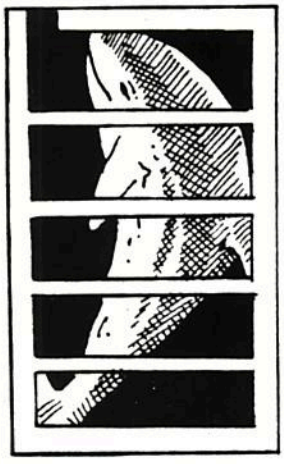

GREENPEACE

CETACEAN MASS MORTALITIES AND THEIR POTENTIAL RELATIONSHIP WITH POLLUTION

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1. INTRODUCTION

In recent years there has been an increase in the frequency and apparent severity of mass mortalities affecting marine mammals. Considerable concern has been voiced that ubiquitous pollution could be contributing to these incidents. The extent, duration and severity of these epizootics are doubtless affected by many interacting factors; included amongst these could be ambient and preceding climatic conditions, high population density, and changes in species distribution, as well as the nature of the diseases concerned (Dietz et al., 1989; Lavigne and Schmitz; 1991, Simmonds, in press).

Since 1987 there have been at least five mass mortalities in marine mammal populations: 1987-1988 - a minimum of 2,500 bottlenose dolphins died on the eastern seaboard of the U.S.; 1987-1988 - some 8,000-10,000 endemic seals died in Lake Baikal, Siberia; 1988 - some 18,000 harbour seals died in the North Sea; 1990 - several hundred bottlenose dolphins died in the early months of 1990 in the Gulf of Mexico and, most recently, in the Summer of 1990, several thousand striped dolphins died in the Mediterranean (Dietz et al., 1989; Geraci, 1989; McKay, 1989; Aguilar and Raga, 1991; Simmonds, in press). All these events (except perhaps that in Siberia) occurred in waters adjacent to highly populated and industrialised coasts. In addition, there is evidence of unprecedented mortalities in the sperm whale populations of the N.E. Atlantic.

2. THE SEAL MORTALITIES

2a. The European Seal Epizootic

Seals and dolphins have many aspects of their biology in common. Both are top predators - feeding largely on fish - and both use insulating blubber stores in a similar way. Seals, however, are easier to study and consequently substantially more is known about the effects of contaminants on them. The 1988 seal epizootic which occurred in Northern Europe is particularly well documented. The event generated tremendous debate about the role of pollution in this and other mortalities (Simmonds, 1991).

Comparison with the past history of epizootics in seals (something which is not true of dolphin species), showed "striking differences between this [1988 seal epizootic] and earlier events, in terms of mortality rate, rapidity and range of spread" (Dietz et al., 1989). The rapid advancement of the disease in the seal herds clearly indicated a non-resistant population (Dietz et al., 1989) exposed to a highly pathogenic disease (Simmonds, 1991).

An investigation, funded by Greenpeace with analyses carried out by the British Institute of Terrestrial Ecology, Monks Wood, compared the pollution burdens of harbour seals, Phoca vitulina (Linnaeus, 1758), which died during the epizootic in eastern Scotland and the Norfolk Wash (Simmonds et al., in prep.). Mortality clearly varied between seal colonies in the UK. It was high off Norfolk (as elsewhere in Europe about 50-60% of the population died) but low, if not negligible in Scotland (Harwood, 1989, 1990 and NERC, 1989) (table 1). The pollution study found that the Scottish harbour seals sampled had far lower levels of contaminants in their tissues than those from Norfolk (table 2).

The two groups of seals were of similar ages and all died during the 1988 event but the Wash samples had a mean DDE (the stable metabolite of DDT) blubber concentration (2.07 ppm) almost twice that of the Scottish seals (1.37 ppm). PCBs were also more concentrated (mean 7.17 ppm, compared with a value of 1.03 ppm found in only one out of the 9 Scottish seals sampled). In fact, of all the organochlorine compounds which might have been detected, only DDE and, in a single individual, PCBs were found in the Scottish animals.

Following intensive scientific and public speculation, a morbillivirus was isolated from the seals. After initial confusion with Canine Distemper Virus, it was identified as a new virus "Phocine Distemper Virus" (PDV). If PDV had not reached Scotland in 1988, or had reached significantly fewer Scottish seals than elsewhere, then the lower mortality observed there might be more easily explicable. This was not the case, however, as similar proportions of the surviving populations in Scotland and Norfolk were found to been infected (Harwood et al., 1989).

Surviving seals carrying PDV-antibodies show that infection did not inevitably lead to death and, even in seals which ultimately died, sudden death was by no means always the case. Some seals survived for many days in a debilitated condition. Typically they had pneumonia-like symptoms and were unable to feed. This would have caused them to mobilise lipids from their blubber-stores and, indeed, depleted blubber layers were observed in some of the seals sampled (Simmonds, 1991). This acute mobilisation of lipids would also have caused an accelerated release of lipophilic compounds (such as PCBs and DDT) from their blubber.

Once present in raised concentrations circulating in the seals' bodies, these toxic contaminants may have had very significant effects - producing an additional stress, which for some seals, tipped the balance from recovery from PDV-infection to death. So, the data indicates that seals from Scottish colonies were indeed not only less contaminated than those living off more polluted coasts but that they also had a higher survival rate.

2b. Other seal mortalities.

Several other seal epizootics have been reported this century and the environmental factors which have been linked to these are outlined in Section 4d. Too little is known about pollution in Lake Baikal presently to comment on its possible implications for the recent die-off of the Baikal seal, Phoca sibirica (Gmelin, 1788). Nevertheless,

in 1987-88 about 10% of the original population of 80,000 to 100,000 died. A similar virus to PDV has been implicated in the mortality (Dietz et. al., 1989).

3. CETACEAN MORTALITIES

3a. The Mediterranean Striped Dolphin Mortality

Very similar recent mass mortalities are now known to have occurred in two dolphin species. The most recent and probably largest of these was in the western Mediterranean.

