

Written Testimony of Carys L. Mitchelmore, Ph.D.

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**Carys L. Mitchelmore, Ph.D.,
Associate Professor,
University of Maryland Center for Environmental Science,
Chesapeake Biological Laboratory,
P.O. Box 38,
Solomons, MD 20688**

Good morning Chairman Whitehouse and members of the subcommittees. I am Carys Mitchelmore and I would like to take this opportunity to thank you for inviting me today to highlight a few examples of the many issues we face today concerning pollutants in our coastal and marine environments.

By way of background: I am faculty at the UMCES Chesapeake Biological laboratory with expertise in studying the fate and effects of pollutants on aquatic organisms. I have been conducting research and publishing books and articles for 15 years concerning the impacts of metals, organic chemicals, biological pollutants, oil and oil spill dispersants on many species, including corals, reptiles, fish and oysters. Today I am representing my views as a researcher in the field of environmental health and as a local resident concerned with the deteriorating Health of the Chesapeake Bay watershed and our global aquatic ecosystems. I feel very strongly about pollution issues, indeed my career path as an aquatic toxicologist was set in place at the young age of 6, after stepping on a tar ball at a local beach. That left a lasting impression on me and I grew up fascinated with the rock pools and, unfortunately the all too often, oil sheens within. A challenge for me today was my choice of specific topics to discuss; we are indeed faced with multiple issues pertaining to pollution of our aquatic ecosystems. My testimony today will focus on four main issues; using case examples from my own research, including those relating to oil and oil spill dispersant toxicity.

Currently, the greatest threats to our coastal and marine environments can broadly be described as;

1. Land-use changes and increasing impervious surface coverage. Urban stormwater run-off has been described as a major source of pollutants, such as sediments, nutrients, metals and organic contaminants including oil, polycyclic aromatic hydrocarbons (PAHs), bacteria and garbage.

2. The expansion of human populations inhabiting coastal locations. The rate of expansion often outstrips the outdated and/or inappropriate infrastructures. Waste water treatment plants (WWTP), rivers and coastlines are often overloaded with nutrients and bacterial contaminants and are inundated with a diverse array of chemicals, from industrial compounds, PAHs, metals, radionucleotides, pharmaceuticals and personal care products.

3. The increased use of our oceans. Increased shipping traffic has led to pollution including, garbage and trash, sewage (bacteria), metals and organic pollutants. It has also enhanced the global movement of species to non-native habitats (invasive species).

4. New products and technological expansions. Huge numbers and diversity of manufactured chemicals have been produced since the industrial revolution and there appears to be little slowing of this trend. Numerous new chemicals are entering the market place, many with unknown (or un-predicted) environmental risks. For example, the environmental risks associated with nanoparticles have not been fully addressed, although these are currently the focus of many new initiatives and programs (such as the National Nanotechnology Initiative).

The four key points I would like to raise during my testimony are the following:

1. The issue of contaminant mixtures and multiple sources.

- Unknown and un-predicted interactions occur between contaminants (and additional interactions can also occur with environmental variables).

2. Emerging contaminants of concern.

- New products (Case study 1; flame retardants).
- Are we replacing the 'bad' with the 'ugly' or the 'good'?

3. Sub-lethal toxicity issues.

- Increased knowledge of the subtle, yet deadly, effects of contaminants.
- Multiple ways in which contaminants act on biological systems; there are common themes (mechanisms) exhibited by many chemicals.
- Examples; bioenergetic and behavioral toxicants.
- Case study 2; oil and oil spill dispersants.
 - sublethal and delayed effects in sensitive species.
 - altered routes of exposure.

4. Ecosystem-based approaches.

- The effects of chemicals on food webs (indirect toxicity mechanisms).
- Sensitive species.

Overview and Introduction:

Our coastlines contain highly diverse complex ecosystems, teeming with multiple and interacting species that carry out many important ecosystem services. Coastlines (and connected estuaries and rivers) are often important nursery grounds for numerous

organisms, including oceanic species. The pollution of our aquatic ecosystems, including our coastlines and oceans is increasing. Water, sediment and the tissues of resident organisms contain a multitude of industrial chemicals. Even species far from pollution sources have been shown to contain chemical contaminants in their tissues. For example, flame retardants (e.g. polybrominated diphenyl ethers or PBDEs) have been detected in Arctic seals and polar bears (REF 1).

