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| **From:** | Council |
| **To:** | Barbara Brenner; Barry Buchanan; Rud Browne; Satpal Sidhu; Timothy Ballew; Todd Donovan; Tyler Byrd |
| **Cc:** | Becky Boxx; Matt Aamot; Mark Personius |
| **Subject:** | FW: Please extend moratorium at Cherry Point |
| **Date:** | Wednesday, August 08, 2018 2:56:58 PM |
| **Attachments:** | Johnston - Comment on Comp. Plan.pdf |

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**From:** Robert M Johnston [mailto:Robert.Johnston@nau.edu]
**Sent:** Wednesday, August 08, 2018 2:51 PM
**To:** Council
**Cc:** Eddy Ury
**Subject:** Please extend moratorium at Cherry Point

August 8, 2018

Dear Whatcom County Council Members:

I'm writing to urge you to extend the moratorium on fossil fuel projects at Cherry Point. I am unable to attend tonight's county council meeting, or I would come and speak in person. I have done this in the past when the meetings have dealt with fossil fuels and Cherry Point. I have also written to you, and in February of 2016, in particular, I provided you with a document outlining the scientific background on the negative impacts for humans and the environment of some of the components of coal and oil. For your reference, since you may not still have it close at hand, I've attached that document to this e-mail. The information and the argument remain as valid and as current today as they were two years ago.

Please maintain the moratorium until the public has had a chance to review and comment on a more permanent set of guidelines for fossil fuel facilities at Cherry Point. Fossil fuel companies should not be allowed to apply for permits before then and gain vested rights to develop projects under outdated regulations. This would open Cherry Point to risky projects that could cost Whatcom County and its citizens dearly in the future. Please vote to extend.

Thank you for your attention.

Sincerely yours,

Robert Johnston
816 14th St.
Bellingham, WA 98225
(360) 961-6468

cc. Eddy Ury, Re-Sources, Bellingham, WA

**February 6, 2016**

**To: Whatcom County Council: Barry Buchanan, Chair; Barbara Brenner; Rud Browne; Todd Donovan; Ken Mann; Satpal Sidhu; Carl Weimer; 311 Grand Ave. Suite 105, Bellingham, WA 98225.**

**Fr: Robert Johnston, 816 14th Street, Bellingham, WA 98225.**

**Re: Comment on Comprehensive Plan In Support of Lummi Nation's Request for Moratorium on New Coal and Oil Facilities at Cherry Point.**

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## I. Introduction & Abstract:

I am a resident of Bellingham, a husband, father, fisherman, skier, backpacker, and retired college professor. I care about my neighbors, my community, and the world we live in. This includes both the economic activity that provides us with food and shelter and the environment that gives us, among other important things, the water we drink and the air we breathe.

I've prepared this comment, because I care about our community and because I'm concerned about the chances for a healthy, safe, and prosperous future that we'll pass along to our children, to their children, and to future generations. I want them all to have clean air and water, good jobs, and a prosperous future.

My comment bears specifically on one aspect of the Comprehensive Plan that I think is very important. I understand that the Lummi Nation has asked for a moratorium on the development of any new coal and oil export facilities at Cherry Point. I'm asking that you include this in the Comprehensive Plan. I think the Lummis have wisdom on their side --wisdom about the right path to follow toward a healthy and prosperous future.

Coal and oil have fueled amazing advances in the past couple of centuries, but this has come with a stiff price. Part of the bill is now coming due in the form of what we'll have to do

to cope with global climate change. Another part of the cost comes in the form of their impact on local and regional environments. This second "external cost" is the focus of my comment.

Coal and oil have some very nasty, toxic ingredients, and whatever the industry may say, experience has shown that their handling, storage, and transport involves a certain predictable, even guaranteed, amount of spillage into surrounding environments. Coal has heavy metals – lead, mercury, cadmium, selenium, arsenic --, and oil has complex, toxic chemicals, such as polycyclic aromatic hydrocarbons – all of which are highly poisonous to living creatures in the sea, on the land, and including humans. These substances can enter the soil, the water, the sediments, and can be absorbed by organisms, which, if they don't die from the exposure, can concentrate them and pass them along up the food chain. The results can be long-term, chronic contamination of the local environment and beyond. This is the exact opposite of the kind of legacy I think we should be leaving for our children and for generations to come.

Fossil fuels will inevitably stop being our principle sources of energy. The real costs are just too high. Other safer, cleaner, more sustainable alternatives will take their place, like wind, solar, and geothermal. That's the path toward long-term prosperity. That's where we should be investing our resources and building Whatcom County's future.

## II. Explanation and Scientific Background: Coal, Oil & Cherry Point.

At this point, since I am not a scientist, I will incorporate information provided to me by my colleague, Dr. Paul F. Torrence. Dr. Torrence is a research chemist with expertise in medical chemistry and biochemistry. He worked for many years as a researcher and a section leader for research projects at the National Institutes of Health in Washington, D.C., and afterward as a professor of chemistry and chair of the Chemistry Department at Northern Arizona University in Flagstaff, Arizona, where I also taught. I described the circumstances at Cherry Point to him and asked him what he could tell me about coal and oil and their effect on aquatic organisms and environments. He has provided the scientific information, the references, and much of the writing in the following commentary. We've crafted the section to be accessible to non-experts, but the studies on which it is based are top-quality, professional research publications in refereed scientific journals. We've included excerpts from these publications in the "Numbered References" section; these are noted in the text in the background statements. These and the other sources listed in the "Bibliography" section are intended to help show the extent and quality of current research world wide on the problems of coal and oil toxicity.

**The Trouble with Coal:** Coal contains numerous heavy metals that are toxic to humans and other creatures. These include, among others, lead, mercury, nickel, cadmium, selenium, manganese, antimony, and arsenic. Coal also contains the radioactive elements thorium and strontium.

Transporting coal by train or truck and creating stockpiles for shipping sends coal dust into the air, which diminishes air quality and subjects nearby populations to dust inhalation. Health effects from exposure to coal dust include increased asthma, wheezing and cough, especially in children and the elderly. A wide range of serious health problems accompanies exposure to the heavy metals present in coal dust. Coal dust may also be carcinogenic due to the presence of polycyclic aromatic hydrocarbons (PAH).

Each of these metals as well as the spectrum of carcinogenic polycyclic hydrocarbons presents hazards to many species including humans. As may be expected, there is more scientific literature on the effects of these contaminants on humans than on other species; however, by the precautionary principle, it is reasonable to extrapolate data from other organisms to humans and vice versa.

In what follows, we draw particular attention to just one of the toxic heavy metals in coal -- cadmium – and its toxicity for aquatic life in general and shellfish in particular. The dangers from some of the other heavy metals in coal and coal dust, such as lead, mercury, and arsenic are more widely known. Lead in the water system of Flint, Michigan has captured headlines recently. The case offers an example of lead's toxicity and of the need for local governments to protect public health. Cadmium is found in coal and coal dust in smaller quantities than lead and mercury, but its toxicity is such that it may actually pose an even greater danger. Similarly, public attention might more easily focus on larger animals, such as salmon and marine mammals. But oysters, crabs, clams, and other shellfish have significant commercial and recreational value in Whatcom County, in addition to their intrinsic value as part of Puget Sound's unique marine environment. This focus should not diminish concern for all potentially affected species, aquatic and terrestrial, since the ecosystems involved must be considered as integrated systems and shellfish comprise an important part of the food chain. Nor should it diminish concern for public health, since this too depends in myriad ways on environmental factors. Indeed, many of the scientific studies listed in the Numbered References and the Bibliography address other heavy metals along with cadmium.

**Scientific Background on Cadmium in Coal (by P. F. Torrence):** Based upon a literature examination, we believe we must be wary of additional cadmium introduction to natural ecosystems because of established adverse effects on both humans and wildlife. That is not to imply that the other metals and chemicals should not be matters of concern. For example, mercury is certainly a huge hazard also.

Several facts provide a platform for understanding the hazards of cadmium contamination of the environment. First, cadmium occurs in many soils and rocks. It is easily mobilized by various anthropogenic activities such as agriculture, forestry operations and mining. There can, therefore, be levels of cadmium present in soils, waters and sediments that provide a "background" of cadmium that may allow little leeway for added accumulation before toxic concentrations result (e.g., Numbered Reference 1).

