dumping of low level radioactive waste and, in this context, appear appropriate.

This same critical group can also be used to compare extreme exposures and extreme risks associated with radionuclide and chemical carcinogen ingestion through seafood consumption pathways. In order to provide some reasonable basis for comparison among these different sources of exposure and associated risks, a number of important assumptions have to be made. These assumptions are as follows:

(a) Exposures to radionuclides and chemical carcinogens remain essentially constant throughout the lifetime of members of the critical group;

(b) The age distribution of the critical group remains constant throughout the period of continuous exposure.

5.2.1. Risks associated with the presence of naturally occurring radionuclides ingested through seafood consumption

An evaluation of individual doses resulting from exposures to natural radionuclides through seafood consumption has already been made [37]. This paper tabulates data on concentrations in various types of seafood, and the dose per unit intake, for naturally occurring radionuclides. These data can be used to define the dose associated with the intake of such radionuclides in seafood ingested by the hypothetical critical group, and the corresponding risks. These figures are given in Table IX.

TABLE IX. INDIVIDUAL DOSE RATES FROM SIGNIFICANT NATURAL RADIONUCLIDES IN SEAFOOD FOR MEMBERS OF THE CRITICAL GROUP [37]

Nuclide	**Intake (Bq/a)**					**Dose per unit intake (Sv/Bq)**	**Dose rate (Sv/a)**
	Fish	Crustaceans	Molluscs	Algae	Total		
C-14	3300	550	550	550	4900	5.6×10^{-10}	2.8×10^{-6}
K-40[a]	22000	3600	3600	18000	48000	5.1×10^{-9}	2.4×10^{-4}
Rb-87	220	37	36	36	330	1.3×10^{-9}	4.3×10^{-7}
Po-210	330	910	1800	150	3200	4.3×10^{-7}	1.4×10^{-3}
Pb-210	8.8	7.3	110	7.3	130	1.4×10^{-6}	1.9×10^{-4}
Ra-226	22	0.7	11	0.7	34	3.0×10^{-7}	1.0×10^{-5}
U-234	2.6	4.4	11	91	110	7.0×10^{-8}	7.6×10^{-6}
U-238	2.4	4.0	9.9	91	110	6.3×10^{-8}	6.8×10^{-6}
							Total: 1.8×10^{-3}

[a] It should be noted that K-40 is in the homeostatic control in the body.

The critical group member receives a dose of 1.8 mSv/a, corresponding to an individual risk of 9×10^{-5}/a, from exposures to naturally occurring radionuclides through seafood consumption. As can be seen, the largest contributor to this dose is ^{210}Po. Due to its relatively short half life (138 days) the calculation assumes that seafood is consumed freshly caught. Delays associated with processing or bringing the seafood to market will result in a lowering of the associated individual dose.

5.2.2. Risks associated with sea dumping of radioactive waste

The dose to the critical group resulting from sea dumping of radioactive wastes in the North-East Atlantic has been estimated by the Nuclear Energy Agency [36]. These doses are shown in Table X. The reader should be reminded that the exposures and risks given in Table X not only include consideration of seafood consumption pathways but also other ingestion pathways and external exposure pathways. These other pathways, however, provide comparatively insignificant individual doses compared with those associated with seafood consumption.

5.2.3. Risks associated with exposures to organic chemical contaminants in seafood

The concentrations of selected organic chemical contaminants in seafood required for calculating critical group intakes and risks are shown in Table XI. These values are derived either from those for contaminated estuarine fish caught in US coastal fisheries [34] or from other published data [31].