Chemical contaminants affect the health and functioning of these impacted ecosystems in numerous ways, by altering the water itself and/or impacting the resident flora and fauna (in potentially positive and negative ways). Ecosystems and food webs are changing, ecosystem services are lost, species are lost, resources (water and food) are being contaminated. Not only are we losing sources of food, we are also losing species that protect our coastlines from erosion (storm damage). These effects have consequences to our economy, recreation and even lead to potential health issues. Many historic local cultures and ways of life that have been traditional for generations of families have been eroded by declining fisheries. Fish consumption advisories are commonplace for many species of fish, even those residing mainly in the open Oceans. In addition, rivers, lakes and beaches are being closed for recreational use, impacting tourism. We are beginning to lose the memory of what an un-impacted coastline looks like.

Many examples of ecosystem health declines appear to correlate well with the onset of the Industrial revolution and the increased expansion of industrial and residential chemicals (e.g. increases in coral bleaching spatially and temporally (REF 2,3)). Our oceans and coastal systems are receiving a barrage of pollutants, that are numerous, increasing in concentration and are of diverse origins and types. Pollutants are defined as anything that impacts the normal functioning of an ecosystem. These include trash and garbage, nutrients, metals, organic contaminants, sediments, radionucleotides, biological entities including toxins (e.g. for example from harmful algal blooms), bacteria, viruses even invasive species. Pollutants also include alterations in temperature, pH and salinity. Although my testimony is limited to using examples of manufactured chemical contaminants and oil, I highlight these other pollutants as they often work in concert, enhancing the toxicity of chemical contaminants.

Two broad types of chemical contaminants exist; those chemicals that are designed for a specific purpose that inadvertently affect aquatic systems. Second those specifically produced and used for their toxicological properties, e.g. pesticides, antifoulant paints and oil spill dispersants.

1. The issue of contaminant mixtures and multiple sources.

Recent years have shown dramatic increases not just in the sheer volume of chemical contaminants but also in the numbers of different types of chemicals used today. Thousands of different chemicals are present in our aquatic environments. This creates the potential for a toxic 'soup' of unknown effects, which cannot be predicted even if the individual chemical constituents are known. Interactions of chemicals with other physical, chemical or biological entities can change the exposure routes of chemicals, make them

more available to organisms (bioaccumulative) and in some cases even make them more toxic. We are only just beginning to unravel some of the complex interactions that occur when these chemical contaminants make their way ultimately into coastal and oceanic ecosystems.

Chemicals interact with each other often in unknown ways. We know this in the medical field. For example, when we get sick our Doctors and pharmacists carefully check for interactions between prescription medications if we are taking more than one item. In some cases taking drug A with drug B can result in negative consequences, the combination may even be fatal. Yet if you took drug A and B singly at different times using the same doses no adverse effects would be seen. This phenomenon is called synergism. This analogy also holds true for organisms exposed to the barrage of multiple contaminants they are often now faced with. There are numerous examples of these types of synergistic effects in the aquatic environment; where the toxicity of a chemical mixture far exceeds that which could have been predicted based on the individual contaminant toxicities alone. For example, synergism was observed using combinations of organophosphates in toxicity tests examining the response of salmon to these exposures (REF 4).

Furthermore other environmental variables and stressors, including issues related to global climate change (i.e. increased temperatures, acidification) can influence the toxicity of a chemical, either due to inherent chemical properties of the contaminant or the organisms biological response to the contaminant and environmental variables. For example, an organism's exposure to a chemical contaminant can also increase its susceptibility to disease. Many contaminants have been shown to depress an organism's immune system making them vulnerable to infections (REF 5).