Second, certain organisms, such as shellfish, are able to actively sequester cadmium in their bodies due to the presence of a metal-binding protein. This "bioaccumulation" can greatly magnify the concentrations of cadmium in the environment by astounding factors as high as 40,000-fold. As a result, any organism that consumes these shellfish will obtain a dose of cadmium that is much greater than ambient environment levels (e.g., Numbered References 2, 3, 4, 5, 6, 7, 8, 9, & 10).

Third, the human kidney also accumulates cadmium, leading to renal toxicity and, if not controlled, kidney failure. Women, especially those with low iron levels, as well as smokers of both sexes, are particularly at risk from cadmium toxicity. This has become a serious issue in European countries. Cadmium burdens have also been linked to osteoporosis and breast cancer (e.g., Numbered References 11, 12, 13, 14, & 15).

Fourth, cadmium is toxic at part-per-billion concentrations to shellfish and is even more toxic to their juvenile forms (e.g., Numbered References 7 & 17).

Fifth, organisms at higher trophic levels that consume shellfish (as an example) can suffer adverse consequences from consumption of cadmium-containing tissues. This has been well documented for avian species (e.g., Numbered References 16 & 18).

Based on these facts from the scientific literature, we can predict that increased environmental cadmium burdens may cause: 1) increase in shellfish mortality; 2) decrease in shellfish reproduction and population levels; 3) increased cadmium burdens in shellfish tissue with resultant accumulation in wildlife with consequent adverse effects on terrestrial, freshwater, and marine wildlife populations; 4) increased cadmium burdens in shellfish with the potential of shutdown of recreational and commercial harvests and/or increased human body burdens with resultant kidney damage and other pathogenic effects.
Thus the externalized cost of coal has to include (among a panoply of other costs) the negative economic, ecological and human health effects (including healthcare costs) of cadmium toxicities. P. F. T.

As mentioned earlier, many of the numbered references report on research on other heavy metals, in addition to cadmium. Lead and mercury toxicity perhaps need no additional explanation. Among the other heavy metals in coal, selenium deserves further emphasis, since it may be as harmful for wildlife as cadmium. For example, The New York Times ("Mutated Trout Raise New Concerns Near Mine Sites," Leslie Kaufman, February 22, 2012), reports that selenium contamination of just a few parts per billion can cause lethal deformities in a high percentage of the offspring of fish and also waterfowl. A simple Google search for "Selenium Toxicity in Fish and Wildlife" turns up scores of articles by researchers worldwide, including the US Fish and Wildlife Service, the US Bureau of Reclamation, and others.

**Oil and Cherry Point.** Crude oil spills are visually ugly, but the harmful effects remain long after the black and brown, tarry mess has been cleaned up or made to disappear. The denser components of crude oil tend to sink in water, a result that is intensified when surfactants are used to disperse oil on the water's surface. While this removes the oil from view, it can increase rather than decrease the environmental damage. In fact, oil spills can over time become a source of persistent, chronic contamination in the food web. Not only can organisms such as shellfish bio-accumulate toxins, since they filter large quantities of water, they can also bio-magnify their concentration. Predators (both animal and human) who consume these organisms receive concentrations of toxins that can be thousands of times greater than the concentration found in the surrounding environment. Seabirds that consume contaminated shellfish, for example, are especially vulnerable. The concentration of toxins increases, moreover, at each successive level up the food chain.

One example of this dynamic are polycyclic aromatic hydrocarbons (PAHs), which are contained in petroleum products, including ship bunker fuel and crude oil, and which oil spills deliver into the environment. PAHs are associated with cancer, depressed immune systems, neurotoxicity, and fetal mutation in both aquatic and terrestrial creatures and in humans. In aquatic environments, PAHs can remain in sediments indefinitely and affect organisms at all trophic levels. Organisms that do not metabolize PAHs, if they survive their initial exposure to spilled oil, may bio-accumulate them and pass them along to their predators. Organisms that do metabolize PAHs, larger marine species especially, suffer the toxic effects of the carcinogenic by-products from the metabolism process. We must be concerned not only for the aquatic

organisms that inhabit the area near Cherry Point but also for the organisms that would travel up and down the coast carrying the toxins they've taken on. For example, salmon that consume contaminated forage fish could travel up and down the coast where various predators including seals, orcas and humans would consume them.

**Scientific Background on Polycyclic aromatic hydrocarbons (PAHs) (by P. F. Torrence):** PAHs are ubiquitous in the marine environment, occurring at their highest environmental concentrations around urban centers. While they can occur naturally, the highest concentrations are mainly from human activities, and the primary sources are combustion products and petroleum.

PAHs have been shown to bioaccumulate in various organisms including those resident in terrestrial, freshwater, and marine environments. The bioaccumulation and biomagnification values are impressive indeed and constitute a threat to humans and wildlife (e. g., References 19, 32 & 33).

The consumption of PAH– laden prey by higher trophic organisms such as seals can not only cause cancers but can also depress the immune system thereby making viral or other pathogen induced outbreaks more common and placing the victim creatures at higher risk of mortality. It can also be postulated that morbid prey such as seals would be more easily captured by predators, thereby exposing the latter to a greater potential disease burden. (e. g., References 20, 21, & 22).

PAHs can also cause extreme ectopic neural tissue growth, resulting in neonate harbor seal death (Reference 23).

In complete agreement with the predicted bioaccumulation and biomagnification of PAHs (and other persistent toxic anthropogenic chemicals) in high trophic level predators, shockingly high levels of PAHs have been documented in Orcas (e. g., Reference 24).

Studies suggest that PAHs, in contrast with PCBs and OCPs, are more quickly metabolized in marine mammals than in other species like molluscs, oysters and fish. This is an indication of a serious hazard to these animals. The metabolism of PAHs (like bemzo[a]pyrene) involves conversion to hydroxylated derivative. This metabolic process involves the intermediate formation of labile epoxides, which react with DNA, thereby causing documented carcinogenesis and/or mutation. A similar metabolism occurs in humans. The more PAHs ingested, the more carcinogenic intermediates formed, and the greater the risk of cancers.

It follows from the forgoing, that total lifetime exposure to PAHs will be drastically underrepresented by measurements of static PAH levels. It is the metabolism that will be lethal. PAHs may thus be considered as perfect toxic agents to cause diseases (cancers) in marine mammals. As they are accumulated at increasing trophic levels in selected prey species, due to relative metabolism, yet are delivered/consumed by/to species at high trophic levels. And thence metabolized to potent carcinogens

It then follows directly that top predators, such as orcas, would also be victims of PAHs. In the case of orcas, for instance, a true risk level cannot be extrapolated from extant body burdens of PAHs.

The biochemical basis of mammalian carcinogenicity is shown in the diagram in Reference 27, with oxidative metabolism to the ultimate epoxide diol carcinogens that then tract with DNA. The polycyclic aromatic hydrocarbons are biochemically inert, and require metabolism to exert their biologic effects (References 25 & 26). This is a multistep process, it involves the following: initial epoxidation (cytochrome P450, CYP1A1 is an inducible isoform),

hydration of the epoxide (epoxide hydrolase), and subsequent epoxidation across the olefinic bond. The result is the ultimate carcinogenic metabolite, a diol epoxide. It is this that attacks DNA and can initiate cancer (References 27 & 28).

The foregoing discussion and publications make several things clear:

First: The data provided here addresses only the marine environment but concerns equally apply to all to freshwater as well as terrestrial environments including those in habited by avian species.

Second: It is clear that a growing burden of toxic and carcinogenic PAH is carried by a number of species and that as trophic level increases this burden ramps up also. We have shown by the data presented that both seals and orcas are victims of PAH pollution. The immune systems of these animals are being compromised and this alone leads to a greater risk of infection and disease as well as cancer. Moreover it has been abundantly demonstrated that PAHs are a cause of human cancer and cancer in other mammalian species. There is no reason to believe that orcas and seals are any different. They are our close relatives. The simple extrapolation of the human situation to these creatures is a simple deduction that requires no further rationalization. There can be no rationalization, however, that would permit any increased burden of PAH to be added to the environment. Such action is not only counter to the best interests of wildlife it is also counter to the best interests of humans.

Third. Based upon the above discussion and the cited references, it is not relevant to simply use body burdens of toxic and carcinogenic PAH to indicate exposure to these chemicals. We have clearly shown that mammals can effectively metabolize such PAHs, thereby leading to a false estimation of their exposure (Reference 31). To the contrary we have shown that such metabolism itself is the gateway to the ultimate carcinogenic compounds that react with the DNA thereby triggering cancerous growth. It is naïve to expect that in mammals body burden of PAH is related to lifetime exposure, which is after all the appropriate measure of cancer-causing potential. P. F. T.