TABLE X. PEAK DOSES TO THE CRITICAL GROUP FROM SEA DUMPING OF LOW LEVEL RADIOACTIVE WASTE

Scenario	Peak individual dose (Sv/a)	Year of peak dose	Risk per year of intake
Past sea dumping (Scenario A)	2.2×10^{-8}	2150	1.1×10^{-9}
Past sea dumping plus 5 years dumping at rates typical to recent years (Scenario B)	2.6×10^{-8}	2150	1.3×10^{-9}
Past sea dumping plus 5 years dumping at rates ten times those of recent years (Scenario C)	1.3×10^{-7}	2150	6.5×10^{-9}

TABLE XI. ASSUMED CONCENTRATIONS OF ORGANIC CHEMICAL CONTAMINANTS IN SEAFOOD USED FOR CRITICAL GROUP EXPOSURE CALCULATIONS

Seafood type	Contaminant concentrations (mg/kg)					
	ΣPCB	ΣDDT	Dieldrin	Chlordane	HCB	Benzo-a-pyrene
Surface and intermediate depth fish	0.2	0.02	0.01	0.01	0.01	–
Crustaceans	–	–	–	–	–	0.002
Molluscs	0.03	0.01	0.01	0.001	0.005	0.02

TABLE XII. RISKS TO THE CRITICAL GROUP ASSOCIATED WITH INGESTION OF ORGANIC CHEMICALS IN SEAFOOD

Chemical	Intake rate (mg/a)	Lifetime risk from seafood consumption	Annual risk from seafood consumption[a]
ΣPCB	45	7.7×10^{-3}	1.1×10^{-4}
ΣDDT	4.8	6.2×10^{-5}	8.9×10^{-7}
Dieldrin	2.6	3.1×10^{-3}	4.4×10^{-5}
Chlordane	2.2	1.4×10^{-4}	2.0×10^{-6}
HCB	2.4	1.6×10^{-4}	2.3×10^{-6}
Benzo-a-pyrene	0.8	3.6×10^{-4}	5.1×10^{-6}

[a] Assuming an arbitrary value for life expectancy of 70 years.

TABLE XIII. RISKS OF DEVELOPING FATAL CANCER FROM EXPOSURES TO RADIONUCLIDES AND SUSPECTED CHEMICAL CARCINOGENS IN SEAFOOD FOR MEMBERS OF THE CRITICAL GROUP OF SEAFOOD CONSUMERS

Source	**Lifetime fatal cancer risk**	**Annual fatal cancer risk[a]**
ΣPCB	7.7×10^{-3}	1.1×10^{-4}
Naturally occurring radionuclides	–	9.0×10^{-5}
Dieldrin	3.1×10^{-3}	4.4×10^{-5}
Benzo-a-pyrene	3.6×10^{-4}	5.1×10^{-6}
HCB	1.6×10^{-4}	2.3×10^{-6}
Chlordane	1.4×10^{-4}	2.0×10^{-6}
ΣDDT	6.2×10^{-4}	8.9×10^{-7}
Peak value for maximum projected sea dumping of radioactive waste (Scenario C)	–	6.5×10^{-9}
Peak value for past sea dumping of radioactive waste - peak (Scenario A)	–	1.1×10^{9}

[a] The annual risks for radionuclide exposure are risk per year of exposure or intake. The annual risks for chemical carcinogen exposure are the lifetime risks (based on the linearized multistage model) divided by an assumed life expectancy of 70 years. This corresponds to an assumption that the fatal cancer probability commitment per unit intake is independent of age.

Table XII gives the calculated intake rates and corresponding lifetime and annual risks for organic chemical contaminant consumption in seafood. Not all routes of seafood exposure, depicted in the diet of the critical group (Table XI), could be included in these calculations due to the limited data available for the concentrations of chemicals in certain categories of seafood.

5.2.4. Comparison of average individual risks to members of the critical group

Table XIII summarizes the average aggregate risks to individuals in the critical group from all seafood pathways considered previously. Although the risks to members of the critical group are considerably larger than the average risks to individuals in large populations expressed on a per caput basis, the comparative magnitudes of the risks associated with radionuclides from different sources and chemical contaminants assumed to be carcinogenic are similar. The highest risks are those associated with natural radionuclides, ΣPCB and dieldrin. Other chemicals (benzo-a-pyrene, HCB, ΣDDT) appear to pose risks about one to two orders of magnitude lower while radionuclides released from low level radioactive wastes dumped at sea pose a risk that is a further two orders of magnitude lower.