These contaminant mixtures lead to difficulties in determining cause and effect and from a regulatory perspective assigning 'blame' as a large amount of our pollutants are derived from non-point sources. In some cases we are purposely moving pollutants from one area to another. For example in cleaning up our wastewater treatment plant (WWTP) effluents many chemical contaminants become concentrated in sewage sludge (biosolids). A recent survey found a minimum of 38 different pharmaceuticals and personal care products in every composite U.S. biosolid sample analysed. High levels of triclosan (the active ingredient in antibacterial soap) were found with mean concentrations of 12 mg kg⁻¹; REF 6). Biosolids are minimally regulated (primarily for pathogens and metals) and commonly applied onto land as fertilizers. They have the potential to run off the land and contaminate local aquatic ecosystems. A recent research paper published by colleagues at the Virginia Institute of Marine Science (VIMS) jointly with my laboratory demonstrated the presence of multiple organic contaminants in biosolids, often at high levels. Fish exposed to these biosolids exhibited sublethal toxicological effects, such as, increased DNA damage (REF 7). Are we simply transferring pollutants that ultimately may make their way back into aquatic systems? In some cases, for example oil spill dispersants, moving pollutants is indeed one of the main reasons behind their intended use. Dispersants are used to prevent the oil from impacting sensitive shorelines by moving the surface-slick into the water column and potentially also to the benthos (e.g. sea-floor).

Sediment, trash/garbage and potentially nanoparticles have been shown to enhance the toxicity of some chemical contaminants by making them more bioavailable to organisms i.e. changing their route of exposure. For example, dissolved pollutants can stick to sediment particles and be ingested by fish or suspension feeding organisms.

2. Emerging contaminants of concern.

Recent surveys of rivers, groundwater, lakes and coastal waters have highlighted the diversity of pollutants reaching these aquatic environments. Not surprising considering the multitude of chemicals released to the environment on a daily basis. Many new chemicals, so called emerging contaminants of concern have been highlighted, based either on their environmental concentrations and/or their specific chemical/biological properties. Many of these new chemical contaminants are persistent and bioaccumulative, others are known to influence sensitive biological processes. However, in many cases we have very limited knowledge regarding the impact(s) that these chemicals pose to the health of aquatic organisms and ultimately (through drinking water and aquatic food sources) to ourselves. Many pharmaceuticals and personal care products have been detected in water, including caffeine, ibuprofen, antibiotics, hormones and steroids (REF 8). The release of nanoparticles in the environment is also an unknown risk. They may or may not inherently be toxic but as already discussed above their physical/chemical properties may enhance the uptake of other chemical contaminants. We have come a long way, for example, in understanding the basic toxicity of heavy metals; that toxicity is not based on the total amount of metal but rather it's specific chemical form (species). The question now is size also a critical factor? Last year a summer undergraduate research project (NSF, REU student) in my laboratory in collaboration with others at the Chesapeake Biological Laboratory demonstrated that nanoparticulate forms of copper were more toxic to larval frogs compared with their dissolved counterparts, however, larval salamanders were not impacted. These results reflect the differing routes of exposure for copper uptake by herbivorous species (results from this study can be provided if requested).

Coastal sediments have also been shown to contain an array of pollutants, that through benthic organisms and food web interactions may influence a variety of organisms, including us. I will use the polybrominated diphenyl ether flame-retardants (PBDEs) as a case study example for this topic of emerging contaminants of concern. Also included are issues relating to some of the new flame-retardant formulations.

Case Study (1) – Flame retardants.

Undoubtedly the use of flame-retardants has saved hundreds of lives across the U.S., but at what environmental cost? Since PBDEs were first detected in environmental matrices and in human breast milk an exponential increase in research papers has demonstrated that these chemicals are persistent, bioaccumulative and toxic products. Although we really shouldn't be too surprised by this. PBDEs, especially some specific congeners (e.g. BDE-47) show a striking resemblance to natural thyroid hormones (which are part of the endocrine system; see figures within REF 9). These PBDEs have been shown to mimic or disrupt the normal functioning of these natural hormones in many species. The normal functioning of

these hormones is critical in maintaining basic metabolic processes, growth, and development. Additionally, the endocrine system is also tightly coupled with the neurological and immune systems.

In collaboration with Dr Heather Stapleton (Duke University) we have demonstrated the bioaccumulation and metabolism of these compounds in fish and suggested a novel metabolic pathway by which these compounds are converted into potentially more toxic products (REFS 9-11). This metabolism involves the enzymes responsible for the maintenance of normal thyroid levels.

