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Impacts of Metal Release on Marine Organisms During Offshore Gas Production

Petroleum production activities in the marine environment are often perceived as constituting a danger to marine biota. One of the main concerns is related to metal contamination resulting from production activities associated with the petroleum deposit. For example, mercury has been found in hydrothermal fluids used in extraction of petroleum.

A recent study was carried out to assess the impact of metal releases associated with gas production on biota in the Lower Gulf of Thailand, where the Bongkot Field is located.

In the study, the impact on biota was evaluated based on metal concentrations in finfish and on the composition of sediment fauna.

To assess contamination uptake, finfish collected in the vicinity of the main drilling activity were compared with the same species from natural populations in the region; and to assess habitat alteration, the structure and diversity of benthic fauna in sediment near the production platform were compared with those found in areas remote from the Bongkot Field.

Results of the study indicate that metal concentrations, particularly mercury, in species of snapper and grouper collected near the gas production platform were not significantly different from those of the same species of fish caught in non-impacted fisheries. Moreover no significant differences were found in faunal communities in sediments near the petroleum production activities and those from sediments in remote sites.

Although this represents only a small-scale study, the results may substantially alleviate public health concern over marine life contamination.

Source: Marine Pollution Bulletin, Vol. 36, No. 10, 1998.

The US Model for Environmental Regulation

Twenty years after the introduction of controls on acid rain in the United States, emissions of sulfur dioxide, the chief precursor of acid rain, are said to have halved. Moreover, this has been achieved at a lower cost to industry than had been originally forecast.

In the late 1980s, when it was thought that sulfur dioxide emissions – then totaling 25 million tons a year – would have to be reduced by 10 million tons a year, estimates of the cost were running from many hundreds to $1000 for every ton shaved off the total, or a staggering $10 billion a year. Those high prices were based on complying with the standard type of "command and control" emissions regulations, in which regulators made all the decisions. In the 1977 Clean Air Act, for example, regulators decided on a control technology – a "scrubber" that strips the sulfur dioxide from the spent combustion gases before they go up the stack – and they also decided which plants needed scrubbers.

When the US Congress contemplated the next round of emissions cuts, the $10 billion price tag triggered sticker shock. Instead of instituting ever more draconian and expensive command-and-control regulations, Congress took a new tack in the 1990 Clean Air Act Amendments: it commanded reductions but let power plant operators figure out the cheapest way to control emissions. The reductions were to come in two steps. Starting in 1995, 110 mostly coal-burning plants out of thousands in the country – then emitting about 4 pounds of sulfur dioxide per million British thermal units (mBtu) of heat – would be cut back to only 2.5 pounds/mBtu. In Phase II, starting in 2000, more plants are to fall under the plan and emissions will be tightened to 1.2 pounds/mBtu. The total release expected in 2010 is 8.95 million tons per year, a reduction of 10 million tons per year from the amount projected to be released without controls.

Congress made the rules even more flexible by authorizing a limited number of emission allowances, "right-to-pollute" coupons that could be bought, sold, or saved. Such trading with a cap on total releases means emitters are strictly accountable for the end result, but they have flexibility in the methods they adopt.

Meanwhile, Europeans have also been successful in reducing their sulfur dioxide emissions, halving them between 1980 and 1993, but they have not fully embraced trading. Instead European countries have adopted diverse approaches ranging from limited trading to pure command-and-control regulations. No one has calculated the costs of this mixed approach to date, but estimates for Europe's ambitious 2010 goals – to cut sulfur emissions damaging sensitive ecosystems by 60% – are quite high: about $1100 per ton of sulfur dioxide. As a result, Europeans are showing increasing interest in American-style allowance trading.

Source: Science, Vol. 282, No. 5391, November 1998.

Environmental Impact of Phytoremediation Technologies

Metal pollution in soil can affect yields of crops and adversely affect food quality. Even quite low concentrations of metals can interfere with the diversity of microbial communities in the soil and this in turn has serious effects on soil biomass and fertility.