6. DISCUSSION AND CONCLUSIONS

No human activity is without risk. Accordingly, practices involving the release of chemicals and radioactive substances into the environment pose risks. All foodstuffs including seafood contain both natural and artificial radionuclides and also natural and artificial carcinogens. In the preceding sections, estimates have been made of the potential risks of cancer fatality associated with human exposures to radionuclides and known or suspected cancer causing agents through seafood consumption.

The purpose of this document is to provide perspective for the estimated risk of fatal cancer induction associated with sea disposal of low level radioactive waste with other types of risks to humans, and in particular, to present comparisons with the estimated risks arising from other practices that have contaminated seafood with suspected cancer causing chemicals.

In response to the LC 72 request to "review and summarize available scientific information on estimates of risks ... that result from various human activities", a representation of the magnitude of more general risks posed to individuals within developed society is provided in Section 2 and illustrated in Fig. 1. These latter risks, associated with disease, accidents, consumption of substances and natural phenomena, range over about five orders of magnitude from 10^{-2}/a to 10^{-7}/a.

The LC 72 request requires the inclusion of "risk estimates from other human uses, application, disposals and dissemination of potentially hazardous substances" in the review of risks to human well-being. Therefore, estimates of risks associated with carcinogenic substances released by various activities to the marine environment have been calculated. In order to provide a "common and comprehensible basis" for the comparison of the risk associated with the sea dumping of low level radioactive waste with the risk associated with carcinogenic chemicals observed in the marine environment, only risks linked to seafood ingestion pathways have been considered. In this context it is appropriate to differentiate between the risks to high rate seafood consumers (individuals in a critical group) and the risks to 'average' consumers. The latter are represented by average per caput risks to individuals within large populations. Only a few of the man-made carcinogens, and none of the natural carcinogens, have been considered here because of the limited information available to deal with a broad range of these substances in a consistent manner.

The relative risks to a common critical group of high rate seafood consumers are summarized in Fig. 2. The presence of naturally occurring radionuclides, principally ^{210}Po, can result in a risk greater than that corresponding to the ICRP dose limit for members of the public [46, 47]. By comparison, the additional risks associated with the contamination of seafood from sea disposal of radioactive wastes are about five orders of magnitude lower. It would appear that the presence of ΣPCB could represent a similar risk to that arising from naturally occurring radionuclides. The other chemicals present risks that are lower, relative to that of ΣPCB, but still very much larger, by more than two orders of magnitude, than those arising from sea disposal of radioactive wastes.

Also of interest are the average individual, or per caput, risks summarized in Fig. 3. The average risks posed by chemicals that are now ubiquitously present in seafood and those associated respectively with the discharge of low level liquid radioactive waste to coastal waters, the testing of nuclear weapons, and the Chernobyl accident, are all of a similar magnitude. By comparison, the incremental risk arising from deep sea dumping of radioactive wastes is three to four orders of magnitude lower. Even on a global scale, organic chemicals such as ΣPCB, ΣDDT, and no doubt others, potentially present a substantially greater level of risk than exposures associated with sea dumping of radioactive waste.