In response to the overwhelming evidence describing the environmental persistence and toxicity of PBDEs, many of these flame retardant formulations have been phased out with the final, and most highly used formulation (deca-BDE) due to be phased out in the U.S. by 2013. Despite this encouraging news, unfortunately PBDEs, just like the historic polychlorinated biphenyl (PCBs), will be around polluting our waters, sediment, aquatic organisms and ultimately ourselves for years to come.

Fire retardants are required to be used in consumer products. So if not PBDEs what are the chemicals being used? PBDEs are not the only flame-retardants; in fact many alternate products have been in use for a number of years. Two of these formulations are currently being studied in my research laboratory (again in conjunction with Dr Stapleton's laboratory). Firemaster® 550 and Firemaster® BZ-54 are two brominated formulations containing mixtures of chemicals (e.g. TBB and TBPH; see REF 11 for complete chemical names and structures). Both of these chemicals have been measured in environmental matrices including house dust, biosolids and sediments, however, scant toxicological information for these chemicals exist. The only existing toxicological data for TBB and TBPH are the standard acute aqueous toxicity tests (i.e. 96 hour LC50s) summarized on the Material Safety Data Sheets (MSDS) for these formulations.

Are these chemicals bioavailable to aquatic organisms? Do they bioaccumulate, do they have sublethal effects? We have recently shown that these formulations when fed to fish, are bioavailable and bioaccumulate in fish tissues. Furthermore, they are metabolized to various alternate compounds and causes DNA damage in exposed fish. (REF 12). The question remains are we trading off one toxic product for another? How often are these trade offs going to continue? We need to compare new chemicals before their release, not just with other known toxic chemicals but also with natural substrates. There are numerous examples of current chemical contaminants that are structurally very similar to natural thyroid hormones. Thyroid hormone disruption seems to be a common thread for many emerging contaminants.

Often the original, or parent chemical contaminants, are not highly toxic themselves but it is their breakdown products or metabolites (that are not looked for and often unknown) that can be highly toxic. There is a need to fully understand how pollutants are metabolized in aquatic species to fully assess their fate and effects in aquatic ecosystems. Determining the specific metabolic pathway in multiple species together with

understanding sublethal toxicological effects (see below examples) is an ongoing (although unfunded) research project in my laboratory.

3. Sub-lethal toxicity issues.

The focus regarding a chemical's toxicity is often how acutely toxic it is, i.e. at what concentration does it cause death to individuals. But is death really the worse endpoint? Obviously, yes for that individual but not necessarily if we are looking at the bigger picture i.e. at the species, population and ecosystem levels?

Traditional toxicity tests use simple measures, e.g. LC50s (the concentration of a chemical that causes death to 50% of the organisms). These standard tests allow us to compare the toxicities of different chemicals and also the different sensitivities of particular species to the same chemical. These tests (called acute toxicity tests) are of short-term duration (i.e. 24-96 hours) and are of limited value in assessing the effects of pollutants that remain in aquatic systems for long-periods of time (persistent contaminants) or for those that are continually being released at low-levels. Additionally, these tests are usually carried out with single chemicals.

In recent years there have been great advances in further developing standard toxicity tests that assess sublethal effects (i.e. endpoints other than death). Longer-term exposures (weeks to months) of lower (and often more environmentally relevant) concentrations of chemicals (again usually in isolation) are used and sublethal impacts assessed using endpoints such as growth and reproduction. Derivations of these tests, whole effluent toxicity (WET) tests are applied to assess the overall toxicity of effluents and other point source discharges (i.e. assessing the combined toxicities of unknown chemicals and other stressor mixtures). My laboratory together with other colleagues at the Chesapeake Biological Laboratory have run many of these tests to assess questions such as the toxicity of ballast water treatment options (REF 13). These tests are very useful in providing an overall look at potential toxicity to aquatic organisms although they cannot be used to pinpoint the causative agent(s).

Reductions in growth and reproduction are used in these chronic tests as these endpoints may translate to a species reduction in numbers at the population level. Indeed these are endpoints that can be used in population models to predict the potential effects of a chemical contaminant. These endpoints integrate the complex interactions that chemical contaminants may have at the molecular/biochemical and physiological levels.

Studies of chronic toxicity are still in their infancy and are highlighting the numerous subtle ways or mechanisms of action in which pollutants can negatively affect organisms. Our understanding of the complexities of contaminant interactions with organisms has evolved given our increasingly sophisticated forensic toolboxes. However, many more studies are needed to fully investigate these issues, allowing us to group classes of chemicals together that display similar physiological, biochemical mechanisms of action.