## III. Conclusions.

**Why Take the Risk?:** The scientific research on the effects of heavy metals and PAHs on humans and other creatures, together with reason and common sense should warn us that coal, oil, and Cherry Point are a bad combination. Experience and common sense also tell us that the storage, handling, and transport of coal and oil results in leakage, spills, and dispersal into the environment. Coal is lost from train cars along rail routes where it is transported. The winds at Cherry Point –frequent year-round, and gale-force in the winter – would mean that storage and handling of coal would result in dispersal of coal dust for considerable distances. Oil spills from leakage and derailments along rail routes pose various hazards, some of them catastrophic. Storage, loading, and transport of oil by ship from Cherry Point also carries similar risks.

The *BP Dock Draft Environmental Impact Statement* for the second pier at the BP refinery (BP-DEIS), indicates that spills at that site have occurred with some regularity and can reasonably be expected to continue (BP-DEIS 6.4.1; 6.3.1; 5.8.1). In 1972, an equipment failure released 21,000 gallons of crude oil. In 1988, 400 gallons of jet fuel were spilled. Such spills, even if they occur once in 50 or 100 years are extremely significant if they have the potential to contribute to the extinction of a unique population, such as the Cherry Point herring. The BP-DEIS explains that BP has reported a decreasing number of spills with decreasing volume per

spill since that time (BP-DEIS 5.8). At the same time, an average volume of 43 gallons spilled per year (the current average according to BP-DEIS Table 5-37) adds up: 430 in ten years, 860 in twenty, 2,150 in fifty. Although this seems a slow trickle in comparison to larger spills, this amount can be significant also if it remains in the sediments or is absorbed by marine species and passed along to other creatures in the food web. Alone, or in combination with similar amounts from other facilities, it has the potential to cause significant harm (e. g., References 29 & 30).

Catastrophic oil spills remain a possibility as well, especially with increasing vessel traffic in the area and the region. The risk analysis in the BP-DEIS raises this possibility. For example, the *George Washington University Vessel Traffic Risk Analysis* (GWU VTRA) Simulation Calibration for Accidents and Oil Outflow posits a potential of some 37,000 gallons of oil outflow annually due to major oil spills (BP-DEIS 5.3.5, Table 5-2). The BP-DEIS notes that the Exxon Valdez oil spill in Alaska killed 22 orcas in two pods, and now, 25 years later, one pod is recovering and one, instead, is headed for extinction. While risk of such a catastrophe in Puget Sound has been reduced with increased regulation, and because tankers now have double hulls, it is also rising again with increasing vessel traffic. Unfortunately, the oil industry has an established history of spills. Given enough time, the odds point to the likelihood of more spills. And the more species that are added to the state and federal lists of threatened and endangered species, the greater the risk that a spill, large or small, could deal one or more of them a blow from which they could not recover.

**The Myth of "Mitigation."** We believe the concept of "mitigation" for these impacts is misguided. Many potentially affected species are already under stress and suffering declining populations due to various forms of toxic pollution and environmental degradation. These include species familiar to people in Whatcom County-- oysters, Cherry Point herring, orca whales, salmon (Chinook, chum, sockeye, and coho in different locations), as well dozens more on the state and federal threatened and endangered species lists: American white pelicans, brown pelicans, bull trout, steelhead (various locations), rockfish (several varieties), fishers, sea turtles (green, leatherback, loggerhead), whales (blue, fin, humpback, orca, North Pacific right, sei, and sperm), sandhill cranes, and sea otters, to name some. To risk further declines and potential extinction of these species by allowing projects which will surely result in increased introduction of toxic substances into the environment is simply irresponsible. The same applies to negative impacts on human health.

The only reasonable approach is to speak of *guaranteed prevention.* Given the current, available means of transport, storage, and handling of coal and oil and the established record of this activity, we believe that guaranteed prevention is not honestly possible. Any company or its representative that would offer such a guarantee could not possibly be doing so in good faith. The true costs involved in transporting, storing, handling, and using coal and oil *safely and cleanly*, if this were even possible, would be simply too great for there to be any profit in it. We include in the true cost the so-called "external costs," which typically are borne by communities, the general public, and the environment rather than the coal and oil industries. The potential cost to human health and the environment from the accumulation and toxicity of heavy metals, PAHs, and other toxic substances, must be included in the assessment. If it is, we believe it will be obvious there is no satisfactory way the effects can be "mitigated."

One final consideration: The so-called "sixth extinction," which scientists believe may be precipitated by climate change and global warming, may spell the end of up to one-third of the species now existing world-wide. It would be highly irresponsible to subject our own region's

already threatened and endangered species to an additional obstacle to their survival in the form of toxic contamination from additional coal and oil facilities at Cherry Point.

**Better Alternatives:** The Lummi's request that Whatcom County's Comprehensive Plan include a moratorium on any new coal or oil facilities at Cherry Point seems both wise and prudent. In all senses -- human health, the environment, the county's economy – their request points toward a better path. Whatcom County should stay out of the fossil fuels export business. The real costs today and in the future are simply too great. We should not further commit ourselves, our children, and the generations that follow us to industries that will leave a legacy of contamination and that must necessarily be replaced if the world is to survive human-caused climate change. There are many alternatives to building fossil-fuel export facilities that will work much better to help the local and regional economies, create good jobs, protect public health, and preserve the unique environments of Cherry Point, the Salish Sea, and inland along the rail routes that would deliver coal and oil to Washington. We should instead stay on course with existing initiatives to lessen rather than increase our use of fossil fuels, to limit pollution at Cherry Point and along our coast, to protect public health, to protect existing jobs associated with shellfish harvesting and fishing, and to support research and development of wind, solar, geothermal and other safe, clean, alternative forms of energy. Thank you.

## IV. Numbered References,

**Reference 1. "Critical Soil Concentrations of Cadmium, Lead, and Mercury in View of Health Effects on Humans and Animals," deVries, et. al.: "**Assessment of the risk of elevated soil metal concentrations requires appropriate critical limits for metal concentrations in soil in view of ecological and human toxicological risks. This chapter presents an overview of methodologies to derive critical total metal concentrations in soils for Cd, Pb, and Hg as relevant to health effects on animals and humans, taking into account the effect of soil properties. The approach is based on the use of nonlinear relationships for metals in soil, soil solution, plants, and soil invertebrates, including soil properties that affect metal availability in soil. Results indicate that the impact of soil properties on critical soil metal concentrations is mainly relevant for Cd because of significant soil-plant, soil-solution, and soil-worm relationships. Critical Cd levels in soil thus derived are sometimes lower than those related to ecotoxicological impacts on soil organisms/processes and plants, which is especially true for critical soil Cd concentrations in view of food quality criteria for wheat, drinking water quality, and acceptable daily intakes of worm-eating birds and mammals."

**Reference 2.** "**Exploring Spatial and Temporal Variations of Cadmium Concentrations in Pacific Oysters from British Columbia," Feng, CX., et. al. (Abstract):** "Oysters from the Pacific Northwest coast of British Columbia, Canada, contain high levels of cadmium, in some cases exceeding some international food safety guidelines."

**Reference 3. "Cadmium in Shellfish: The British Columbia, Canada Experience…," Bendell LI. (Abstract): "**Over 10 years ago, research scientists in the federal department of Fisheries and Oceans Canada (DFO) were alerted to the presence of high levels of cadmium, a

toxic metal, in the Pacific oyster (Crassostrea gigas) cultured in British Columbia (BC), Canada waters. This mini-review summarizes the most recent published studies on levels of cadmium in shellfish from the Pacific Northwest (BC and Washington State)."

**Reference 4. "Geochemical Survey and Metal Bioaccumulation of Three Bivalve Species," Baudrimont, M. et. al. (Abstract):** "A 15-month experiment combining a geochemical survey of Cd, Cu, Zn and Hg with a bioaccumulation study for three filter-feeding bivalve species (oysters, Crassostrea gigas; cockles, Cerastoderma edule; and clams, Ruditapes philippinarum) was conducted in a breeding basin of the Nord Medoc salt marshes connected to the Gironde estuary, which is affected by historic polymetallic pollution…. Although Cd bioaccumulation of oysters was lower in the basin than in the estuary during the same period (27,000 ng g(-1), dry weight and 40,000 ng g(-1), respectively) these values are largely above the new human consumption safety level (5000 ng g(-1), dw; European Community, 2002)."