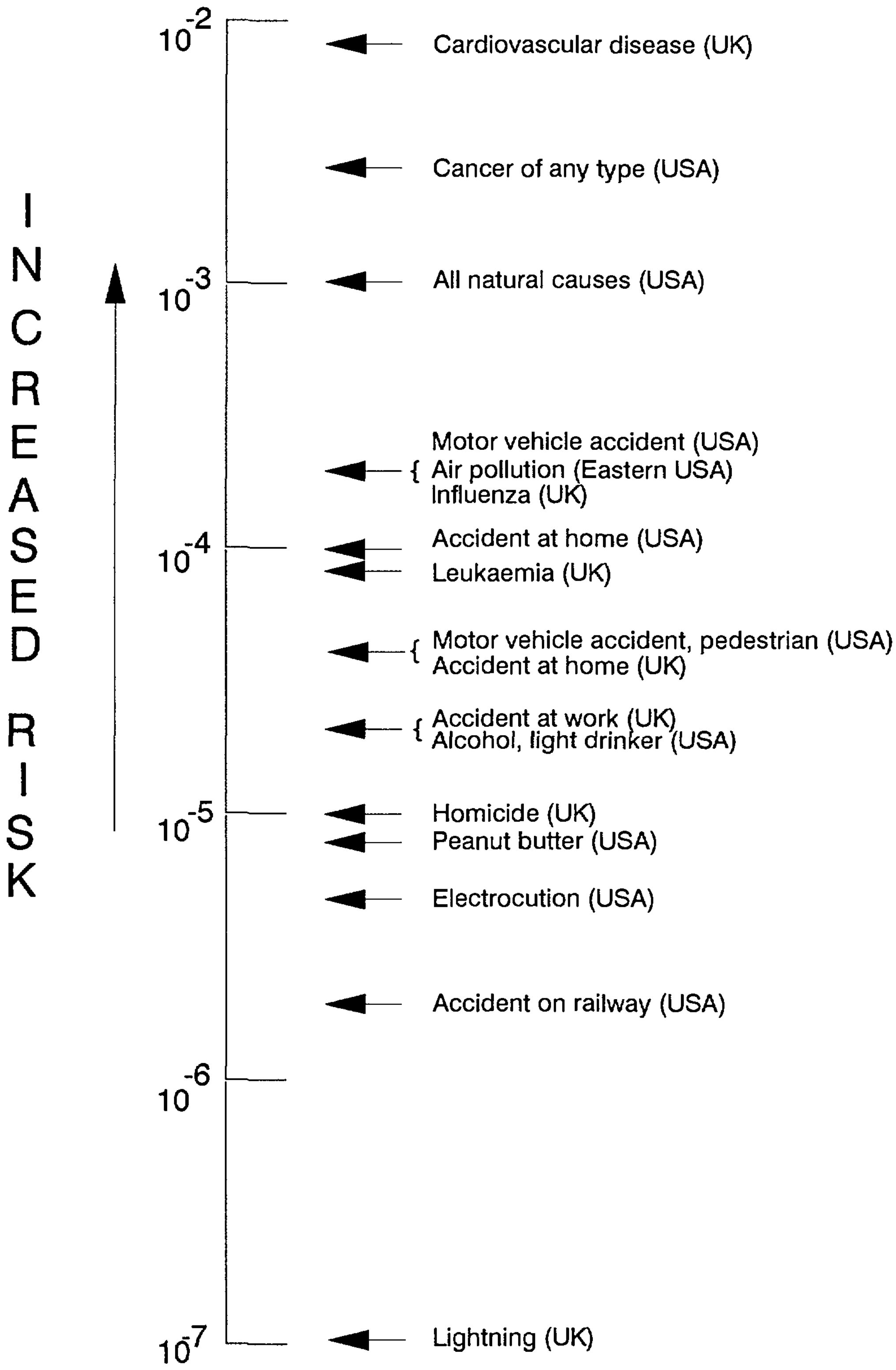

FIG. 1. Annual probability of death from various causes in UK and USA populations.