Of the various technologies that have been developed for extracting metals and other hazardous wastes from soil, phytoremediation technologies show particular promise. Phytoremediation involves the use of certain plants to clean up sites contaminated with metals.

These plants, known as hyperaccumulators, provide an effective and affordable way of removing pollutants that also preserves soil fertility after the metal has been removed.

Research is currently being conducted into certain hyperaccumalating plants to determine their potential for removing a specific spectrum of metal pollutants occurring at a particular site.

Experiments have confirmed that some plants can take up zinc from the soil, and that the amounts removed by the hyperaccumulators were considerably greater than for normal non-accumulator crop species.

Thlaspi caerulescens, or Alpine pennycress, was the most effective species, removing the equivalent of 43kg of zinc per hectare per year. It was estimated that it would take nine harvests on the site under investigation to reduce the soil zinc concentration in the tilled part of the soil to an acceptable level of 300mg/kg of soil.

The chemical similarity of zinc and cadmium means that these elements are often taken up by the same hyperaccumulator species, though there is also evidence of some discrimination in their uptake. *T. caerulescens* accumulated cadmium at 150g/ha, as did *Cardaminopsis halleri,* which removed the equivalent of 35g cadmium/ha in a single harvest. While this amount is small compared with the amount of cadmium added to the site in sewage sludge, it is nevertheless equivalent to more than ten years input to soils in many industrial countries arising from the use of phosphate fertiliser and deposition from the atmosphere.

Copper and cobalt hyperaccumulators are also known to exist, and a thallium hyperaccumulator has recently been reported. Lead is a difficult metal to remove from soil because it is tightly bound by organic matter and soil minerals, and only a few plants have so far been found that accumulate this metal.

(Continued on page 8)

Screening Chemicals for Their Possible Effects as Endocrine Disruptors

In response to the uncertainty that exists with regard to the possible health effects of the group of chemicals known as endocrine disruptors, the US Environmental Protection Agency (EPA) decided to set up an Endocrine Disruptor Screening and Testing Advisory Committee, which in September 1998 issued its findings recommending that 15,000 chemicals should be screened and, if necessary, tested on animals for health effects.

The EPA has accepted these recommendations and has now prepared a regulatory screening process. Chemicals identified as endocrine disruptors include some pesticides, industrial chemicals, drugs and contaminants.

The EPA decision has been made in advance of the publication of a long-awaited report on assessment of endocrine disruptors by the US National Research Council (NRC), a report which is intended to provide a definitive assessment of endocrine disruptors for the US government.

The NRC study was commissioned in March 1995 and the panel of experts which first met in October of that year had expected to complete its work by mid 1997. However, this deadline has slipped, reportedly due to disagreements between experts with conflicting views. It is anticipated that the final version of the report when published will be cautious in its assessment of the extent to which endocrine disruptors pose a risk to human health.

Source: Nature, Vol. 395, No. 6705, October 1998.

Scorpion Venom Applied to the Targeting of Primary Brain Tumors

Gliomas are primary brain tumors that arise from differentiated glial cells through an as yet poorly understood malignant transformation. They are among the most deadly forms of cancer for which there are currently no effective treatment strategies. This is due in part to their high resistance to radiation and chemotherapy, and also to their unusual ability to disperse and invade healthy brain tissue.

Although glioma cells retain some genetic and antigenic features common to glial cells, they show a remarkable degree of antigenic heterogeneity and variable mutations in their genome.

In recent research studies, glioma cells have been shown to express a glioma-specific chloride ion channel (GCC) that is sensitive to chlorotoxin (CTX), a small peptide purified from *Leiurus quinquestriatus* scorpion venom.

Now a research study using native and recombinant ^{125}I-labeled CTX has shown that toxin binding to glioma cells is specific and involves high affinity and low affinity binding sites.