**Reference 5. "Cadmium Toxicity Among Wildlife in the Colorado Rocky Mountains," Larison, James R., et. al. (Abstract): "**Our results suggest that cadmium toxicity may be more common among natural populations of vertebrates than has been appreciated to date and that cadmium toxicity may often go undetected or unrecognized. In addition, our research shows that ingestion of even trace quantities of cadmium can influence not only the physiology and health of individual organisms, but also the demographics and the distribution of species."

**Reference 6. "Cadmium Toxicity to Three Species of Estuarine Invertebrates," Pesch, Gerald and Nelson E. Stwart (Abstract):** "Three species of estuarine invertebrates, *Palaemonetes pugio* (grass shrimp), *Pagurus longicarpus* (hermit crab) and *Argopecten irradians* (bay scallop), were exposed to Cd in flowing seawater at concentrations of 0·06, 0·12, 0·25, 0·5 and 1·0 mg/litre. Incipient $LC_{50}$ values of 0·53 and 0·07 mg/litre were estimated for bay scallop and hermit crab, respectively. The toxicity curve for grass shrimp had not stabilised, but the incipient $LC_{50}$ value was estimated to fall within a range of 0·2 to 0·3 mg/litre. Short-term response, as measured by time to 50% mortality at the highest Cd concentration, was 10, 21 and 23 days for the bay scallop, hermit crab and grass shrimp, respectively. Scallop growth was inhibited at all exposure concentrations with a measured 42-day $EC_{50}$ value of 0·078 mg/litre Cd. Byssal thread detachment precedes death in bay scallops. An $EC_{50}$ value of 0·54 mg/litre Cd for byssal detachment was measured on day 8 of the bioassay before appreciable mortality. This compared favourably with the incipient $LC_{50}$ value of 0·53 mg/litre Cd. Cadmium accumulation occurred at all concentrations in bay scallop and grass shrimp."

**Reference 7.** "**Acute Toxicity of Copper, Cadmium, and Zinc to Larvae of the Crab *Paragrapus quadridentatus* (H. Milne Edwards), and Implications for Water Quality Criteria," M Ahsanullah and GH Arnott (Abstract):** Acute toxicity tests were carried out on the larvae of *P. quadridentatus* and 96-h LC50 values of 0.17, 0.49, and 1.23 mg/l were determined for copper, cadmium, and zinc respectively. Potency ratios of the three metals were as follows: Cu/Cd 3.1, Cu/Zn 7.2, and Cd/Zn 2.4. Larvae were found to be nine times more sensitive to zinc and at least 29 times more sensitive to cadmium than were adults. The larval 96-h $LC_{50}$ values multiplied by an application factor of 0.01 (as recommended in Victorian water quality criteria) results in derived 'safe' concentrations, which in the case of copper and zinc are

below the stated 'minimal risk concentrations' of 10 and 20 µg/I respectively. In view of the known greater sensitivity of larvae of many taxa to heavy metal toxicity, the validity of using the same application factor for both adult and larval stages is questioned."

**Reference 8. "Bioaccumulation of Cadmium in Marine Organisms," Frazier, JM. (Abstract):** "A general review of cadmium concentrations in marine organisms and studies of cadmium bioaccumulation is presented. Factors which influence cadmium concentrations, such as regional differences, seasonal fluctuations and salinity, are discussed and species which are likely to accumulate cadmium identified. Experimental studies designed to investigate the influence of some of these factors on cadmium bioaccumulation in a filter feeding bivalve mollusk, the American oyster (Crassostrea virginica), are presented. Field studies of seasonal dynamics of cadmium in oysters indicate patterns which may be correlated with seasonal physiological activity. The bioaccumulation of cadmium following input to estuarine systems by natural phenomena is observed. Cadmium concentrations in oysters collected from regions of different salinity suggest an inverse relationship between cadmium concentration and salinity. Laboratory experiments designed to investigate mechanisms of cadmium accumulation demonstrate that an inducible cadmium binding protein, similar to metallothiomein, is present in the American oyster."

**Reference 9: "Bioaccumulation of Cadmium in Marine Organisms," Ray, S. (Abstract): "**It has been established that, although Cd occurs in the marine environment in only trace concentrations, most marine organisms, especially molluscs and crustaceans, can accumulate it rapidly. Cadmium is not uniformly distributed in the body and selectively accumulates in specific organs like liver, kidney, gills, and exoskeleton. The concentrations in muscle tissues are several orders of magnitude lower. The disposition of Cd in the organisms in the laboratory studies generally parallels those in nature. A number of biotic factors like body size, maturity, sex, etc. influence bioaccumulation but extensive studies are still lacking. The chemical form of Cd in the environment is of prime importance in bioaccumulation by marine organisms. Salinity can affect the speciation of Cd, and bioaccumulation is affected by both temperature and salinity. The ultimate level of Cd in the organisms will depend not only on the biotic and abiotic factors but also on metabolism of the metal by the organisms…. Much of what is known about Cd bioaccumulation by marine organisms has come from laboratory studies and there are inherent dangers in trying to extrapolate the results to field situations. In spite of tremendous progress made over the years, the basic understanding of the bioaccumulation process is still very nebulous and will remain so until the uptake, storage, and elimination processes are fully understood."

**Reference 10. "The Comparison of Heavy Metal Accumulation Ratios of Some Fish Species in Enne Dame Lake (Kütahya/Turkey)." Uysal, K., et. al. (Abstract):** "The metal accumulation levels for muscle, skin, gill, liver and intestine tissues of some Cyprinidae species (Carassius carassius, Condrostoma nasus, Leuciscus cephalus and Alburnus alburnus) in Enne Dame Lake (Kütahya/Turkey), which is mostly fed by hot spring waters, were investigated…. . In all tissues and the species, while the bioaccumulation factors (BAFs) of Mn, Zn, Fe and Cu were remarkably high, the BAFs of Mg, Cr, Co, and B were also fairly low or none. Although the heavy metal accumulation levels for the muscle were generally lower than other tissues, there

were some exceptions. Cd level in the muscle of C. carassius was higher than the permissible limit stated by Turkish legislation, FAO and WHO."

**Reference 11. "Health Effects of Cadmium Exposure…," Järup, L. et. al. (Abstract):** "The diet is the main source of cadmium exposure in the Swedish nonsmoking general population. … It has been shown that a high fiber diet and a diet rich in shellfish increase the dietary cadmium intake substantially. Cadmium concentrations in agricultural soil and wheat have increased continuously during the last century. At present, soil cadmium concentrations increase by about 0.2% per year. Cadmium accumulates in the kidneys. Human kidney concentrations of cadmium have increased several fold during the last century…. In general, women have higher concentrations of cadmium in blood, urine, and kidney than men. The population groups at highest risk are probably smokers, women with low body iron stores, and people habitually eating a diet rich in cadmium. According to current knowledge, renal tubular damage is probably the critical health effect of cadmium exposure, both in the general population and in occupationally exposed workers. Tubular damage may develop at much lower levels than previously estimated, as shown in this report…. Even if the population average kidney concentration is relatively low for the general population, a certain proportion will have values exceeding the concentration where renal tubular damage can occur. It can be estimated that, at the present average daily intake of cadmium in Sweden, about 1% of women with low body iron stores and smokers may experience adverse renal effects related to cadmium. If the average daily intake of cadmium would increase to 30 micrograms/day, about 1% of the entire population would have cadmium-induced tubular damage. In risk groups, for example, women with low iron stores, the percentage would be higher, up to 5%. Both human and animal studies indicate that skeletal damage (osteoporosis) may be a critical effect of cadmium exposure."