The estimated annual communal harm caused by cancer is summarized in Fig. 4. The health detriment arising from naturally occurring radionuclides in seafood is about 1% of that which could be attributable to the natural radiation background [37]. It is also at least two orders of magnitude greater than that associated with any other source of radionuclides, and some six orders of magnitude greater than that arising from the deep sea disposal of radioactive wastes. Quite clearly, none of the estimated cancer deaths

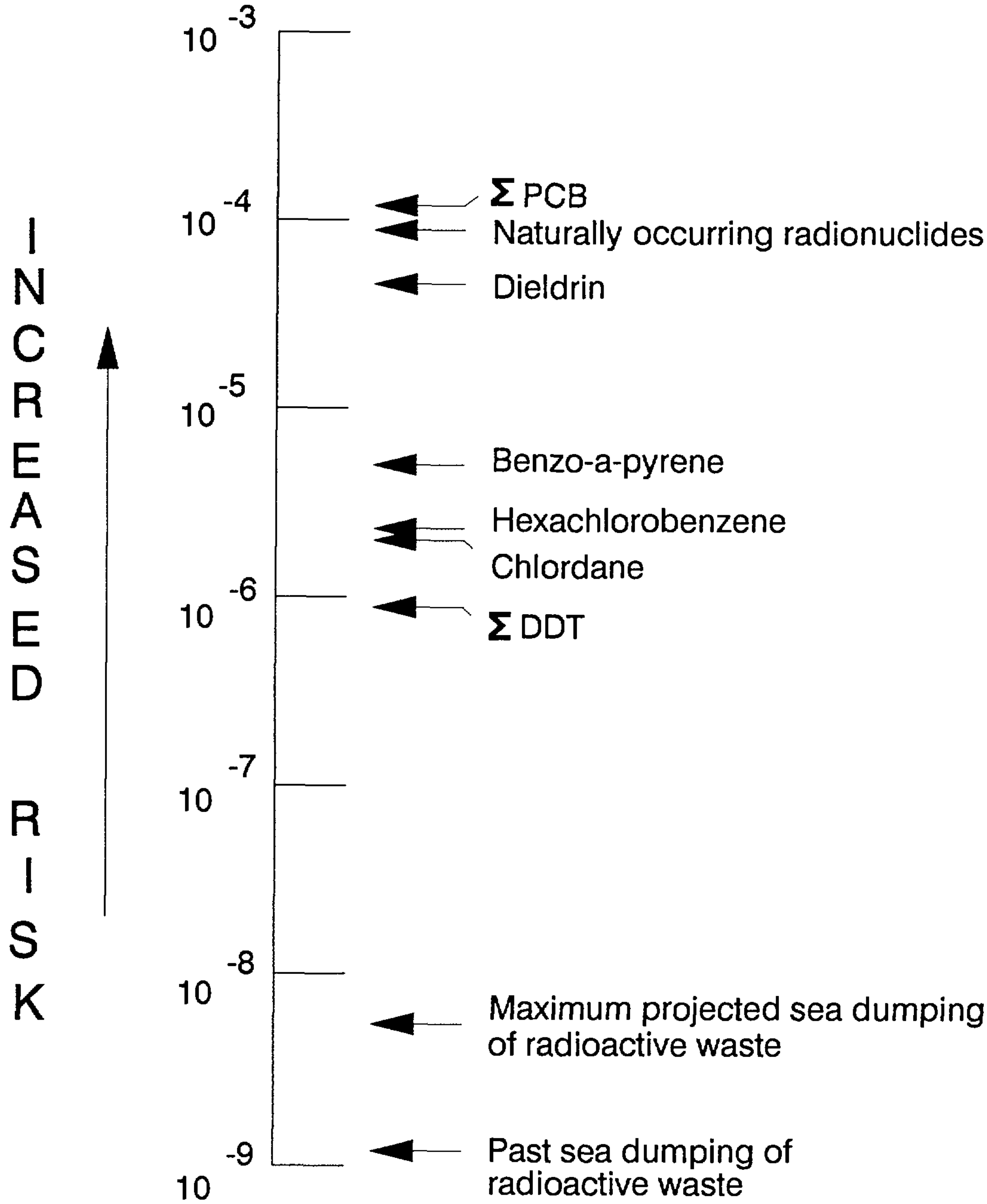

FIG. 2. Annual risk of fatal cancer induction associated with the ingestion of radionuclides and organic chemical carcinogens by members of the most exposed group.

from such sources could ever be directly determined by epidemiological studies, and must therefore stand as estimates based upon a comparative theoretical exercise. It is nevertheless interesting to note the comparatively high rate of fatality potentially associated with exposures to organic contaminants through seafood consumption which rank, in some instances, close to those associated with natural radionuclides.