Since the summer of 1990 abnormally high numbers of dead and dying striped dolphins, Stenella coeruleoalba, (Meyen, 1833) have been washing ashore on Mediterranean coasts. The first few dolphins were found on beaches near Valencia, in early July, but the full gravity of the situation was not realised until later in the year (Aguilar and Raga, 1991). By the end of October the number recorded from Spanish coasts had reached four hundred. They also stranded on French and Italian shores as the mortality spread, comprising, in total, some 700 recorded strandings. Only a small proportion of the dead will have been washed into shore as the striped dolphin is a deep water species, normally living far off the coast. The mortality is, therefore, believed actually to have involved several thousand dolphins.

The Spanish Oceanic Institute, Greenpeace, ICONA and others initiated a programme to collect dolphin bodies for pathological and toxicological examination. The Universities of Barcelona and Valencia co-ordinated. Initial epidemiological studies indicate that the event seemed to spread rapidly eastwards, perhaps matching the pattern of dolphin migration. The dead and dying animals exhibited a range of symptoms. Lesions, attributable to viral infection, were present in lungs, nerve tissue and the lymphatic system (Aguilar and Raga, 1991).

Furthermore, despite sporadic strandings of other dead cetaceans at much the same time and in the same area, it was clear that only one species was affected and that it was suffering a very high mortality rate. A survey of the remaining populations out to sea (Aguilar et al., 1991) found only very depleted schools in the region. So, in terms of severity, species-specificity and symptoms this mortality resembled the 1988 seal epizootic. Once again suggesting the presence of a highly infectious, pathogenic agent and a susceptible population. A morbillivirus was proposed as the cause and this led to fears that the highly endangered Mediterranean monk seal, Monacus monachus (Hermann, 1779), might also be threatened (Pain, 1990). However, whilst a morbillivirus has since been found in the dead dolphins, pathology has revealed a complex picture (Aguilar and Raga, 1990) which indicates that factors including, but not limited to, a morbillivirus infection were involved.

Morbillivirus infections are generally fast-acting but the dolphins seemed to have been in poor condition for some time prior to the infection. Lipid reserves were around 45% of the value expected in healthy striped dolphins. Parasite burdens were very heavy - including numbers of ecto-parasites, indicating that the striped dolphins had probably been debilitated (swimming slowly) for a relatively long

period of time. Furthermore, practically every dolphin examined had significant liver lesions which did not appear to have been caused by a morbillivirus infection.

Earlier studies on striped dolphins in the Mediterranean revealed high levels of contaminants in their tissues. Toxicological studies on the dead dolphins from this mortality, however, (conducted by the Department of Animal Biology, University of Barcelona) revealed even greater contaminant concentrations (especially PCBs) (Aguilar and Raga, 1990). The earlier studies had found average blubber concentrations of 326 ppm (Aguilar and Raga, 1989) but, in dolphins from the 1990 epizootic, values exceeding 2,500 ppm were recorded. Aguilar and Raga have put forward the hypothesis that the high body burdens of PCBs produced immunological deficiencies making the animals more vulnerable to infection and, once the infection had been initiated, less capable of combating it.

The mobilisation of lipids which preceded the mass mortality is likely to have been caused by an earlier infection (or some other stressing event). The liver damage (a lesion not compatible with being of viral origin) would then have been caused by elevated levels of PCBs released from the blubber into their general body circulation.

The high concentrations of PCBs found in the dead dolphins in 1990 may be there because depletion of lipids in the blubber not only released the PCBs but also caused those remaining in the depleted blubber layer to become more concentrated. It is also possible, of course, that it was the more highly contaminated individuals within the population which died and then washed ashore.

Alex Aguilar of Barcelona University (cited in Luke, 1991) noted, however, the poor nutrient status of the western Mediterranean Sea preceding the mortality. The weather pattern seemed to be unusual with less water mixing than normal and low rainfall causing comparatively little nutrient-bearing run-off to enter the sea. This, in turn, could have reduced the abundance of the dolphins' normal prey species. Low prey abundance could, in turn have led, or contributed, to the dolphins' initially poor condition prior to morbillivirus infection. A schematic diagram showing the events which may have led to the mortality is given in Figure 1.

The potential contribution of "natural" toxins to this mortality, specifically those produced by some phytoplankton, has also been considered (Aguilar and Raga, 1990). The Mediterranean at this time, however, was not only relatively nutrient poor but there was no contemporaneous record of major algal blooms.

3b. The US East Coast die-off

In June 1987, unprecedented numbers of Atlantic bottlenose dolphins, Tursiops truncatus, Montagu, 1821, began to wash ashore along the coast of New Jersey, USA, and the US National Oceanic and Atmospheric Administration (NOAA) established a team to investigate. Over 740 bottlenose dolphins stranded along the Atlantic coast during the 11 months that followed. Again only a fraction of the dolphins which actually died are thought to have come ashore and more than half of the inshore migratory population are believed to have died. The die-off

progressed southwards in a wave of strandings of dead and dying individuals, following the course of the dolphins' migration in the autumn and winter of 1988. The whole event generated great media interest and public sympathy and concern and the researchers in their final report on this mortality noted that, at that time, "the event was unparalleled" (Geraci, 1989).

The dolphins suffered a range of disturbing symptoms. They commonly had large open "crater" lesions on their heads and in their mouths (Geraci, 1989). On some animals, considerable areas of skin were found to be sloughing off, exposing reddened dermis - associated with the breakdown of blood vessels. These and other symptoms seemed to be the result of massive bacterial and fungal infections, which were also identified as the final cause of death of many of the dolphins. Related oedema was found in many organs and blood-tinged fluid in the thoracic and abdominal cavities. Whilst significant, the bacterial and fungal invasions appeared (as in many other disease events) to be opportunistic secondary infections.

In fact, many of the dolphins' symptoms were in accord with immune system dysfunction. Lymphoid follicles in the spleen, lymph nodes and intestine were found to be depleted (Geraci, 1989). This is again comparable with the 1988 seal epidemic when it was secondary pathogens which, in the majority of cases, ultimately caused death (Simmonds, 1991).