For example, chemicals that impact the normal functioning of the endocrine system are called EDCs or endocrine disrupting chemicals. This classification represents an integration of many numerous molecular/biochemical insults to the endocrine system.

Case Study (2) – Sub-lethal effect examples;

(a) Bioenergetic impacts: There are numerous subtle sublethal events that can result ultimately in population level declines. For example, an organism may cope with the toxicological insult by protecting itself through the up-regulation of one or more metabolic systems. However, organisms have a finite source of energy, if they use more of this energy to try to stay alive and cope with the toxicological insult then energy is directed away from growth and reproductive processes. These bioenergetic effects of chemical contaminants are a focus of research at the Chesapeake Biological Laboratory. For example, bioenergetic assessments in shrimp exposed to Baltimore harbor (MD, U.S.) sediments demonstrated elevated metabolic rates that translated into lower lipid contents (food reserves) and significant reductions in growth and reproduction (REF 14). In addition, this study, like many others, has raised issues concerning the importance of the maternal transfer of pollutants to developing and sensitive offspring. This transfer and novel exposure route of chemicals in the aquatic environment may be a significant way in which the young of a species are dying. This is particularly important in longer lived organisms and species higher up the trophic level that bioaccumulate much higher levels of contaminants compared with lower life span or lower food chain organisms. Increased body burdens (bioaccumulation) of chemicals are, in part, a reflection of the time and concentration of the chemical that an organism is exposed to. Maternal transfer occurs in mammals (including ourselves) by placental transfer and breast milk although egg-laying animals are particularly at risk. Persistent organic chemicals (e.g. PCB's, PBDE's) bind to lipids and are stored, often away from harm for the adult, but are then passed during reproduction into the yolk reserves in eggs for developing embryos. High levels of chemical contaminants have indeed been found in eggs (see reptile example REF 15).

(b) Behavioral toxicants: Aquatic organisms often have highly developed nervous, sensory and behavioral systems that are important for their timing of migration, reproduction, mating, in finding food and in predator avoidance. For thousands of years humans have been aware of chemicals that impact the brain and, for example, alter behavior. Aquatic organisms are also sensitive to certain pollutants in a similar way. Declining salmon populations off the West coast of the U.S. led to research investigating why this was occurring; was it pollution, if so, by what and how? Although this is still under debate one interesting article recently published demonstrated a potential link between salmon survival and urban stormwater runoff affecting their sensory systems (REF 16). Our development of coastal locations has led to increased pollution entering our coastal waterways and estuaries. Pollutants accumulate on impervious surfaces (e.g. roads) and are transported to aquatic habitats via stormwater runoff. Noteworthy is that during the first rains of a wet season these first stormwaters can carry huge loads of pollutants into local waterways that often contain reproducing organisms and their sensitive young offspring. This study demonstrated that the levels of copper often seen in estuarine

systems, impacts the olfactory system of juvenile salmon. They concluded that sensory physiology and predator avoidance behaviors of the salmon were significantly impaired. These fish populations may be decreasing simply because they are unable to avoid their predators. Recently pesticides were also implicated to negatively alter fish olfactory processes (REF 17), potentially influencing their ability to migrate to spawning grounds.

Case Study (3) – Oil and Oil Spill dispersants

Unfortunate recent events in the Gulf have once again brought to the forefront issues pertaining to the impacts and effects of oil, oil spill dispersants and dispersed oil in our coastal and marine ecosystems. I have focused much of my research (albeit recently limited due to funding constraints) trying to understand the effects of oil; it's components and oil spill dispersants on aquatic organisms (REFS 19-27). We are all fully aware that organisms can die if they are coated with, inhale or ingest large amounts of oil. Often these are the enigmatic species that are highlighted in the news; the oil coated birds washed onshore, the dead marine mammals exposed to the oil slick because they come up to the surface to breath. Oil coated shorelines not only decimate intertidal food reserves for ourselves (e.g. oysters, crabs, fish) and other organisms but cripple recreational activities and local economies. Sensitive coastal habitats, such as wetlands, often serve as nursery grounds to numerous species, including species that migrate long distances to these breeding areas.