In radioreceptor assays, ^{125}I-labeled CTX binds to a protein with M_r = 72,000, presumably GCC or a receptor that modulates GCC activity. *In vivo* targeting and biodistribution experiments were obtained using ^{125}I- and ^{131}I-labeled CTX injected into severe combined immunodeficient mice bearing xenografted gliomas. CTX selectively accumulated in brain of tumor-bearing mice with calculated brain to muscle ratios of 36.4% of injected dose/g (ID/g), as compared to 12.4% ID/g in control animals. In the tumor-bearing severe combined immunodeficient mice, the vast majority of the brain-associated radioactivity was localized within the tumor (tumor to muscle ratio, 39.13% ID/g; contralateral brain to muscle ratio, 6.68% ID/g). Moreover, ^{131}I-labeled CTX distribution, visualized through *in vivo* imaging by gamma ray camera scans, demonstrates specific and persistent intratumoral localization of the radioactive ligand.

Immunohistochemical studies using biotinylated and fluorescently tagged CTX show highly selective staining of glioma cells *in vitro, in situ,* and in sections of patient biopsies. Comparison tissues including normal human brain, kidney, and colon were consistently negative for CTX immunostaining. These data suggest that CTX and CTX-conjugated molecules may serve as glioma-specific markers with diagnostic and therapeutic potential.

Given the high affinity of interaction between CTX and GCC, the specificity of binding of CTX-based moieties to human glioma cells, the small size of the CTX molecule, and its potential for chemical and genetic manipulation, this molecule seems to be an ideal candidate to engineer additional chimeric reagents to be used as diagnosic tools and therapeutic modalities. These approaches may offer novel strategies in addressing this uniformly fatal disease.

Source: Cancer Research 58, November 1998.

HEAVY METAL CONCENTRATIONS IN CARCINOMA OF THE GALL BLADDER

A group of researchers from the Institute of Medical Sciences, at Banaras Hindu University, Varanasi, India, have carried out studies to investigate whether cancer of the gall bladder might be associated with exposure to heavy metals.

Carcinoma of the gall bladder is the third most common malignancy of the gastrointestinal tract in the eastern Uttar Pradesh and western Bihar regions. These regions lie downstream of the river Ganges, which is the main source of drinking and irrigation water over a wide area. The river receives untreated domestic sewage as well as industrial and agricultural effluents.

High concentrations of cadmium as well as other heavy metals have been reported in sewage, irrigation water, and vegetables grown in the area.

In order to investigate whether gall bladder cancer might be associated with these heavy metals in the environment, the research team measured concentrations of cadmium, chromium, and lead in the gall bladders of 38 patients with gall bladder cancer and 58 with gall stones.

These patients were all admitted to the surgical unit of the University Hospital, Varanasi from January 1995 to March 1996. All were from the same geographical area.

Using histochemical techniques, the researchers found that the expression of metallothionein was 70% in the patients with gallbladder cancer and 25% in those with gall stones. The study showed that cadmium, chromium and lead concentrations were all significantly higher in carcinoma of the gall bladder than in gall stones.

Source: BMJ, Vol. 317, No. 7168, November 1998.

MYOTOXIC EFFECTS OF CHRONIC LOW LEVEL LEAD EXPOSURE

Lead toxicity is known to be linked in humans with motoric polyneuropathy (PNP) which develops with blood levels above 80 µmol and has been studied in various animal models.

Recent attention has focused on toxic myopathies since with increasing concern over environmental pollution, the etiology of some ideopathic myopathies has been elucidated by identification of the causative agents. Although studies have reported changes in the central nervous system and other organs as a result of low level lead exposure, until recently, there has been no investigation on the effect of low level lead exposure on striated muscles despite the fact that varying neuromuscular changes in different species has been known for some time.