It was not until February 1989 that NOAA announced the investigation's conclusions at a major press conference. The researchers said that the dolphins were poisoned by eating fish tainted by a naturally-occurring toxin, "brevetoxin", from "red tide" algae (NOAA press conference and reported in McKay, 1989). The basic evidence for this was that the toxin was found in eight out of seventeen dolphins which were tested. Pollution was at that time categorically rejected as a significant factor in the die-off.

The investigators' final report (Geraci, 1989) was released later, in April, and repeated the algal poisoning theory. The hypothesis was that part of a "red tide" (a poisonous algal bloom) was transported to the East coast of Florida by the Gulf Loop Current - Florida Current - Gulf Stream system. Such an event had actually been recorded in the autumn of 1987, although the toxin was found three months earlier in stranded dolphins in Virginia. Planktivorous fish migrating north could, the investigators suggested, have consumed algae from an earlier and undetected bloom. Dolphins, also moving north and feeding on these fish (or their predators) were then exposed to the toxin over a prolonged period, with effects only starting to become manifest as they reached the mid-Atlantic coast.

Not all dolphins could have been poisoned by directly eating fish because brevetoxin was also found in the livers of 3 nursing calves and must have been transferred to them in their mothers' milk (Geraci, 1989). In this respect, therefore, brevetoxin was proposed to be acting rather like fat-soluble contaminants (such as PCBs) which can also be passed on in milk.

The brevetoxin-poisoning hypothesis was apparently without precedent, although in the final report it was presented as the trigger for the

die-off rather than the sole cause. A number of significant problems exist with this theory and these are summarised in table 3. The known effects of brevetoxin and PCBS are compared with the effects observed in the dolphins in table 4.

It is reported that the investigators, at an early stage, decided to ignore the implications of the animals' contaminant burdens (McKay, 1989). A memorandum dated August 1987 between government scientists carried the instructions that "no special attention will be drawn relative to these data [PCB and pesticide levels] and that a blanket statement will be made that the levels of these components were not out of the ordinary".

A Congressional hearing on the conclusions of the die-off investigation was held in May, 1989. The hearing found that the conclusion released to the world's press - death by natural causes - was less definitive than it had appeared (McKay, 1989). In fact, however, although the algal-poisoning theory was challenged, the notion that brevetoxin alone was responsible had already been widely reported by the world's press.

One other interesting discovery about the US dolphins (which seemed to have been made between the February press conference and release of the final report) was that serological titres to a morbillivirus were raised in six out of 13 blood samples examined. Bearing in mind the other recent morbillivirus infections in marine mammals, this deserves more investigation. Morbilliviruses are difficult to detect. The presence of detectable antibodies in only a proportion of animals does not preclude the possibility that they were all infected by one of these highly pathogenic agents. Similarly, the significance of the high contaminant burdens of the dolphins cannot simply be dismissed.

3c. The Gulf of Mexico Mortality

A subsequent mortality of the same species has occurred in the Gulf of Mexico between January and May 1990 (McKay, pers. comm.). No report has yet been produced on this event although several hundred dead animals are thought to have come ashore. Again, many others may have been lost at sea - some to the local shark population.

Initially, during the winter, there was speculation that an exceptionally cold spell in the Gulf might be responsible. The animals, however, went on stranding at abnormally high levels for several months after this. It should also be noted that brevetoxin-producing blooms are a common occurrence in the Gulf, but not off the US East coast. Dolphins, however, seem to have co-existed with them in these comparatively warm waters for millennia without mortalities being induced. What is new here, however, is the heavy industry which now lines the shores and estuaries of the region and which pumps its wastes into the sea.

3d. The humpback whale deaths.

Other marine mammal mortalities similarly, hypothetically, linked to algal toxins are very rare. Between November 1987 and January 1988, however, 14 humpback whales, Megaptera novaeangliae, Borowski, 1781, were stranded dead along the beaches of Cape Cod Bay and the northern

Nantucket Sound. In this case, researchers reported that the whales died after eating Atlantic mackerel containing saxitoxin (another algal poison) (Geraci et al., 1989).

Again, a complex scenario resulting in algal-intoxification of the whales was proposed, along with the idea that yet another marine mammal species is vulnerable to a ubiquitous marine toxin. "Saxitoxin" was found to be present in fish sampled at the time and place where the whales were feeding. Extracts from whale kidneys (in 3 out of 8 kidneys sampled), livers (4/7) and stomach contents (7/9) caused "mortality characteristic of STX [saxitoxin] in mice" but STX was not identifiable using standard diagnostic-analytical (HPLC) techniques, nor was it detected in New England shellfish or plankton sampled in the vicinity and at the time of the mortality. The researchers propose that either the fish carried STX from planktonic sources in the St. Lawrence Gulf to Cape Cod in October-November or the toxin in the mackerel was actually a bacterial metabolite.

Overall, the idea that cetacean mortalities relate to the occurrence of algal toxins in their environments seems, for the present at least, to be based on scant evidence. By contrast, evidence for pollution having a significant impact on the health of marine mammals is considerable and reviewed in Section 4 following.

3e. The sperm whale strandings

Before moving on to the toxicological evidence, however, it is important to note that the list of recent marine mammal mortalities is probably not complete. This may be true of whales, in particular, which could die at sea and never be found. There is some evidence that another significant mortality of a great whale species has occurred because abnormally high numbers of sperm whales, Physeter macrocephalus, Linnaeus 1758, have stranded in recent years on European and Norwegian coasts (Christensen, 1990 and Berrow et al., 1991).

Details are as follows: in 1988, in Norway, 27 stranded (and 9 were observed drifting offshore); in Sweden 1; Denmark 1; Belgium 1 and on the Faroe Islands 2. Making a total of 32. Similarly in 1990 on UK and Eire west coasts approximately 12 stranded and in Norway 20 - making again a total of 32.

Data on previous levels of sperm whale strandings is scant but the reported levels of these whales stranding on British coasts (where there has been an observer system since 1913) indicate that only 12 stranded between 1913 and 1966. Strandings schemes have been incomplete elsewhere but there may have been an overall increase in British sperm whale strandings this century, with a significant increase since the 1950s (Berrow et al., 1991). This could reflect an increase in the density of these whales in the NE Atlantic but researchers have also noted that the 1990s strandings should not simply be interpreted in this way. Christensen (1990) has posed the question of whether these mortalities relate to other recent marine mammal die-offs.