When oil is spilled response decisions must be quickly made and are based on numerous and continually changing variables; what specific type of oil is spilled, how much?, what are the weather conditions (including oil trajectory), what response options are available and what and where are the sensitive habitats and species. Ultimately the question is often, what do I need to protect the most and what do I have available to do it? Using the recent Gulf oil spill as an example, decisions were made to protect the very sensitive coastal wetlands, that currently contain many species that are in their breeding season. To achieve this 100,000's of gallons of the dispersant, Corexit has been sprayed onto the open ocean slick to prevent it from coming ashore in huge quantities. This is an example of a known pollutant, albeit one classified as having low to moderate toxicity to environmental organisms, purposely added to the marine environment. It is used because its overall benefit to the environment offsets its risk. However, it actually represents an environmental trade-off, the protection of one habitat is at the cost of another. In this case the protection of shoreline species at the expense of organisms residing in the water column and potentially also those at the seabed given that some dispersants have been used to disperse oil at the source of the oil leak.

As many have asked in the past weeks, potentially what will the environmental consequences be of the applied dispersants, what will be affected, to what extent and how? This is impossible to predict for many reasons.

First, the sheer volume of dispersants applied is unprecedented; no spill in U.S. waters has used the amount of chemical dispersants that have currently been used. Additionally, dispersants are usually only applied to surface slicks, which is one of the main reasons why they are pre-approved for use in the open ocean. The movement of dispersed oil (and

dispersants) in all dimensions in such a huge volume of water (as in the open ocean) results in a plume of dispersed oil (and dispersants) that is quickly reduced to low levels i.e. at depths >10m water it is estimated that the concentration of dispersed oil is <12.5ppm (REF 18). In addition, this dispersal effectively increases the surface area to volume ratio of oil so that micro-organisms (bacteria) that naturally degrade oil can be more effective at doing so. However, organisms that live in the surface waters under the dispersed slick will now be exposed to dispersed oil (in addition to dispersants). Without dispersant application the oil slick would have floated over the top of these water column organisms and these organisms would not have been exposed. Although it should be noted that a small portion of the oil does dissolve in the water column and some droplets are also formed as a result of natural physical processes (i.e. from wind and wave action).

The second reason is that limited prior toxicological information exists to fully assess risks to exposed organisms. The majority of toxicity data regarding the dispersant Corexit 9500 and dispersed oil are those detailing acute and short-term effects (see summary tables in Chapter 5 of the 2005 NRC report, REF 27). Oil is a mixture of 100's of different chemicals all with their own specific physical, chemical and biological properties. Different oils contain different amounts of these individual components. In addition, dispersants contain mixtures, including proprietary chemical components so that we do not know exactly what the exact chemical make-up of the dispersed oil plume is. As summarized in recent NRC publications (see REFS 27, 28) oil and oil spill dispersants can cause a variety of effects, including death and a variety of sublethal impacts including reduced growth, reproduction, cardiac dysfunction, immune system suppression, carcinogenic, mutagenic and teratogenic effects and alterations in behavior. Some aquatic species are more sensitive than others to dispersants and /or dispersed oil (again see tables within Ref 27). However, often it is the early life stages, e.g. eggs and larvae that are at particular risk. These early life stages are at risk because of numerous reasons relating to their physiological and biochemical make-up but also how they are exposed to the oil (i.e. differing exposure routes) also plays a role.

There are still many unanswered questions that we need to know to fully assess the risks involved with dispersants and dispersed oil. These were highlighted in the 2005 NRC report (REF 27). Although this report was specifically tasked to address the potential risks of dispersant use in near-shore environments many of the conclusions of the report are valid in open-ocean spills (note in the Gulf example, these dispersants were applied in the open ocean in waters of greater depth). This report highlighted that there were many areas that lacked adequate research and in addition there were other areas of study in which conflicting data existed.

These include questions and issues, such as;

1. What are the potential-long term effects of dispersant and dispersed oil, even after a brief exposure, to aquatic organisms? What are the sublethal effects? Will there be delayed effects?
2. Limited studies on sensitive at risk organisms (see example on corals below).
3. Does dispersed oil reduce or enhance uptake/bioavailability of oil to organisms?
4. Does dispersed oil enhance microbial degradation?