A new study covers the investigation of striated muscles of rhesus monkeys chronically exposed to lead. In this study rhesus monkeys were exposed pre- and postnatally to lead acetate in the diet (350 ppm or 600 ppm) over 9 years, followed by a lead free period of 32 months, while a control-group received regular diet. No signs of muscular dysfunction were evident. To elucidate neuromuscular pathomorphology, frozen sections of the vastus medialis muscle were processed for routine and enzymohistological staining (Hematoxilin and Eosin, Sudan Black, Gomori, NADH, ATPase). Resin histology was processed for electron microscopy. Morphometric analysis was made with commercial softwere. Light microscopy revealed dose-related signs of myopathy in the lead-exposed groups. The scatter of fibre diameters was increased, and split fibers and internal nuclei were more frequent. Fibres became separated from each other by copious endomysial connective tissue. Ultrastructural examination showed hydropic mitochondria and a massively dilated sarcotubular system in the 600 ppm group. Dose-related extracellular collagen deposition increased. A heavy fibrosis was seen in the 600 ppm group. These findings are interpreted as myopathical reaction due to chronic low level lead exposure, as there were no signs of neurogenical lesion. It remains unknown how the fibrosis developed. A primary fibrosis could be based upon a developmental delay of satellite cells (expressing metalloproteases for collagen-catabolism). Lead is known to inhibit regular development in many ways if exposure has started prenatally. As the skeletal muscle is a common target of toxicity, the myotoxic effects of chronic low level lead exposure come into question.

Source: Neuro Toxicology 19(4-5), 1998.

The Toxicokinetics of Cadmium

Knowledge of the toxicokinetics of chemicals is an important basis for a better understanding of the nature and mechanisms of the adverse effects of chemicals both on human health and on the environment.

Experimental and epidemiological evidence has shown that cadmium has a long half-life. Depending on the different methods of investigation used, the half-life has been estimated between 15 and 20 years in humans.

Toxicokinetic studies of cadmium have revealed important data in our understanding of the toxic effects of the substance.

In acute and chronic animal experiments 5-20% of inhaled cadmium has been recovered in the lungs. Respiratory absorption in humans can be expected to vary from 2.5 to 50 per cent of the inhaled amount depending on particle size of the Cd-aerosol inhaled. The majority of particles deposited in the alveoli will be absorbed sooner or later depending on solubility. Data in humans showed that about 50% of the cadmium inhaled via cigarette smoke could be absorbed. In addition to particle size of the inhaled aerosol, the length and level of exposure, the chemical species of cadmium compound, and interspecies differences influence the three processes in the lungs, i.e. deposition, mucociliary – and alveolar clearance, and these factors should be taken into account in extrapolation models of inhaled cadmium between experiments and from animals to humans.

Exposure to cadmium via food and drinking water is an important route in the general population. Factors affecting the gastrointestinal absorption of the cadmium include animal species, chemical species of compound, dose, frequency of administration, nutritional status including iron status. Low iron status increases absorption of cadmium. Other factors important for cadmium toxicity are pregnancy and lactation, presence or absence of drugs, and interaction of cadmium with various nutrients. Gastrointestinal absorption of ingested cadmium is about 2 to 6% under normal conditions. It has been demonstrated that neonatal and young mice absorbed cadmium to a much greater extent than adult mice.

Cadmium absorbed from the lungs or the gastrointestinal tract is mainly stored in the liver and kidneys, where more than half of the body burden will be deposited. Highest tissue concentration is mostly found in the renal cortex. Exposure level may however influence the proportion of body burden that will be found in the kidney.

The role of metallothionein (MT) proteins in the kinetics of cadmium is well documented and the conclusion has been drawn that MT is the main transport and storage protein for cadmium.

Pulmonary deposition, clearance and effects of inhaled soluble and insoluble cadmium compounds have been intensively studied. Inhalation of cadmium leads to some biochemical changes. Animal experiment showed that intratracheal instillation of cadmium oxide induces MT synthesis, which reaches a maximum at 2 days, while reduced glutathione concentration increases slightly at 4 days, suggesting that MT plays a key role in detoxification of instilled cadmium oxide, but the antioxidants have a minimal role. Other observations suggest that glutathione could be an intrinsic protector against cadmium toxicity, while induced MT is involved in cellular defense as the consequence of toxic metal stress. The fact that cadmium aerosol pretreatment induces pulmonary tolerance to cadmium seems to confirm the protective role of MT. These events may be of importance in relation to pulmonary carcinogenicity of Cd. Data show that lung cancer develops in animals and in humans after inhalation of cadmium. The induction of lung cancer can be considered as the critical effect. However, it is uncertain whether a quantitative assessment of risk can be made.