The stranded sperm whales were in too poor a condition for any pathological investigations, but the possibility that one of the "great

whales" has also been involved in an epizootic has significant implications for their conservation and must not be ignored. There is no reason to hope that the bigger whales are exempt from the factors causing mass mortalities in other marine mammal species. Unfortunately, little is known about the amount of contamination in any of these larger species. Baleen whales, as filter feeders, may be lower on the trophic web and, perhaps, do not accumulate such high concentrations of pollutants. This would not, however, be true of the other large "toothed" species - including the sperm whales.

4. THE TOXICOLOGICAL EVIDENCE

4a. The PCB story.

By contrast, a considerable amount is known about the kind of compounds which preferentially accumulate in the ample fatty tissues of many marine mammals. Most is probably known about the PCBs (the polychlorinated biphenyls). It is important to note that this is only one group of chemicals but they are, in many ways, typical of this class of persistent yet highly biologically active chemicals presently of greatest concern with respect to marine wildlife - the halogenated hydrocarbons.

Brief consideration of the PCB family (209 compounds) will help explain their significance. The compounds differ from each other according to the number and position of chlorine atoms in the molecule. They are poorly or non-degradable, accumulate in the food chain and are "toxic" (Johnston and Simmonds, 1989; van de Gaag and Marquenie, 1991). PCB-mixtures are used in a range of products where chemical stability is important and some also have useful dielectric properties. PCBs are, therefore, found in flame-resistant coatings, drilling equipment, fluorescent light ballasts and transformers. Commercially available mixtures are often contaminated with very toxic dibenzofurans and PCB mixtures which have been exposed to high temperatures also contain these highly toxic and persistent compounds.

More than two thirds of the estimated global production of PCBs (an estimated 2 million tonnes) is presently still in use in industrialised countries. Shortly, much PCB-containing equipment will reach the end of its useful life and many scientists feel that the threat from the ultimate release of these substances is extremely acute. Dutch scientists (van de Gaag and Marquenie, 1991) have noted the following -

"An uncontrolled release of only part of this 'chemical time bomb' would mean a world-wide threat to vulnerable animal species. Predators at the top of food webs in aquatic ecosystems would be the first victims".

Furthermore, Japanese researchers have estimated that even if as much as 50% of the world's existing PCBs were to be destroyed in the next ten years, a reduction in PCBs in marine mammals would only occur far off in the 21st century.

PCBs are ubiquitous environmental contaminants, which spread world-wide primarily in 2 ways; via atmospheric and by aquatic transport. About 1-10% of global production is estimated to have so far reached the

oceans (van der Gaag and Marquenie, 1991). Areas where sedimentation occurs are likely to be especially badly impacted by water-borne contaminants. It is, therefore, no surprise that PCB concentrations are particularly high in seals and sea birds, for example, in the Wadden Sea. PCBs and related compounds are, however, even detectable in remote polar regions in wildlife and even the indigenous human populations. PCBs, chlordane-related compounds, DDT and other organochlorines, for example, are even detected in arctic cod, polar bears and ringed seals (Muir et al., 1988).

4b. The Toxic Effects of PCBs

It is already widely acknowledged that fish- and shellfish-eating top predators (potentially including humans) are the major group at risk. Put simply, this is because mammals and birds consume highly contaminated food and accumulate PCBs (and related compounds) in their fatty tissues and elimination of PCBs is very limited except during reproduction processes (van der Gaag and Marquenie, 1991). Birds, for example, mobilise their fat stores to produce eggs and and thus fat-soluble of "lipophilic" contaminants may be deposited inside the eggs in substantial quantities. Similarly, mammals utilise lipids in their adipose stores during gestation and lactation. PCBs and other compounds liberated at this time may be transmitted in high and even potentially lethal concentrations to offspring.

Consideration of the toxic effects of PCBs on mammals is a complex issue (see Safe, 1984; Martineau et al., 1987; Johnston and Simmonds, 1989; and van der Gaag and Marquenie, 1990;). One problem is the variety of PCBs (and other contaminating compounds) present in the original commercial mixtures which are released to the environment. Moreover, the composition of PCBs found in the environment, including in animal tissues, differs from those in the original commercial mixtures because of various transformation and degradation processes. The more highly chlorinated PCB compounds tend to persist.

In laboratory studies PCB mixtures have been shown to cause a wide-range of responses in animals, these are summarised in table 5. They have been found to impair significantly the immune function of chickens, ducks, guinea pigs, mice, rats, monkeys, rabbits and seals and reproductive effects in another range of species (see Table 6 and Safe, 1984). Further reproductive effects have also recently been found in humans and should be added to this list: a possible correlation has been found between miscarriages and levels of circulating PCBs (Leoni et al., 1989) and a similar "hypothesis of correlation" exists between late haemorrhagic disease in newborn babies and organochlorines in mother's milk (Koppe and Olie, 1989).

Other human developmental problems mediated by exposure to pollution have been revealed by an intensive study of fish consumers around the Canadian Great Lakes (Fein et al., 1984 and Thomann et al., 1987). The Great Lakes system is highly contaminated and the ingestion of fish caught there was found to lead to significant PCB exposure in the womb, related to subsequent low birth weight, small average head circumference, shortened pregnancy duration and in newborn infants poor autonomic and reflex functioning. Moreover, further infant exposure occurs via mother's milk.

Returning to wildlife - Gilbertson (1989) has noted that "over the past twenty-five years there has been increasing evidence of outbreaks of disease among fish and wildlife (epizootics) which could not be attributed to conventional etiological agents, such as microorganisms and parasites". These events are linked to environmental halogenated hydrocarbon (especially organochlorine) contamination and summarised in table 7.

Added to this list should be the recent work of Mason (1989) who has concluded that pollution has played a major role in the decline of a number of otter populations, especially in Western Europe. He noted that otters are "the top carnivores of wetland ecosystems" and "as such the most vulnerable of animals to pollution and can be considered indicators of the health of the wetland environment".