**Reference 12. "Current Status of Cadmium as an Environmental Health Problem," Järup, L., & A. Akesson. (Abstract). "**Cadmium is a toxic metal occurring in the environment naturally and as a pollutant emanating from industrial and agricultural sources. Food is the main source of cadmium intake in the non-smoking population. The bioavailability, retention and toxicity are affected by several factors including nutritional status such as low iron status. Cadmium is efficiently retained in the kidney (half-time 10-30 years) and the concentration is proportional to that in urine (U-Cd). Cadmium is nephrotoxic, initially causing kidney tubular damage. Cadmium can also cause bone damage, either via a direct effect on bone tissue or indirectly as a result of renal dysfunction. After prolonged and/or high exposure the tubular injury may progress to glomerular damage with decreased glomerular filtration rate, and eventually to renal failure. Furthermore, recent data also suggest increased cancer risks and increased mortality in environmentally exposed populations. Dose-response assessment using a variety of early markers of kidney damage has identified U-Cd points of departure for early kidney effects between 0.5 and 3 microg Cd/g creatinine, similar to the points of departure for effects on bone. It can be anticipated that a considerable proportion of the non-smoking adult population has urinary cadmium concentrations of 0.5 microg/g creatinine or higher in non-exposed areas. For smokers this proportion is considerably higher. This implies no margin of safety between the point of departure and the exposure levels in the general population. Therefore, measures should be put in place to reduce exposure to a minimum, and the tolerably daily intake should be set in accordance with recent findings."

**Reference 13. "Cadmium Exposure in the Population: From Health Risks to Strategies of Prevention," Nawrot, TS., et. al. (Abstract):** "We focus on the recent evidence that elucidates our understanding about the effects of cadmium (Cd) on human health and their prevention. Recently, there has been substantial progress in the exploration of the shape of the Cd concentration-response function on osteoporosis and mortality. Environmental exposure to Cd increases total mortality in a continuous fashion without evidence of a threshold, independently of kidney function and other classical factors associated with mortality including age, gender, smoking and social economic status. Pooled hazard rates of two recent environmental population based cohort studies revealed that for each doubling of urinary Cd concentration, the relative risk for mortality increases with 17% (95% CI 4.2-33.1%; $P < 0.0001$). Tubular kidney damage starts at urinary Cd concentrations ranging between 0.5 and 2 μg urinary Cd/g creatinine, and recent studies focusing on bone effects show increased risk of osteoporosis even at urinary Cd below 1 μg Cd/g creatinine. The non-smoking adult population has urinary Cd concentrations close to or higher than 0.5 μg Cd/g creatinine. To diminish the transfer of Cd from soil to plants for human consumption, the bioavailability of soil Cd for the plants should be reduced (external bioavailability) by maintaining agricultural and garden soils pH close to neutral (pH-H(2)O of 7.5; pH-KCL of 6.5). Reducing the systemic bioavailability of intestinal Cd can be best achieved by preserving a balanced iron status. The latter might especially be relevant in groups with a lower intake of iron, such as vegetarians, and women in reproductive phase of life. In exposed populations, house dust loaded with Cd is an additional relevant exposure route. In view of the insidious etiology of health effects associated with low dose exposure to Cd and the current European Cd intake which is close to the tolerable weekly intake, one should not underestimate the importance of the recent epidemiological evidence on Cd toxicity as to its medical and public health implications."

**Reference 14. "Cadmium Linked to Breast Cancer, " Brown, Anthony":** "Women with the highest levels of cadmium in their urine have more than a two-fold higher risk of breast cancer than women with the lowest levels, according to a new study. However, further studies are needed to determine if these elevated levels are a cause or effect of breast cancer. Although cadmium, a heavy metal, has been classified as a probable cancer-causing substance by the US Environmental Protection Agency, until now no human studies have investigated its link with breast cancer."

**Reference 15. "Health Concerns of Consuming Cockles (Cerastoderma edule L.) from a Low Contaminated Coastal System," Figuera E., et. al. (Abstract):** "Commercial and recreational harvesting of shellfish within the coastal systems is usually very extensive. Since these ecosystems are frequently subjected to contamination, namely from agricultural, urban and industrial activities, and shellfish generally display a high capacity to bioaccumulate metals, populations may be at risk in terms of toxic metal exposure as a consequence of the harvesting and ingestion of near shore coastal marine organisms."

**Reference 16. "Sea Ducks and Aquaculture: the Cadmium Connection," Bendell LI. (Abstract):** "Elevated concentrations of cadmium have been reported in the kidneys of sea ducks that forage along the Pacific Northwest, and cadmium has been postulated as a possible cause of population declines. The blue mussel (Mytilus spp.) which occurs in dense numbers on aquaculture structures and are a primary prey item for sea ducks also contain elevated cadmium

concentrations. To determine if foraging on mussels associated with aquaculture structures could pose a toxicological risk to sea ducks, amounts of cadmium ingested per body weight per day by a representative sea duck species, the surf scoter (Melanitta perspicillata), were estimated and compared to the reported avian cadmium NOAEL (no observable adverse effect level) and LOAEL (lowest observable adverse effect level). Results indicate that in some locations within the Pacific Northwest, sea ducks could be exposed to toxicologically significant levels of cadmium associated with mussels foraged from aquaculture structures. This raises the possibility that such exposure could be contributing to observed population declines in these species."

**Reference 17: "Toxicity of Cadmium to Six Species in Two Genera of Crayfish and the Effect of Cadmium on Molting Success," Wigginton, AJ, and Birge W J. (Abstract): "**Nine acute (96-h) toxicity tests were conducted on six species of crayfish (Cambaridae). Six tests focused on adults, and three tests examined juveniles…. Crayfish sensitivity to Cd varied by a factor of nine among species tested as adults and by a factor of 17 among species tested as juveniles. Molting was a sensitive life stage for crayfish. Most individuals that molted shortly before or during exposure to Cd died, whereas all controls that molted in the adult assays survived. Because molting is a sensitive, recurring life-cycle event, molting individuals should be included in toxicological analysis despite some contrary recommendations."

**Reference 18: "Cadmium Hazards to Fish, Wildlife, and Invertebrates…," Eisler, Ronald (Summary):** "Cadmiuim contamination of the environment is especially severe in the vicinity of smelters and urban industrialized areas. There is no evidence that cadmium, a relatively rare heavy metal, is biologically essential or beneficial; on the contrary cadmium is a known teratogen and carcinogen, a probable mutagen, and has been implicated as the cause of severe deleterious effects on fish and wildlife. The freshwater biota is the most sensitive group; concentrations of 0.8 to 9.9 ug Cd/L (ppb) in water were lethal to several species of aquatic insects, crustaceans, and teleosts, and concentrations of 0.7 to 570 ppb were associated with sublethal effects such as decreased growth, inhibited reproduction, and population alterations…. Freshwater and marine aquatic organisms accumulated measurable amounts of cadmium from water containing Cd concentrations not previously considered hazardous to public health or to many species of aquatic life; i.e., 0.02 to 10 ppb. … It is now conservatively estimated that adverse effects on fish or wildlife are either pronounced or probable when cadmium concentrations exceed 3 ppb in fresh water, 4.5 ppb in saltwater, 100 ppb in the diet, or 100 g $Cd/m^3$ in air."

**Reference 19. "Exposure Level and Bioaccumulation of Polycyclic Aromatic Hydrocarbons (PAHs) in Edible Marine Organisms." ISAAC A. OLOLADE, and LABUNMI LAJIDE. (Abstract):** "The results show that total PAHs (with bioconcentration factors in brackets) varied from less than minimum detectable limit (MDL) to 6.87 μg/g (7.83 – 44.45) in fish, 0.02 to 85.6 μg/g (234.5 – 279.6) in crab and <MDL to 79.43 μg/g (219.8 – 243.9) in periwinkle. PAHs composition pattern was dominated by 2 to 3-rings (83.3%) followed by those of 4-rings (12.5%) and 5-rings (4.2%). Higher PAH content was found during the wet season in periwinkles (62.8%) and crab (59.5%) unlike in fish. The bioconcentration factors (BCFs) ranged from 7.83 – 44.45; 219.8 – 243.9 and 234.5 – 279.6 in fish, periwinkles and crabs respectively. Biota-sediment accumulation factor (BSAF) for the 4- and 5-member rings are much higher than those for the 3-member rings PAHs. The BSAF tend to decrease with

increasing PAHs concentration in sediments. Fish showed little tendency to bioaccumulate PAHs due to their rapid metabolism, unlike crab and periwinkle with very poor metabolic capacity. With regard to PAH bioaccumulation, whole body of periwinkles seems to be the species best suited as an indicator organism."