It may well be argued that such estimates of annual communal harm are not as important as estimates of committed harm. Unfortunately, such comparisons can only be made for radiation exposures. Accordingly, the only means of putting the committed harm estimated for deep sea disposal of radioactive wastes into perspective is to draw a comparison with the health detriment associated with naturally occurring radionuclides. The latter constitutes an essentially constant source, and therefore deep sea disposal of radioactive waste constitutes a level of harm at least six orders of magnitude lower,

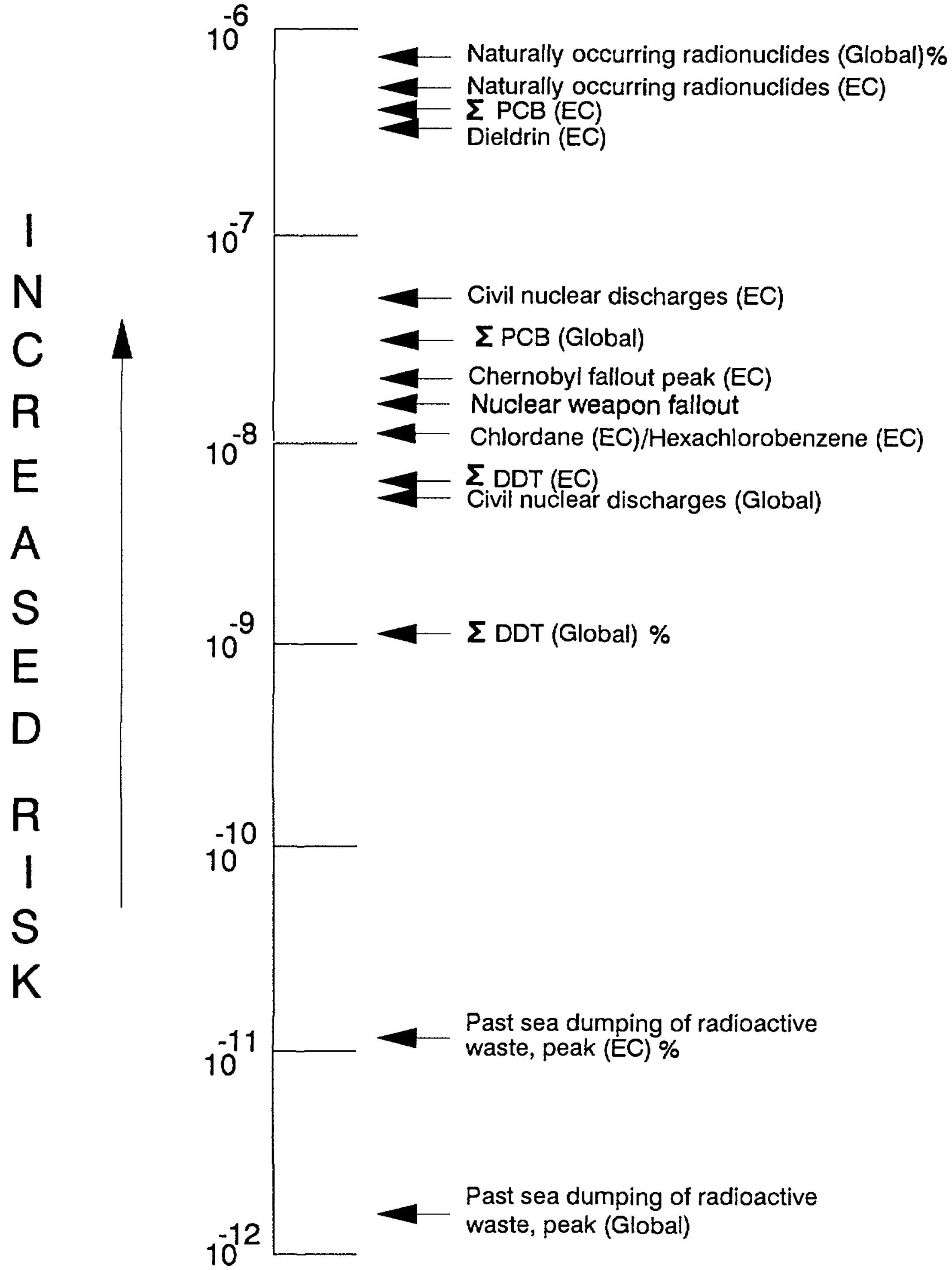

FIG. 3. Per caput annual individual risk of fatal cancer induction within the global and EC populations associated with ingestion of radionuclides and organic chemical carcinogens through seafood pathways.

regardless of the period of integration. It would be of interest to draw comparisons between the committed communal harm from low level radioactive waste disposal at sea with that arising from chemicals having carcinogenic properties that are present in the marine environment. It should not prove to be too difficult to do so given the appropriate information. Mathematical models that have been used to derive the estimates for radioactive waste disposal contain all of the essential features required to make similar comparisons with other chemicals. The most important requirements are information on