5. Is dispersed oil less 'sticky' to biological surfaces and sediment?
6. What is the route of exposure to organisms to dispersed oil? Is it dissolved PAHs or the oil droplets, or both.

In the last few years my research group has in part investigated topics relating to questions 2, 3 and 6 (RFES 20-23). Knowing that there was a lack of information on the toxicity of dispersants and dispersed oil on sensitive species such as corals a series of laboratory experiments were conducted to investigate the effects of Corexit 9500 and dispersed oil (Corexit 9500 and weathered Arabian light crude oil, 1:25 ratio) on symbiotic cnidarians (anemones and corals). In summary, soft corals died in low ppm concentrations of Corexit 9500 (LC50 8 hours ~30ppm; LC50 96 hours <16.5ppm). Sublethal behavioral effects (narcotic response resulting in the cessation of coral pulsing) were observed within hours at low (10ppm) dispersant exposures. In attempting to mimic a dispersed oil plume moving through a coral reef the soft corals were exposed for 8 hours to dispersant alone (at 20ppm i.e. a 1:25 dispersant:oil ratio), dispersed oil (dissolved PAHs and oil/dispersant droplets and dispersant) and undispersed oil (i.e. dissolved PAHs under an oil slick) using an oil loading of 0.5g l^{-1} oil:water. After 8 hours of exposure these corals were placed in clean seawater to follow potential delayed effects and sub-lethal repercussions of exposure. After 32 days growth was significantly reduced in dispersed oil and dispersant exposures and delayed effects (further death in the dispersed oil treatments) were observed (see EXHIBIT 1). Our research also demonstrated that cnidarians accumulated PAHs from both the dissolved oil components and the oil droplets.

These results have been submitted to the funding agency in the form of a final report and peer-reviewed publications are pending. I will be happy to provide any further information on these subjects.

4. Ecosystem based approaches; Sensitive species and food web effects (indirect toxicity).

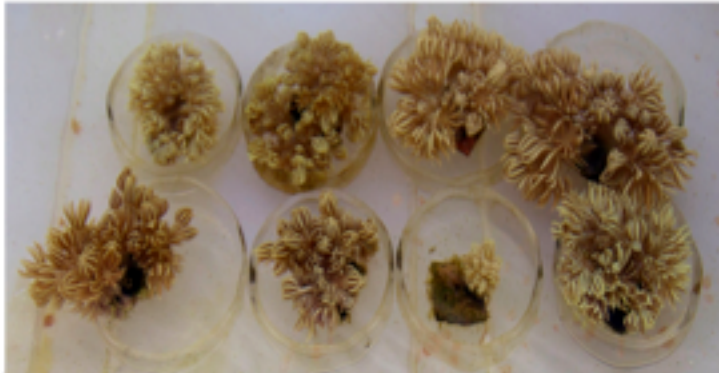
A species of interest does not need to be exposed and take up a chemical contaminant directly for that chemical to have a detrimental effect upon it. Contaminants alter food webs, for example, it may kill a lower trophic level species (e.g. phytoplankton or zooplankton). This has knock on effects for the more enigmatic higher trophic level species (e.g. fish). The fish is faced with a reduction in it's food source so that it may die from starvation. More subtly it may not grow or reproduce well as it has to expend energy travelling further distances to locate it's prey or it may be feeding off alternate food sources that are of lesser quality. Not only does this increased foraging for food have bioenergetic consequences but it may inadvertently make the organism more susceptible to predation given that it has to move out of it's usual habitat. Even subtle changes in food webs can have drastic effects on other organisms. Some organisms are much more sensitive than others, these species may not be the most enigmatic, but are none-the-less hugely important to ecosystem functioning.