The different absorption of Cd-MT and cadmium salts suggests that MT may play a role in the cadmium kinetics in the gastrointestinal tract. It was found that pretreatment with zinc (po or sc) induced MT in intestinal mucosa, which caused a reduction in the cadmium concentration in the liver and an increase in the kidney. Mucosal MT in the small intestine might trap Cd absorbed from the intestinal lumen and transport it to the kidney.

Upon exposure, cadmium causes multiple-system effects, for example, kidney dysfunction, lung diseases, calcium metabolism disturbance and bone effects. In preventive medicine, it is of essential importance to identify the critical effect and critical organ. The kidney is the critical organ for chronic cadmium poisoning, and critical effect is renal tubular dysfunction, which is most often manifested as low molecular weight proteinuria.

The various hypotheses on the pathogenesis of cadmium nephrotoxicity all assume a role of MT that is still being intensively studied, and there is an acknowledged need for further studies on the relationship between cadmium neurotoxicity and metallothionein.

Source: Neuro Toxicology 19(4-5), 1998.

Signalling Properties of the Calcium Ion and Apoptosis

Very high concentrations of the calcium ion, Ca^{2+}, in the human body can lead to the disintegration of cells (necrosis) through the activity of Ca^{2+} – sensitive protein-digesting enzymes. Calcium has also been implicated in the more orderly programme of cell death known as apoptosis. Apoptosis is important during both normal development (the formation of tissue patterns, for example) and pathological conditions such as AIDS, Alzheimer's disease and cancer. A protein that is mutated in cancerous cells, called Bcl-2, prevents the cell death that would normally limit the survival and proliferation of cancer cells. Bcl-2 mediates some of its anti-apoptotic action by modifying the way in which organelles such as the endoplasmic reticulum and mitochondria (where respiration occurs) handle Ca^{2+}.

With regard to the relationship between Bcl-2, Ca^{2+} signalling and apoptosis, in many cells, mitochondria participate in the recovery phase of normal Ca^{2+} transients – they sequester some of the Ca^{2+} signal, which is later returned to the endoplasmic reticulum. So, during normal Ca^{2+} signalling, there is a continuous shuttling of Ca^{2+} between these two organelles. Normally, most of the Ca^{2+} resides within the lumen of the endoplasmic reticulum, with very little in the mitochondria. These high levels of Ca^{2+} are essential. Not only do they form a reservoir of signal Ca^{2+} in the endoplasmic reticulum, but they are also essential for the synthesis and processing of proteins there.

Source: Nature, Vol., 395, October 1998.

Public Health Concerns from Disease in River Fish

In the brackish region of the St. Lucie Estuary around the juncture of Florida's Indian and St. Lucie rivers there has been a recent outbreak of disease in which fish are affected by open sores reminiscent of those associated with the toxic dinoflagellates *Pfiesteria piscicida*. Currently at least 33 species of fish are affected.

However, although the lesions mimic those of the *Pfiesteria* outbreaks that have killed large populations of fish in Maryland and North Carolina, there may be a different cause for the outbreak in Florida.

Water samples from the Indian and St. Lucie rivers revealed the presence of *Cryptoperidiniopsis* ("crypto") one of 10 recognized *Pfiesteria-like* species of microalgae.

Like *Pfiesteria,* crypto is a heterotrophic dinoflagellate that feeds on microalgal prey. However, whether it is the cause of the lesions and sores affecting the fish in the St. Lucie is not clear. Of the 2,000 known species of dinoflagellates, about 65 have been shown to produce toxins, and currently there is no evidence that crypto is toxic. Although studies are incomplete, crypto appears to have little, if any, effect on humans. However, from a public health perspective it is clearly important to determine the cause of the disease, and researchers are attempting to determine what has caused an ancient species of microalgae to strike the fish in the current outbreak.