What applies to otters - applies equally, or perhaps even more so, to other aquatic fish eating mammals. Bioaccumulation occurs along their food chain - fat-soluble compounds accumulate in the upper microlayer of the oceans and in particulate matter (including plankton). This is ingested by microfauna which in turn is eaten by larger animals and then fish. At each step contamination levels increase several fold. Ultimately concentrations of fat-soluble-bioaccumulative materials thousands of times higher than those in the surrounding environment are reached. These materials are not locked away in the animal's blubber but a dynamic situation exists with respect to the rest of the animal's body (Reijnders, 1980). Exchange is increased when lipids are mobilised.

4c. Detoxification Mechanisms

As has already been noted, some PCB compounds are more frequently encountered in biological samples than others. A study of PCBs present in seal food compared with seal blood, for example, showed a significant difference (Boon et al., 1987), indicating metabolism of some PCB compounds. The precise effects of metabolism on actual PCB toxicity is, however, unclear. Metabolites can either be rapidly excreted ("detoxification"), or the more stable metabolites (and/or highly reactive intermediates) may cause adverse effects "intoxication" (or "poisoning").

One enzyme system - the cytochrome P-450 monooxygenase system - is a central catalyst in the oxidative metabolism and elimination of organic xenobiotics. There are more than 150 forms of cytochrome P-450 and each catalyses the metabolism of a comparatively specific group of lipophilic substrates. PCBs can interact with the cytochrome P-450 system in several ways: as inducers, substrates and inhibitors. Organochlorines and other chemicals which induce microsomal enzymes are classified according to their induction activities. Those behaving like the drug phenobarbital are classed as "PB-inducers" and those like methylcholanthrene as "MC-inducers". In rats, PB-type chemicals have been found to induce hepatic cytochromes P-450 a, b and e and MC-type induce a, c and d. PCBs, however, cause mixed-type induction actively as some are PB-inducers and others MC-inducers (Safe, 1984). In seals the mixed function oxidase induction activity referred to as the CYP2B subfamily (the same as that induced by the model compound phenobarbital) is much lower than that observed in other species.

Cetaceans, however, actually seem to lack the CYP2B group entirely (Boon et al., in press). Harbour seals and cetaceans appear to be only able to metabolise PCBs of certain types (technically - those with o,m vicinal H-atoms in combination with at maximum one ortho-chlorine atom) (Boon et al., in press) and seals have a significantly greater capacity than cetaceans to metabolise certain PCB compounds (those with vicinal H-atoms in the m and p positions). Similarly, data from a study of chlorinated biphenyls in the tissues of some marine mammals from the North Sea and Atlantic Ocean, indicated that seals had a greater ability to metabolise these compounds than porpoises. Open sea cetaceans were the least capable (Duinker et al., 1989).

The capacity of small cetaceans to metabolise PCBs has also been considered by Japanese researchers (Tanabe et al., 1988). The spectrum of PCB compounds accumulated in the tissues of several species of higher animals (including humans) was compared with those in Dall's porpoises, Phocoenoides dalli (True, 1885) and the data suggests that in most higher animals PCB-breakdown proceeds in a similar way but that small cetaceans had a significantly lower ability than the others. Comparison of two enzyme systems based on a "metabolic index" (which is based on tissue ratios of certain PCB compounds) indicates, again, that the important CYP2B enzyme subfamily is missing in cetaceans. The Japanese researchers noted that "... small cetaceans may be vulnerable to the reproductive effects of these chemicals because of a deficiency of PB-type enzymes and low activity of MC-type enzymes also" (Tanabe et al., 1988). Direct examination of the specific profile of liver microsomal cytochrome P-450 in dolphins and whales, not surprisingly, also reveals very low activity of two parts of the enzyme system (Watanabe et al., 1989).

In addition to their detoxification function, the P-450 enzymes are also important in the synthesis and/or metabolism of endobiotics, such as fatty acids, steroids and prostaglandins. The relationship between their presence, concentration or absence and other aspects of animal's metabolism needs further investigation. Every investigation conducted so far, however, indicates that seals and even more so cetaceans, are especially vulnerable to the accumulation and toxic effects of halogenated hydrocarbons.

4d. Other evidence from wild marine mammals

In seals, studies on reproductive failure in the Baltic and Wadden Seas and in Liverpool Bay in the UK, have been correlated in the field and in the laboratory with the presence of PCBs and DDT (Reijnders, 1980, 1986; Baker, 1989); reduced blood levels of retinol (a precursor for vitamin A) and thyroid hormones in seals fed heavily PCB-contaminated fish caught in the Wadden Sea (Brouwer et al., 1989); and various deformities in seals reported from the highly-polluted Baltic (Zakharov and Yablokov, 1990). The most dramatic increase in DDT and PCB levels in the Baltic occurred after 1955. Skulls from seals born before 1940 were compared with those from animals born after 1960. Thirty-seven characters were studied and the more recent sample had increased levels of asymmetry in almost every one.

Examples of reproductive failures in marine mammal populations linked to organochlorine contamination have recently been reviewed (Addison,

1989) and are summarised in table 7.

Whilst a detailed chain of events showing how whole organism responses to PCBs has not been established (Addison, 1989), changes in circulatory hormone levels in harbour seals, experimentally exposed to high PCBs by their diet, have now been found (Brouwer et al., 1989). So the mysterious mechanisms between causes and effects (pollution and reproductive failure) are now being determined.

Considering mass mortalities in marine mammals themselves as evidence of pollution's impact on these animals is perhaps a circular argument. Many different factors have been put forward as significant contributors to epizootics (see table 9). In any disease event a complex series of factors will interact controlling the expression of the disease, including its severity and spread. Some factors may be considered "natural", such as the density of the afflicted population; others "unnatural" such as pollution-induced immuno-suppression. Examination of the list of recent mortalities (table 9) shows that of the five which have occurred since 1987, at least four (and perhaps the other one in Siberia as well) occurred along highly contaminated coastlines.