**Reference 20. "Proliferative responses of harbor seal (Phoca vitulina) T lymphocytes to model marine pollutants." Neale JC[1], van de Water JA, Harvey JT, Tjeerdema RS, Gershwin ME. (Abstract):** "In recent years, population declines related to viral outbreaks in marine mammals have been associated with polluted coastal waters and high tissue concentrations of certain persistent, lipophilic contaminants. Such observations suggest a contributing role of contaminant-induced suppression of cell-mediated immunity leading to decreased host resistance. Here, we assessed the effects of the prototypic polycyclic aromatic hydrocarbon (PAH), benzo[a]pyrene (B[a]P), and two polychlorinated biphenyls (PCBs), CB-156 and CB-80, on the T-cell proliferative response to mitogen in harbor seal peripheral lymphocytes. Despite the variability associated with our samples from free-ranging harbor seals, we observed a clear suppressive effect of B[a]P (10 uM) exposure on T cell mitogenesis. Exposures to 10 uM CB-156 and CB-80, and 1.0 and 0.1 uM B[a]P, did not produce significant depression in lymphoproliferation. Exposure to the model PAH at 10 uM resulted in a 61% (range 34-97%) average reduction in lymphoproliferation. We were able to rule out a direct cytotoxic effect of B[a]P, indicating that observed effects were due to altered T cell function. Based on our in vitro results, we hypothesize that extensive accumulation of PAH by top-trophic-level marine mammals could alter T cell activation in vivo and impaired cell-mediated immunity against viral pathogens."

**Reference 21. " PAH- and PCB-induced alterations of protein tyrosine kinase and cytokine gene transcription in harbor seal (Phoca vitulina) PBMC." Neale JC[1], Kenny TP, Tjeerdema RS, Gershwin ME. (Abstract):** "Mechanisms underlying in vitro immunomodulatory effects of polycyclic aromatic hydrocarbons (PAHs) and polychlorinated biphenyls (PCBs) were investigated in harbor seal peripheral leukocytes, via real-time PCR. We examined the relative genetic expression of the protein tyrosine kinases (PTKs) Fyn and Itk, which play a critical role in T cell activation, and IL-2, a cytokine of central importance in initiating adaptive immune responses. IL-1, the macrophage-derived pro-inflammatory cytokine of innate immunity, was also included as a measure of macrophage function. Harbor seal PBMC were exposed to the prototypic immunotoxic PAH benzo[a]pyrene (BaP), 3,3',4,4',5,5'-hexachlorobiphenyl (CB-169), a model immunotoxic PCB, or DMSO (vehicle control). Exposure of Con A-stimulated harbor seal PBMC to both BaP and CB-169 produced significantly altered expression in all four targets relative to vehicle controls. The PTKs Fyn and Itk were both up-regulated following exposure to BaP and CB-169. In contrast, transcripts for IL-2 and IL-1 were decreased relative to controls by both treatments. Our findings are consistent with those of previous researchers working with human and rodent systems and support a hypothesis of contaminant-altered lymphocyte function mediated (at least in part) by disruption of T cell receptor (TCR) signaling and cytokine production."

**Reference 22. "Toxic Effects of Various Pollutants in 11B7501 Lymphoma B Cell Line from Harbor Seal (Phoca Vitulina)." Frouin H[1], Fortier M, Fournier M. (Abstract):** "Although, heavy metals and polycyclic aromatic hydrocarbons (PAHs) have been reported at

high levels in marine mammals, little is known about the toxic effects of some of these contaminants. In this study, we assessed the immunotoxic and genotoxic effects of seven heavy metals (arsenic, vanadium, selenium, iron, zinc, silver and chromium) and one PAH (benzo[a]pyrene or B[a]P) on a lymphoma B cell line from harbour seal (Phoca vitulina). A significant reduction in lymphocyte proliferation was registered following an exposure to 0.05 microM of B[a]P, 5 microM of arsenic or selenium, 50 microM of vanadium, 100 microM of silver and 200 microM of iron. On the contrary, zinc increased the lymphoproliferative response at 200 microM. Decreased phagocytosis was observed at 20 microM of arsenic, 50 microM of B[a]P or selenium, 200 microM of zinc and 500 microM of vanadium. Micronuclei induction occurred with 0.2 microM of B[a]P, 100 microM of vanadium and with 200muM of arsenic or selenium. Exposure to 50muM of arsenic decreased G(2)/M phase of the cell cycle. Chromium did not induce any effects at the concentrations tested. Concentrations of heavy metals (except silver and vanadium) and B[a]P inducing an toxic effect are within the environmental ranges reported in the blood tissue of pinnipeds. The reduction of some functional activities of the harbour seal immune system may cause a significant weakness capable of altering host resistance to disease in free-ranging pinnipeds."

**Reference 23**. **"Congenital neuroglial heterotopia in a neonatal harbor seal (Phoca vitulina richardsi ) with evidence of recent exposure to polycyclic aromatic hydrocarbons."** **Harris HS[1], Facemire P, Greig DJ, Colegrove KM, Ylitalo GM, Yanagida GK, Nutter FB, Fleetwood M, Gulland FM. (Abstract):** "A male neonatal Pacific harbor seal (Phoca vitulina richardsi) stranded off the coast of California, USA, was presented for rehabilitation with numerous partially haired, soft tissue masses around the mouth and in the oropharynx. Because of the extent of the lesions, the seal was humanely euthanized. Histologically, the masses consisted of subepithelial connective tissue and subcutis expanded by a proliferation of streams and bundles of spindle to stellate cells. Morphology of these cells suggested a neural origin, which was confirmed by positive immunohistochemistry for two neural markers, S-100 protein and glial fibrillary acidic protein, so the masses were diagnosed as neuroglial heterotopia. Heterotopic neuroglial tissue is a rare lesion comprised of benign mature neural tissue in an ectopic location with no connection to the central nervous system. Results of polycyclic aromatic hydrocarbon (PAH) metabolite analysis of bile indicated recent exposure to a petroleum source. Although fetal exposure to PAHs in utero can cause neurotoxicity and affect normal embryonic development, it is unknown whether gestational exposure occurred in this case."

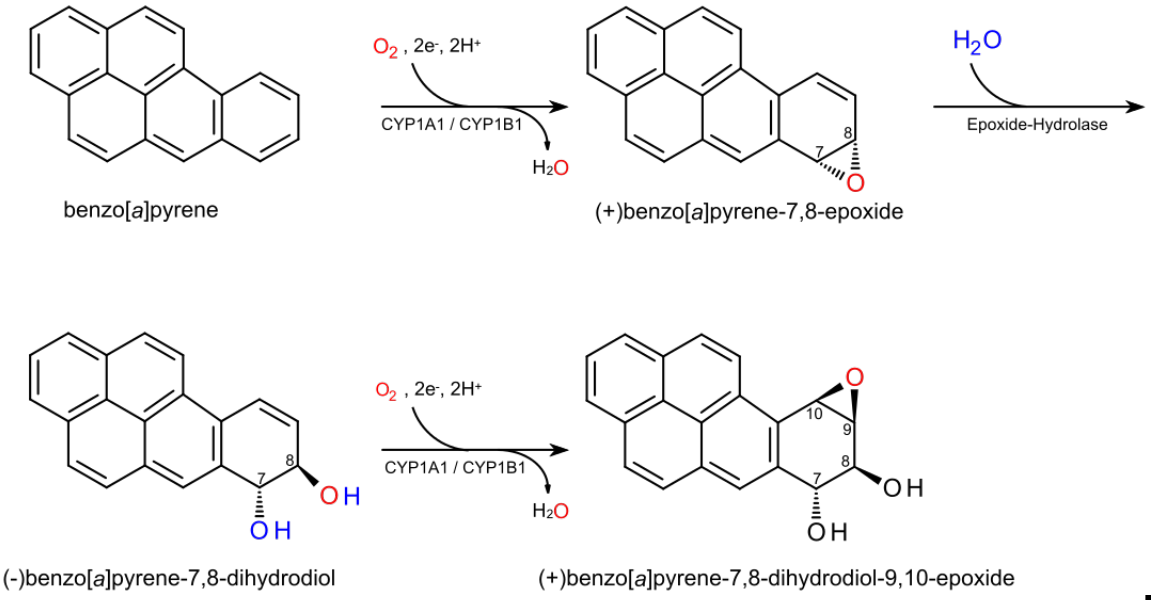
**Reference 24. "Assessment of current dietary intake of organochlorine contaminants and polycyclic aromatic hydrocarbons in killer whales (Orcinus orca) through direct determination in a group of whales in captivity. Formigaro C[1], Henríquez-Hernandez LA[2], Zaccaroni A[1], Garcia-Hartmann M[3], Camacho M[2], Boada LD[2], Zumbado M[2], Luzardo OP[4]. (Abstract):** "We determined the levels of 16 polycyclic aromatic hydrocarbons (PAHs), 19 organochlorine pesticides (OCPs) and 18 polychlorinated biphenyls (PCBs) in the plasma of captive adult killer whales and in their food. The goal of this research was the assessment of the dietary exposure of killer whales to these pollutants to gain insight on what is the actual magnitude of the exposure in this species, which is considered among the most contaminated in the planet. Plasma median ∑OCP and ∑PCB contents were 3150.3 and 7985.9 ng g(-1)l.w., respectively. A total of 78.9% of the PCBs were marker-PCBs, and 21.1% were dioxin-like PCBs (6688.7 pg g(-1)l.w. dioxin toxic equivalents). This is the first report of the