Four times over the last 20 years, the St. Lucie Estuary has suffered outbreaks of fish disease. Each followed unusually high discharges from the St. Lucie Canal into the St. Lucie River, in which the South Florida Water Management District flushed fresh water from Lake Okeechobee through the canal to prevent flooding. There is an apparent causal relationship between massive discharges and the outbreak of ulcers. Nutrient runoff into the canal is believed to promote microalgal growth while the force of the discharges is thought to push the growth up from the bottom sediment.

Source: Environmental Health Perspectives, Vol. 106, No. 9, September 1998.

Problems of Dealing with Nitrate Pollution

Because nitrates are a natural and essential part of the ecosystem, they pose a problem for environmental protection. Nitrates are essential for the normal functioning of a healthy ecosystem, and only when present in excess can they be considered as pollutants. Deciding whether nitrate levels at a particular site are within expected, natural variation or are dangerously high is sometimes a difficult task.

However, nitrates are held to be responsible for destabilization of entire ecosystems, and many governments are taking action to deal with the problem of nitrate pollution.

Some US states have brought in integrated management programs to control the way that fertilisers – one of the prime sources of nitrate pollution – are applied. Countries that belong to the European Union are bound by a directive designed to protect public and environmental health by identifying "nitrate vulnerable zones" (NVZs) in which nitrates have reached such dangerous concentrations that plans must be developed to reduce inputs. Where public drinking water is affected, the directive demands that action be taken if nitrates exceed 50 milligrams per litre – the level deemed to be dangerous to human health.

Source: New Scientist, Vol. 161, No. 2170, January 1999.

Cadmium-induced Apoptosis in Mouse Liver

Apoptosis is a mode of cell death with morphological features quite distinct from those of necrosis which features in the toxicity of many chemical toxicants. It may precede necrosis or occur concurrently. DNA is often the target of toxicants, and DNA damage is currently thought to be the leading candidate for initiating the process of apoptosis.

Apoptosis provides a means for eliminating critically damaged cells without disturbing tissue structure or function and can thus be considered as a guard against genetic damage.

A recent research study has focused on the occurrence of apoptosis in Cd toxicity.

In the study, mice were injected with 5-60 umol/kg ip of Cd and their livers were removed 1.5-48 h later and examined by light microscopy. Cd induced both a time-and dose-dependent increase in apoptotic index, severity of necrosis, and mitotic index. Apoptotic index peaked at 9-14 h after Cd administration and then decreased. The time course of apoptotic DNA fragmentation index, monitored by quantification of oligonucleosomal DNA fragments, correlated with the results obtained by histopathological analysis and a commercial *in situ* apoptotic DNA detection kit. Liver necrosis, as demonstrated by histology and serum alanine aminotransferase and sorbitol dehydrogenase assays, was most severe 14-48 h after Cd injection. Apoptosis was decreasing by 24 h while necrosis persisted. Replacement of liver tissue by blood lakes (peliosis hepatis) was observed after 14 h. The mitotic index increased gradually with time, indicating compensatory liver cell regeneration. There was a progressive increase in the severity of necrosis, apoptotic index, and mitotic index with increasing dose of Cd. These data demonstrate that apoptosis is a major mode of elimination of critically damaged cells in acute Cd hepatotoxicity in the mouse, and it precedes necrosis.

Source: Toxicology and Applied Pharmacology, 149, 1998.

NATURAL REMEDIATION OF PCBS

Scientists hold widely divergent views about the future promise of natural dechlorination as a remediation technique. The process is not always reliable, in part because the dechlorinating microbes are unpredictable.

A number of different groups of bacteria that use hydrogen and acetate as food live in the oxygen-free depths of sediments. These anaerobes appear to breathe substances such as sulfate, iron (III), carbon dioxide, or chlorinated organic compounds. In the process, they reduce these electron receptors, converting them to sulfide, iron (II), methane – or in the case of chlorinated organics – to less toxic compounds. These unusual bacteria are at the end of a strange food chain in which other organisms break down and ferment organic matter to generate the hydrogen and acetate.