4e. Other effects reported in wild dolphins populations

i. Japanese research

A study made on striped dolphins showed that abnormalities in lipid metabolism (fatty livers) occurred in individuals with relatively high PCB and DDT burdens (Kawai, et al, 1988). Other Japanese research has found a direct relationship between increasing residue levels of PCBs and DDE in the blubber of oceanic Dall's porpoises and decreasing blood testosterone concentrations (Subramanian et al., 1987). The results indicated that even in oceanic species, present levels of organochlorines are causing imbalance in sex hormones and consequently reproductive abnormalities. The levels of DDE in the blubber correlated with this effect were less than 20 ppm - which compared to existing levels in control species is quite low.

ii. South African research

Cockcroft et al. (1989) have recently made a major investigation into organochlorines in 105 bottlenose dolphins from the coastal waters of South Africa. They found the same pattern that had been reported from other small cetaceans; organochlorine burdens are closely related to age until animals reach sexual maturity, thereafter there is a marked drop in concentrations in females whereas males continue to accumulate these compounds throughout their lives. They also calculated the quantities and period of the passage of organochlorines to calves (table 10). As shown, rapid transfer of high concentrations of organochlorines occurs in mother's milk. The speed of transfer may constitute a greater risk than the levels themselves might indicate. Dolphin calves may also be more susceptible to PCB toxicity at this stage of their lives, as they may have comparatively poor immune response. The researchers noted that the contaminants could actually lead to "a possible mortality of first born calves and a concomitant reduction of male reproductive efficiency" which would probably vary

according to the organochlorine burden of animals in each area. As in other mammals, organochlorine compounds are also transferred via the placenta to developing dolphin foetuses (Tanabe et al., 1982) and enzyme activities are reported to be even lower in cetacean foetuses than older animals (Watanabe et al., 1989). It should be noted that the concentrations found in southern hemisphere animals are likely to be far lower than those in cetaceans living in more polluted waters.

iii. The Cardigan Bay Dolphin Calf

The significance of contaminants in European bottlenose dolphin calves was recently illustrated in Cardigan Bay, Wales (WWF, 1988; Morris et al., 1989). During a detailed ecological study of a resident group of bottlenose dolphins in the Bay, in August 1987, a calf was born to one of the adult female members. The development of the calf seemed normal but in June 1988 it was found floating offshore dead. The corpse was examined and blubber thickness seemed normal and hepatitis was the immediate cause of death. It was not possible, however, to determine if this was of toxic or viral origin. Extremely high levels of organochlorine compounds were found in the fat-rich tissues of the calf, lending support to the theory that pollution was to blame (i.e. in the blubber 290 ppm wet weight total PCBs, 150 ppm wet weight total DDT). Levels were comparable to those reported in the very highly contaminated Belugas of the St. Lawrence Estuary, Canada, and seals in the Dutch Wadden Sea. The researchers noted that they had not anticipated finding such high concentrations (although there are apparently no previous studies of contaminants in dolphins in Cardigan Bay or the adjacent waters of the Irish Sea). The calf had, throughout its brief life, remained within a home range of not more than 200 square miles and would still have been obtaining part of its nutrients from its mother's milk - the most likely source of its high contamination burden.

iv. Research on St. Lawrence Belugas

Some of the most acutely polluted cetaceans in the world are the St. Lawrence beluga whales, Delphinapterus leucas (Pallas, 1776). These form a small population restricted to parts of the Gulf of St. Lawrence and the St. Lawrence estuary (Sergeant and Hoek, 1988). Their numbers have decreased dramatically during the present century. Initially, heavy hunting reduced the population from about 5,000 to several hundred. This pressure lessened appreciably in the 1960s and the beluga has been fully protected since 1979 but the population has never recovered. Researchers have suggested that "organochlorine contamination should be considered as a prime cause for the low recruitment observed in this population" (Martineau et al., 1987).

The reproduction rate of the St. Lawrence belugas is half, or less than half, of that estimated for Arctic animals (Sergeant and Hoek, 1988). Concentration of PCBs in the beluga adipose tissues range from 5.7-576 ppm and, not surprisingly, the whales have also been found to be suffering from a range of pathological abnormalities including bladder cancer (Martineau et al, 1985) which is rare in animals.

5. CONCLUSIONS

The evidence presented in this paper is summarised in table 11. What it shows is that certain ubiquitous pollutants - which preferentially accumulate in marine mammal tissues - are known commonly to produce a range of detrimental effects in a variety of species. There is no reason to hope that these effects will not also be induced in marine mammals. Indeed, consideration of their biology and metabolism leads to the belief that seals and cetaceans are an especially vulnerable part of this planet's wildlife.

Diseased animals are clearly affected by many stresses in addition to those imposed directly by a primary infection (such as a morbillivirus). Many factors - for example their nutritional state prior to infection - will have a significant role in determining both an animal's initial susceptibility to infection and its subsequent ability to recover once infected. Any dysfunction in an animal's immune system will make it more vulnerable to infection and less able to recover. Toxic substances released into the body of a sick animal may contribute to the overwhelming of vital organs (which may already be damaged by infection). The liver, for example, would be important in removing from an infected animal's system the toxic products of invading bacteria. Elevated levels of PCBs would be expected to interfere with critical liver functions.

At the time of writing (July, 1991) it seems most unlikely that the Mediterranean striped dolphin mortality can be simply explained in terms of a morbillivirus infection. Furthermore, the earlier US east coast bottlenose dolphin die-off has just been brought under the spot-light again in a recently released paper. This new investigation into chemical residues in dolphins involved in the die-off, conducted by the U.S. Environmental Protection Agency, far from supporting the natural "brevetoxin" theory, concludes the following:

"The data reported here show that the U.S. mid-Atlantic bottlenose dolphins are contaminated with high concentrations of PCBs and PBBs [polybrominated biphenyls], strong immunosuppressive agents, as well as other toxic pesticides....[and]....yet unidentified polychlorinated and polybrominated chemicals. Although the impact of these contaminants is not fully known, their role as causative agents in this recent mass mortality must be considered...." (Kuehl et al., 1991).
In fact an increase in disease and even severe epizootics could actually have been predicted from the data available a few years ago. This is exactly what has happened. Now what is being forecast is the possible pollution-induced extinction of marine mammals in coastal waters (Cummins, 1988; Marquenie, 1990). It is time to act to prevent this.