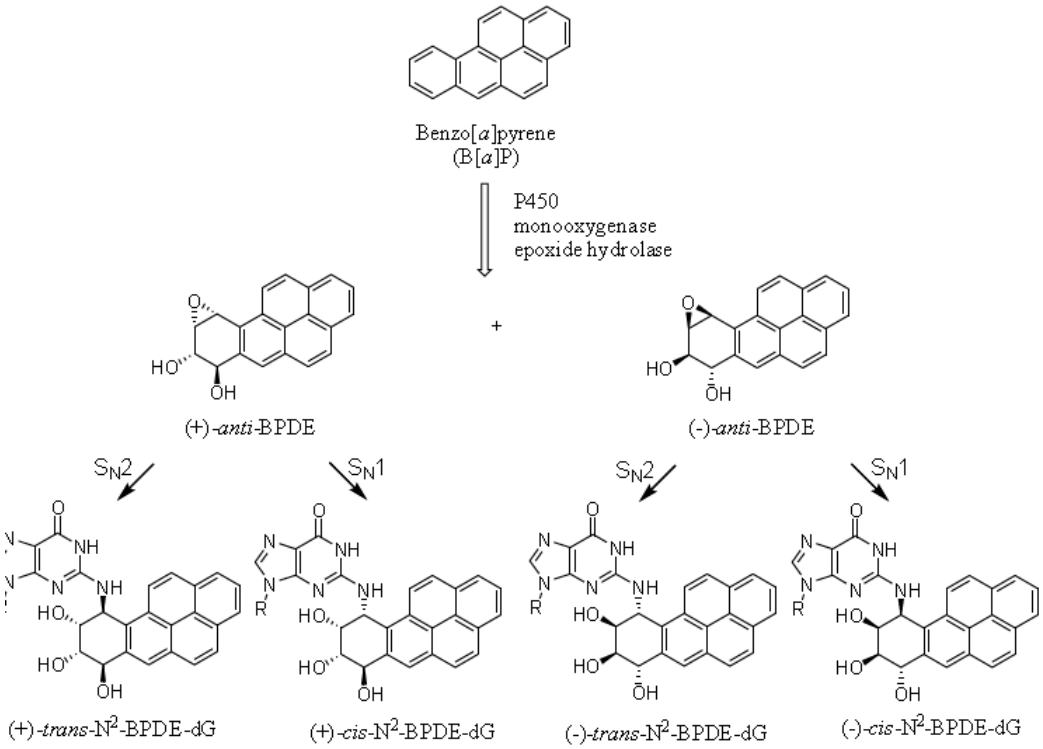
blood levels of PAHs in killer whales, and their median value was 1023.1 ng g(-1)l.w. In parallel, we also determined the levels of these contaminants in the fish species that are used to feed these animals to estimate the orcas' average daily dietary intake of pollutants. All the contaminants in the fish were detected in the plasma of the killer whales, and proportionality between the intake and the blood levels was observed in all the animals. The calculated intake was extremely high for certain contaminants, which is a concern, giving a glimpse of what possibly occurs in the wild, where exposure to these contaminants can be even higher. Therefore, although many of these chemicals have been banned for decades, even today, the levels of these chemicals could reach very toxic concentrations in the tissues of these endangered animals because of their diet."

**Reference 25.** "**Metabolism of chemical carcinogens." Guengerich FP1. (Abstract):** "The transformation of chemicals is important in carcinogenesis, both in bioactivation and detoxification. Major advances in the past 20 years include appreciation of the migration of reactive electrophiles, the ability of Phase II conjugating enzymes to activate chemicals, understanding of the human enzymes, the realization that DNA modification can result from endogenous chemicals, and the demonstration that cancers can result from the metabolism of chemicals to non-covalently bound products. Pathways of transformation in which major insight was gained during the past 20 years include nitropolycyclic hydrocarbons, polycyclic hydrocarbons and their diols, vinyl halides and dihaloalkanes. Advances in analytical methods and recombinant DNA technology contributed greatly to the study of metabolism of chemical carcinogens. Major advances have been made in the assignment of roles of individual enzymes in reactions. The knowledge developed in this field has contributed to growth in the areas of chemoprevention, molecular epidemiology and species comparisons of risk. Some of the areas in which future development relevant to carcinogen metabolism is expected involve pathways of transformation of certain chemicals, regulation of genes coding for many of the enzymes under consideration and genomics."

**Reference 26. "Carcinogenesis by chemicals: an overview--G. H. A. Clowes memorial lecture." Miller JA.**

**Reference 27 (following page).**


O2 , 2e-, 2H+
CYP1A1 / CYP1B1
H2O
H2O
Epoxide-Hydrolase
benzo[a]pyrene
(+)benzo[a]pyrene-7,8-epoxide
(-)benzo[a]pyrene-7,8-dihydrodiol
(+)benzo[a]pyrene-7,8-dihydrodiol-9,10-epoxide


Benzo[a]pyrene
(B[a]P)
P450
monooxygenase
epoxide hydrolase
(+)-anti-BPDE
(-)-anti-BPDE
SN2
SN1
(+)-trans-N2-BPDE-dG
(+)-cis-N2-BPDE-dG
(-)-trans-N2-BPDE-dG
(-)-cis-N2-BPDE-dG

**Reference 28**. General Reference on PAH and Cancer: ***Cancer Medicine,*** **6th edition. Holland-Frei. Edited by Donald W. Kufe, et. al.**

**Reference 29**. This article is important, Not so much for what it says specifically about sealanders, but for what it says about residual sedimentary captured PAHs that provide a toxic background of exposure of wildlife, even in the absence of a catastrophic oil spill. **"Sediment-associated aliphatic and aromatic hydrocarbons in coastal British Columbia, Canada: concentrations, composition, and associated risks to protected sea otters." Harris KA[1], Yunker MB, Dangerfield N, Ross PS. Author information (Abstract):** "Sediment-associated hydrocarbons can pose a risk to wildlife that rely on benthic marine food webs. We measured hydrocarbons in sediments from the habitat of protected sea otters in coastal British Columbia, Canada. Alkane concentrations were dominated by higher odd-chain n-alkanes at all sites, indicating terrestrial plant inputs. While remote sites were dominated by petrogenic polycyclic aromatic hydrocarbons (PAHs), small harbour sites within sea otter habitat and sites from an urban reference area reflected weathered petroleum and biomass and fossil fuel combustion. The partitioning of hydrocarbons between sediments and adjacent food webs provides an important exposure route for sea otters, as they consume ~25% of their body weight per day in benthic invertebrates. Thus, exceedences of PAH sediment quality guidelines designed to protect aquatic biota at 20% of the sites in sea otter habitat suggest that sea otters are vulnerable to hydrocarbon contamination even in the absence of catastrophic oil spills."

**Reference 30**. This article relates carcinogenic PAH in sediments to bottom–dwelling–foraging fish. It establishes A direct link between such PAH and cancerous lesions in those fish. **"Epizootics of cancer in fish associated with genotoxins in sediment and water." Baumann PC. (Abstract):** "Neoplasm epizootics in fish from a wide variety of freshwater, marine, and estuarine locations have been associated with genotoxins in sediment or water. The majority of cases have involved benthic or bottom feeding fish living in habitats with sediment contaminated by PAHs. The most common lesions involved in such epizootics include liver neoplasms, both biliary and hepatic, and skin neoplasms. Laboratory research has demonstrated the ability of fish to metabolize carcinogenic PAHs such as B(a)P into the ultimate carcinogen with the resulting formation of DNA adducts. Fish dosed with B(a)P or sediment extracts containing carcinogenic PAHs have developed skin and liver neoplasms. In the Black River, OH, neoplasm prevalence in wild brown bullhead has reflected PAH exposure as the latter has changed due to coke plant closures and remedial dredging activity. The weight of evidence supports a cause-and-effect relationship between exposure to genotoxins in sediment and water and neoplasm epizootics in wild fish populations."