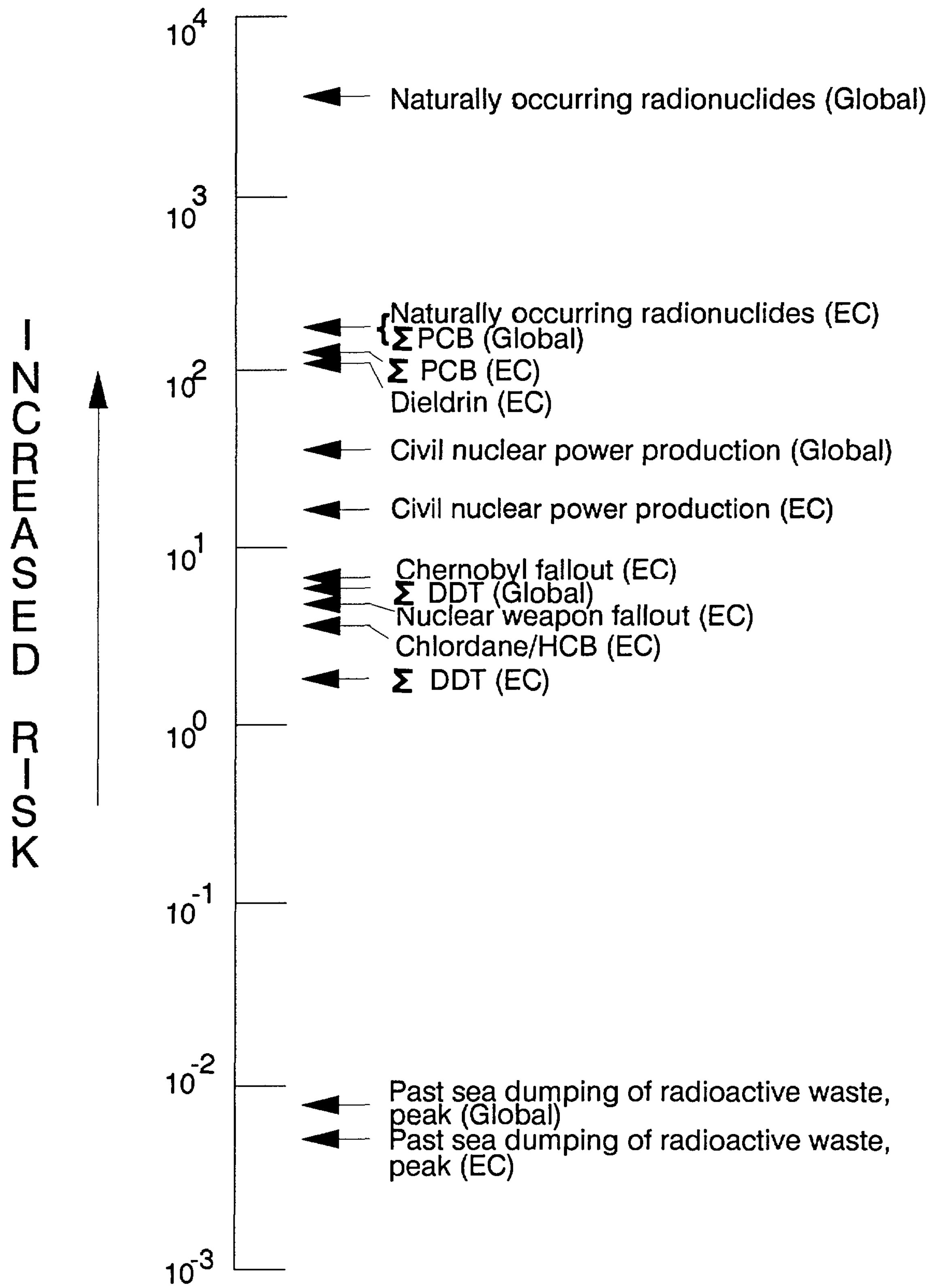

FIG. 4. Annual harm (incidence of fatal cancer) potentially associated with the ingestion of radionuclides and organic chemical carcinogens through seafood pathways in global and EC populations.

the rates of breakdown of the chemicals (analogous to radioactive decay) and the production of any cancer causing daughter products, plus information on particle-water partition coefficients, biological concentration factors, and the annual rates of input to the marine environment for these substances.

A most important point to make, however, is that much greater effort to assessing the risks and detriment arising from potentially carcinogenic substances in seafood is warranted. This would both enhance public appreciation of the potential of the risks associated with the dissemination of such chemicals into the environment and also provide an improved perspective with which to judge the risks associated with sea disposal of radioactive wastes. Only a few such substances were considered in this assessment. These were selected on the basis of their widespread occurrence in fish and evidence for their carcinogenicity (e.g. dieldrin, chlordane). There are several other chemicals for which both human and animal data are sufficient to conclude that the substances are human carcinogens. Benzene, benzidine, bis(chloromethyl)ether, chloromethyl methyl ether, chlorambucil, chromium, nickel, oestrogens, and vinylchloride belong to this group. The number of relevant compounds is even larger when differences between the results of experimental animal and human exposures are ignored. There is a more extensive list of chemicals that have been shown to be carcinogenic to animals but without data (or only inadequate data) on human carcinogenicity. Whether human carcinogenicity is proven or suspected, it must be appreciated that, for risks to humans associated with seafood consumption, a chemical is only relevant when it can induce cancer through ingestion. It is therefore suggested that the prevalence of substances that may induce cancer following dietary intake be further investigated. This includes the chemicals considered in this report so that all important exposure pathways, both regional and global, can be evaluated more authoritatively.

Some additional comment on these comparisons among individual risks is warranted. The incremental risks estimated here cannot be summed because of the nature of the uncertainties involved in the assumptions and calculations. It is nevertheless questionable whether the comparison between risks posed by nuclear practices, which take account of the potential effects of **all** radionuclides released by a practice, and the risks associated with **individual** chemicals, such as dieldrin, in the environment is an equitable one. Individual agro-industrial practices often release a variety of chemicals to the environment and the most equitable comparison with nuclear practices would take account of the aggregate risks associated with exposures to all such chemicals.

No judgements have been made in this report as to whether any of these levels of risk could be considered acceptable or unacceptable, either to those who make decisions concerning waste disposal practices or to members of the public. Nevertheless, it is inevitable that comparisons will be drawn between these risks and the other risks to which the community at large is commonly exposed.

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