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Figure 1 Events which may have led to the death of several thousand striped dolphins in the Mediterranean in 1990-91.

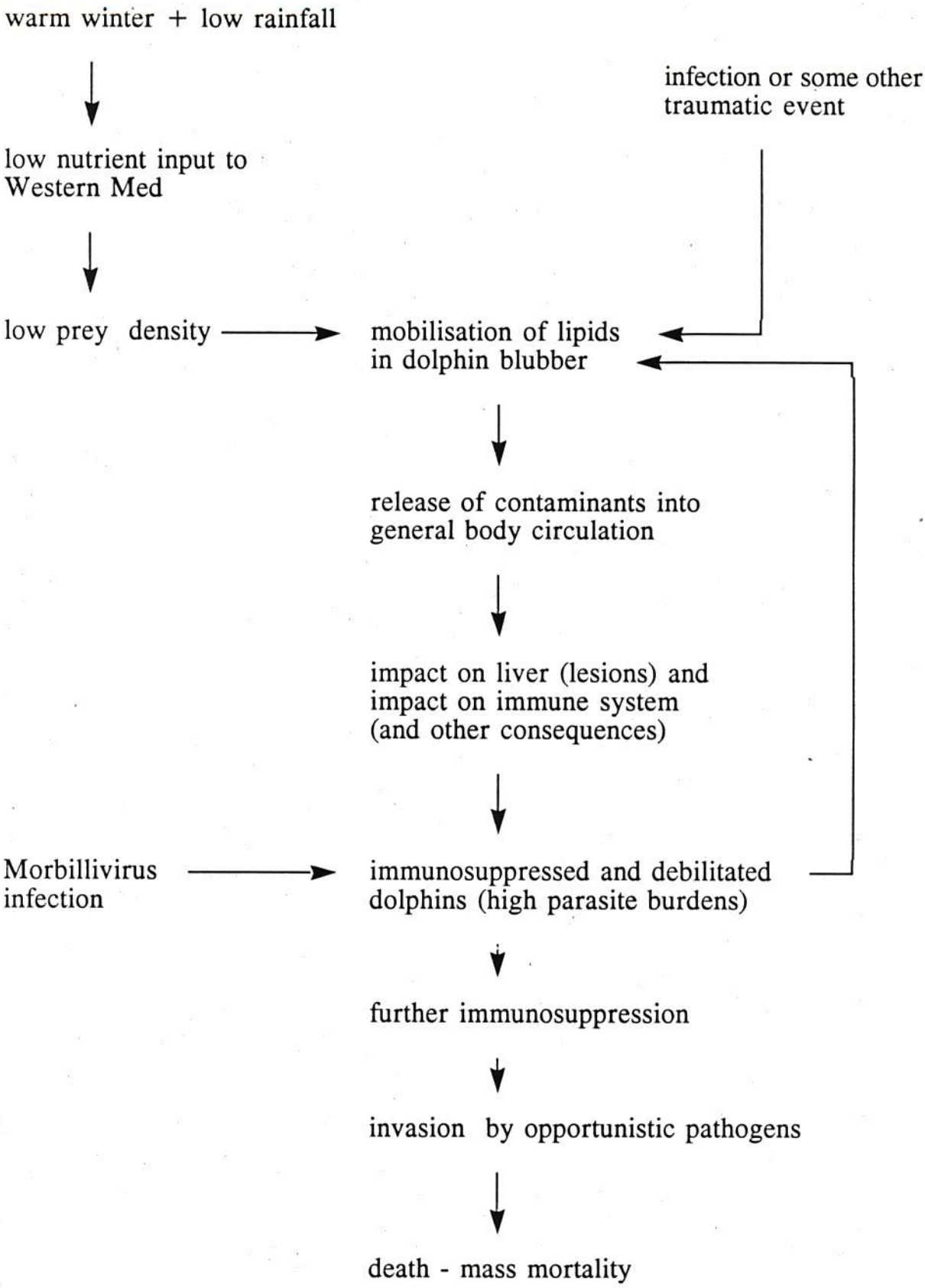

Table 1 **Population trends post-epizootic (Sources Harwood, 1989; 1990; NERC, 1989)**

West Scotland, Skye to Mull	+ 3%
Orkney	+ 6%
East Scotland (Moray Firth)	- 15%
Norfolk Wash	- 48%

Table 2 **Contaminants in blubber samples from harbour seals which died during the seal epidemic of 1988-89.**

i) On the East coast of Scotland. (Concentration in ppm) (9 seals sampled)

Contaminants	Mean concentration
Mercury	0.03
DDE	1.37
PCBs	0.11

Age: 6 were classed as yearlings/pups
2 as "sub adults" and one is unknown

ii) From Norfolk (14 seals sampled)

Mercury	0.212
HCB	0.014
DDE	2.07
Dieldrin	0.031
PCBs	7.17
HCH	0.131
TDE	0.02
DDT	0.24

Age: All seals were classed as yearlings or pups except for one adult.

Another recent report, (Laws *et al.*, 1989) found the following in a similar group of young Norfolk harbour seals (10 seals sampled)

HCB	0.004
DDE	3.1
PCBs	23.0

(other organochlorines were also detected)

Table 3 **Problems with the "brevetoxin theory" (after Martineau, 1989)**

1. Evidence of brevetoxin intoxification is limited. Brevetoxin only detected in 8/17 carcasses and 3 fish (one inside the stomach of a dolphin).

2. All lesions found in the 240 carcasses examined were synonymous with PCB intoxification. Including some which are relatively specific for PCB toxicity.

3. Brevetoxin is not known to attack organs or cause lesions.

4. High concentrations of organochlorine contaminants were found in the dolphins.

5. PCBs are known to be immunosuppressive and brevetoxin has not been reported to have such an effect.

6. Dolphins have been exposed to algal toxins for thousands of years and are therefore likely to have metabolic pathways to degrade it.

7. Red tides are common in the Gulf of Mexico along with the same species of dolphins but no previous similar events have been reported.