**Reference 31**. This article describes the kind of experiment that would be needed to properly monitor exposure of wildlife to DNA damaging chemicals like PAH—simple concentration levels won't give the correct answer! **"Use of DNA adducts to identify human health risk from exposure to hazardous environmental pollutants: the increasing role of mass spectrometry in assessing biologically effective doses of genotoxic carcinogens." Farmer PB[1], Singh R. (Abstract):** "The carcinogens to which humans are exposed are normally in the form of complex mixtures, and much effort has gone into determining the nature of the most significant carcinogenic components in these mixtures and their mechanisms of action.

Essential to achieving this aim in exposed populations is the use of biomarkers, which can characterize the chemical nature of the carcinogens involved and identify key biological effects that result from the exposure. DNA adducts are particularly appropriate as biomarkers in the case of genotoxic carcinogens as they indicate the biologically effective dose of the genotoxin in the target tissue under study. This review considers in particular the use of mass spectrometry (MS), which is having an increasing role in the determination of DNA adducts. Compared to other existing DNA damage detection methods, such as 32P-postlabeling, HPLC-fluorescence or electrochemical detection, immunoassay-based techniques and modified Comet assays, MS provides improved structural characterization of adducts. Greater selectivity in the analyses is achieved by the use of tandem MS with selected reaction monitoring or constant neutral loss of ions. Use of capillary/nano liquid chromatography and micro/nano electrospray ionization improves the analytical sensitivity and higher throughput may be obtained by the use of online-column switching. The application of microfluidics technology offers exciting new possibilities for interfacing sample preparation to the mass spectrometer. Despite these improvements in the use of MS for adduct detection, the main current requirement is to validate these methods both analytically and in molecular epidemiology studies. More knowledge of the stability of stored samples is required. Development of sensitive mass spectrometric DNA adductomic screening systems, and of long-term biomarkers (e.g., phosphotriester adducts that are not repaired efficiently) seems important areas for the future assessment of the effects of human exposure to environmental genotoxins, together with studies of dose-response relationships at low doses."

**Reference 32**. Again this article repeats the theme, with a different species, that carcinogenic hydrocarbons cause disease in fish– that means they are taken out and stored at some level in fish. Predators would therefore also be eating PAHs and accumulating them

**"Relationships between hepatic neoplasms and related lesions and exposure to toxic chemicals in marine fish from the U.S. West Coast." Myers MS[1], Landahl JT, Krahn MM, McCain BB. (Abstract):** "English sole (Parophrys vetulus) inhabiting polluted waterways and embayments of Puget Sound, Washington, are affected with a variety of multiple, co-occurring idiopathic hepatic lesions, including unique degenerative conditions, putatively preneoplastic foci of cellular alteration, and neoplasms. Results of a statistical analysis of the patterns of co-occurrence of these lesions in wild English sole are consistent with the concept that these lesions represent morphologically identifiable steps forming a sequence of progression ultimately leading to the development of hepatic neoplasms. This progressive sequence parallels the pattern identified in experimental models of chemically induced hepatocarcinogenesis in rodents. The rationale for the hypothesis that these lesions in wild English sole can be caused by exposure to certain hepatoxic and hepatocarcinogenic xenobiotic compounds in the marine environment is based on the demonstration of significant and consistent statistical associations between levels of aromatic hydrocarbons (AHs) in sediment and prevalences of these idiopathic liver lesions; a significant contribution by sediment AHs to the variability in hepatic neoplasm prevalence in a logistic regression model; significantly increased probabilities for several idiopathic lesions in sole from chemically contaminated sites in Puget Sound; significant correlations between uptake of polycyclic aromatic hydrocarbons, as measured by levels of fluorescent metabolites of aromatic compounds in bile of sole, and prevalences of several hepatic lesion types; and experimental induction of unique degenerative, proliferative, and putatively preneoplastic focal lesions in English sole injected with either benzo(a)pyrene or a polycyclic aromatic

hydrocarbons (PAH) enriched fraction of an extract from a contaminated urban sediment from Puget Sound."

**Reference 33**. **"Overview of studies on liver carcinogenesis in English sole from Puget Sound; evidence for a xenobiotic chemical etiology. I: Pathology and epizootiology."** **Myers MS[1], Landahl JT, Krahn MM, Johnson LL, McCain BB.** (**Abstract):** "Livers of wild English sole (Parophrys vetulus) from polluted waterways and embayments of Puget Sound, Washington, are affected by a spectrum of multiple, co-occurring idiopathic hepatic lesions, including neoplasms, putative preneoplastic foci of cellular alteration, and unique degeneration conditions. Results from a statistical analysis of the patterns of co-occurrence of these lesions in wild English sole indicate that these lesions represent morphologically identifiable steps leading to the development of hepatic neoplasms. This sequence parallels the lesion progression in experimental models of chemically induced liver carcinogenesis in rodents. The hypothesis that these lesions in wild English sole can be caused by exposure to certain xenobiotic hepatotoxic and hepatocarcinogenic compounds in Puget Sound is based on: a) statistical associations between levels of aromatic hydrocarbons (sigma AHs) in sediment and prevalences of these idiopathic liver lesions, b) the contribution of sigma AHs in accounting for the variability in hepatic neoplasm prevalence in a logistic regression model, c) elevated odds ratios for several idiopathic hepatic lesion types in sole from polluted sites in Puget Sound, d) significant correlations between prevalences of idiopathic hepatic lesions and levels of fluorescent metabolites of aromatic compounds (FACs) in bile of English sole, and e) experimental induction of putatively preneoplastic focal lesions in English sole injected with a PAH-enriched fraction of an extract from a contaminated urban sediment from Puget Sound, that were morphologically identical to lesions found in wild English sole from the same site."

**V. Bibliography** (The majority of published articles listed here are available on-line via a Google search for the title or the PMID number)**:**

**"Acute Toxicity of Copper, Cadmium, and Zinc to Larvae of the Crab *Paragrapus quadridentatus* (H. Milne Edwards), and Implications for Water Quality Criteria."** M. Ahsanullah and G.H. Arnott. *Australian Journal of Marine and Freshwater Research* 29 (1) 1 – 8.

**"Assessment of current dietary intake of organochlorine contaminants and polycyclic aromatic hydrocarbons in killer whales (Orcinus orca) through direct determination in a group of whales in captivity."** Formigaro C[1], Henríquez-Hernandez LA[2], Zaccaroni A[1], Garcia-Hartmann M[3], Camacho M[2], Boada LD[2], Zumbado M[2], Luzardo OP[4]. Sci Total Environ. 2014 Feb 15;472:1044-51. doi: 10.1016/j.scitotenv.2013.11.127. Epub 2013 Dec 15.

**"Bioaccumulation of Cadmium in Marine Organisms."** Frazier JM. *Environ Health Perspect.* 1979 Feb; 28:75-9. PMID: 488051.

**"Bioaccumulation of Cadmium in Marine Organisms."** Ray S. *Experientia Suppl.* 1986; 50:65-75. PMID: 3525217.

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http://www.nws.usace.army.mil/Portals/27/docs/regulatory/NewsUpdates/BPDock2014/BP%20Cherry%20Point%20Dock%20DEIS%20May%202014.pdf

**"Cadmium Exposure and Breast Cancer Risk."** McElroy, Jane, Martin M. Shafer, Amy Trentham-Dietz, John M. Hampton, and Polly A. Newcomb. *Journal of the National Cancer Institute,* June 21, 2006. Pp. 869-73.

**"Cadmium Exposure in the Population: From Health Risks to Strategies of Prevention."** Nawrot TS, Staessen JA, Roels HA, Munters E, Cuypers A, Richart T, Ruttens A, Smeets K, Clijsters H, Vangronsveld J. *Biometals.* 2010 Oct; 23(5): 769-82. Epub 2010 Jun 3. Source: Centre for Environmental Sciences, Hasselt University, Diepenbeek, Belgium. PMID: 20517707.

**Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.** Eisler, Ronald. Biological Report 85(1.2), July 1985. Contaminant Hazard Reviews Report No. 2. Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD, 1985.

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