We need to be protecting species at the foundation of the food chain and those that serve important ecosystem services. Coral reefs are a perfect example of this. Corals are the primary producers, in which the entire reef system depend upon. In addition, corals are extremely important for biodiversity, fisheries, coastline protection and local economies. The loss of corals will have huge impacts to our Oceans on a global scale. Corals are extremely sensitive to environmental disturbances (increased warming, ocean acidification, UV and pollutants) and have been a focus of research in my laboratory for the past 10 years. Again we still do not fully understand exactly how these species work, there are many biochemical and molecular pathways we do not know the significance of let alone how pollutant stressors impact them. For example, corals when stressed release large amounts of a biochemical compound called dimethylsulfide (DMS). This compound in open ocean algae has been stated to be responsible for over 30% of the global sulfur cycle, the significance of corals in this cycle is unknown but it may alter local climate. Dimethylsulphoniopropionate (DMSP) is the precursor of DMS that is found at high concentrations in coral symbionts. We are only just beginning to investigate it's role and function(s) in corals. It potentially may be extremely important in determining a corals sensitivity to pollutant stressors. Indeed, we have recently shown alterations in levels in copper exposed corals, which may reflect an antioxidant protective role for this natural chemical (REF 30).

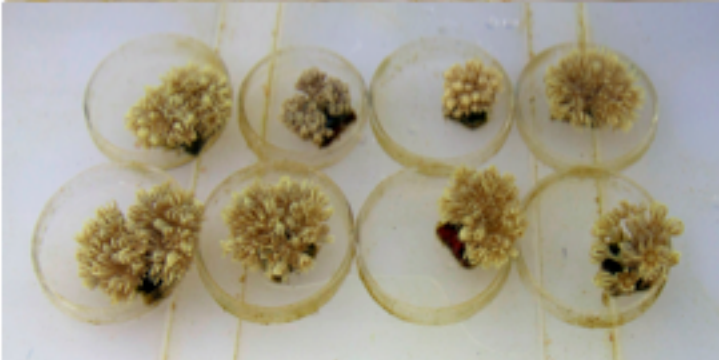
In summary

I would like to thank you again for allowing me to testify today on a topic I care deeply about. We face huge challenges to protect our coastal and oceanic ecosystems from the barrage of insults we, as a single species, have created. Pollution cannot simply be treated as 'out of sight out of mind' or that 'the solution to pollution is dilution'. The mind boggling huge numbers and types of diverse chemical contaminants, coupled with other interacting pollutant stressors has led to a 'toxic' soup for which we often do not fully understand the impacts of. New chemicals are released onto the market daily for which we have very little or no environmental toxicity data for. We are constantly unraveling, even for historic chemicals, new and more subtle sublethal toxicological pathways that ultimately have dire consequences to a species survival consequences of which, alter the fine balance of food-webs, alter ecosystem services, and the overall health of the environment. These events will impact our global economies, food sources, recreational activities and make our coastlines increasing sensitive to erosion. Human exploitation of species, habitat destruction and global climate change are impacting species, but let's not forget the often severe effects of our continued reliance and release of the thousands of industrial chemicals and other pollutants into these sensitive systems. We cannot even begin to understand some of their detrimental impacts and unless we act now the tipping point for the health of our coastlines and oceans may soon be reached.

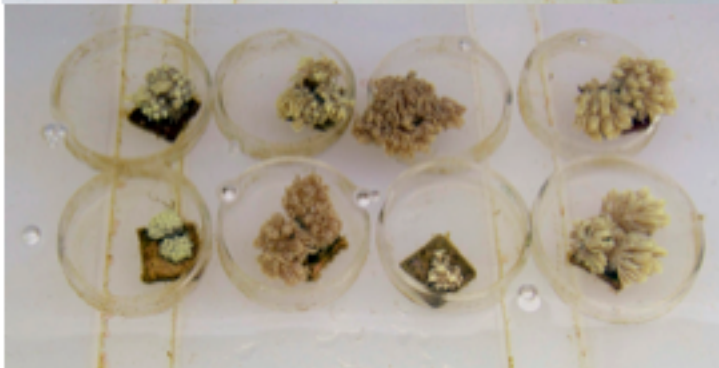
Exhibit 1: Photo depicting corals held in clean seawater 32 days after an exposure to Corexit 9500 and dispersed oil (using Corexit 9500 and weathered Arabian light crude oil). Significant reductions in growth were observed compared with controls.



CONTROL SOFT CORALS



**SOFT CORALS EXPOSED TO
COREXIT
9500 (20ppm, 8 hours).**



**SOFT CORALS EXPOSED TO
DISPERSED OIL (using 20ppm
Corexit (1:25 ratio dispersant:oil)
and 0.5g l⁻¹ weathered Arabian light
crude oil with 8 hour exposure).**

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