Table 4 **Pathology of bottlenose dolphins from the 1988 US die-off and comparison with known effects of PCBs and brevetoxin (after Martineau, 1989).**

Symptom	Previously reported as inducible by	
	Brevetoxin	**PCBs**
Parakeratosis (thickening of superficial skin)	Unknown	Yes
Liver		
- severe hepatic lipidosis	Unknown	Yes
- single cell necrosis	Unknown	Yes
Immune system - lymphoid depletion	Little known (does not impair immunity in mice)	Yes

Basic known effects on mammals

Brevetoxin	**PCBs**
Potent neurotoxin	Potent immune suppressors
Haemolytic agent	(and see other tables)

Table 5 **A summary of PCB toxicity (reviewed in Safe, 1984)**

Type of Effect	Examples
Liver toxicity	- Liver function disturbed - Enzyme systems activated - Hormone metabolism altered
Immuno-toxicity	- Atrophy of lymphoid tissue - Increased disease susceptibility
Neurotoxicity	- Behavioural changes and "reduced intelligence"
Reproductive impairment	- Increased abortions - Reduced fertility - High embryonic mortality - Birth defects
Skin damage	- Chloracne
Cancer promotion	- Increased malignancies

Table 6 **Evidence of impact of organochlorines on the reproductive systems of mammalian species (information from Safe, 1984)**

i.	microsomal enzyme induction leading to hydroxylation of steroids such as oestrogens (various species)
ii.	various impacts on the female reproductive tract (rats)
iii.	prolonged oestrous cycle - decline in number of implanted ova (mice)
iv.	incurred foetal deaths (sows)
v.	embryonic resorption, abortion, stillbirth, irregular menstrual cycles (rhesus monkeys), (embryonic resorption also recorded in mink)
vi.	development of ovarian-like tissue in embryos (gulls)
vii.	induction of testosterone hydroxylase (rats)
viii.	duplication of DDT effects by oestrogen (chickens)

Table 7 **Examples of wildlife events thought to be associated with PCB or related compounds (after Gilbertson, 1989)**

Problem	Location and Species
Fish disease (various symptoms)	Irish Sea plaice, flounder and dab; US Dover sole, starry flounder, Atlantic tomcod, striped bass, English sole, Canadian cod, Swedish hag fish, Baltic salmon, flounder, herring. Riverine trout (Rhine), Great Lakes trout, coho salmon, white sucker and goldfish.
Bird disease (various symptoms - notably embryonic and developmental problems)	Great Lakes: Herring gulls, black-crowned night-heron, ring-billed gull, common tern, Caspian tern, Foster's tern. British peregrine falcons, sparrow hawks and golden eagles, American peregrine falcons, bald eagles and ospreys.
Mammal disease (freshwater only - reproductive failure and associated population decline)	US wild and ranched mink US otters

Table 8 **Reproductive failures in marine mammals linked to organochlorine contamination (Addison, 1989).**

	Locality and Species	Symptoms
1.	Californian Sea Lions	Premature births
2.	Baltic grey and ringed seals	Population decline Uterine occlusions
3.	Dutch Wadden Sea harbour seals	Population decline Reproductive failure
4.	Canadian (St. Lawrence) Beluga whales	Population decline Various pathological abnormalities Cancers

Table 9 **Factors, other than pollution, postulated as associated with marine mammal mass mortalities.**

Mortality Date	Location	Species	Approx. Size	Factor A	B	C	D
A SEALS AND OTHER PINNIPEDS							
1918	Iceland	Harbour seal	1,000	+	-	-	-
1955	Antarctic	Crabeater seal	3,000	+	+	-	-
1978	Bering Strait	Walrus	1,200	+	-	-	-
1979/80	New England	Harbour seal	500	+	+	-	-
1987/88	Siberia	Baikal seal	8,000	+?	-	-	+
1988	European	Harbour seal	18,000	+	+	-	+
B CETACEANS							
1987/88	US (E. coast)	Bottlenose dolphins	2,500	-	-	+	+
1990	US (Gulf of Mexico)	Bottlenose dolphins	300	-	-	-	-
1990/91	Mediterranean	Striped dolphins	7,000		?	-	-+

KEY
A = Mortality preceded by 3 months when air temperature higher than the ten year average.
B = Population known to be at high density.
C = Algal bloom associated.
D = Morbillivirus associated.

Data from Dietz *et al.*, 1989; Geraci, 1989; Lavigne and Schmitz, 1990 and Aguilar and Raga, 1991.

Table 10 **Organochlorine transfer in S. African dolphins (data from Cockcroft *et al.*, 1989)**

1. Almost 80% of the organochlorine burden of a female bottlenose dolphin is passed to her first born calf.

2. Assuming 20% concentration of organochlorines in mother's milk and calf consumption of four litres/day -

   a) Milk will contain 2.7 ppm PCBs
   2.25 ppm DDT
   0.08 ppm dieldrin

   b) Input to calf/day 10.8 mg/day PCBs
   9 mg/day DDT
   0.32 mg/day dieldrin

   (approx 4% of mother's total burden/day)
   (total load transfer occurs in seven weeks)

   c) Burdens in 2 month old calf
   = >30 ppm PCBs and DDT
   1 ppm dieldrin

[This is all based on mother's burden of only 13.5 ppm PCBs, 11.25 ppm DDT and 0.38 ppm dieldrin].

Table 11 **Pollution and Epizootics**

A Considerable evidence exists that organochlorines impact animals in a variety of ways - particularly affecting the immune and reproduction systems.

B Marine mammals accumulate such materials in their substantial fatty tissues and pass them in substantial quantities to their young.

C Marine mammals (particularly small cetaceans) seem to be even more vulnerable to organochlorines than terrestrial mammals.

D Sick marine mammals are likely to mobilise their lipid stores - thereby releasing extra quantities of organochlorines into their disease-stressed bodies.

E There is evidence of this (D) in the 1988 seal epidemic and the 1990 striped dolphin mortality and possibly the E. coast dolphin die-off.

F. The frequency of marine mammal die-offs seems to have increased in recent years and they appear to be centred along highly industrialised coasts.

G Levels of implicated substances (i.e. PCBs) are set to increase in coastal waters.