In this competitive environment, the "dechlorinators" are often less successful than other bacteria, so that their use in remediating contaminants must be evaluated carefully. To make reductive dechlorination work as a cleanup method, researchers either have to limit their choices to sites with the right conditions to give the dechlorinators a natural advantage or they have to figure out how to give the dechlorinators an artificial advantage. Approaches include finding a way to make them more competitive than other organisms or adjusting field conditions in their favor. Environmental conditions and bacterial types vary at every site, however, and obtaining desired results is difficult.

For some chlorinated solvents, the requirements for remediation are becoming clear. For perchloroethylene and trichloroethylene, which are the most common groundwater contaminants at many hazardous waste sites, there are now good natural attenuation prerequisites,

However, although a reductive dechlorination mechanism for polychlorinated biphenyls has been identified, dechlorination has been found to have little effect on the total mass of PCBs but causes a significant reduction of their overall toxicity. This is because the process selectively affects the most highly chlorinated PCBs which are also the most toxic.

Since 1991, General Electric has conducted field experiments in the Hudson River of the aerobic biodegradation of PCBs by naturally occurring microorganisms. However, although some success is claimed, the view of the US Environmental Protection Agency (EPA) is that dechlorination will not naturally remediate contaminated sediments.

To improve the effectiveness of the aerobic bacteria that degrade PCBs, researchers at the Center for Microbial Ecology at Michigan State University are turning to genetic engineering.

Naturally occurring aerobic PCB degraders only partially degrade PCBs and cannot be used as growth substrates. The researchers are trying to create aerobic strains that can grow directly on PCBs and degrade them. Such strains should be more competitive than naturally occurring PCB degraders.

Source: Environmental science & Technology, Aug., 1998.

OZONE DEPLETION AND HUMAN HEALTH

The depletion of ozone in the upper atmosphere has serious implications for human health. Ozone is thinning in the stratosphere because of exposure to manufactured chemicals. This is serious because it is the ozone layer that shields us from overexposure to ultraviolet (UV) radiation from the sun.

The **UV spectrum** is divided into three bands. **UV-C** (200 to 280 nanometres) is the most energetic and damaging, followed by **UV-B** (280 to 320 nanometres) and **UV-A** (320 to 400 nanometres). In the atmosphere, oxygen absorbs UV radiation at wavelengths below 242 nanometres, while ozone absorbs mainly in the range between 230 and 290 nanometres.

In this way, oxygen and ozone prevent molecules within living organisms, including humans, from absorbing too much UV. Industry uses UV-C to kill microorganisms on equipment and food. Fortunately, UV-C is absorbed by the ozone layer and does not reach the Earth's surface. UV-A, by contrast, is little affected by ozone, while most, though not all, of UV-B is absorbed.

The colour of our skin is the main factor determining how much we suffer from overexposure to UV radiation. Darker skins are richer in the brown pigment called melanin, which acts as a natural sun block by absorbing UV and so accounts for the much lower incidence of sunlight-related skin problems in people with dark skins.

People with paler brown or white skin are less lucky. UV-B contributes to suntanning, but also causes sunburn, inflammation and other severe photobiological effects. These include damage to DNA molecules, leading to mutation, and alterations to the structural and enzymatic properties of proteins. UV-A, which causes tanning or weak inflammation, is less dangerous, but exposure to it over many years is also thought to be harmful.

The most serious outcome of UV exposure is skin cancer – the most common human tumours. These arise after many years of high UV exposure, which cause mutations that gradually accumulate within cells and eventually lead to the formation of tumours. The UN Environment Programme estimates that globally, some 2.2 million skin cancers occur every year.

Research suggests that the incidence of skin tumours increases by about 5 per cent for every 1 per cent decrease in stratospheric ozone. For children the picture could be graver: some estimates predict that the lifetime risk that they incur today could be from 10 to 16 per cent greater than if there were no ozone depletion.

Source: New Scientist, No. 2164, December 1998.

Environmental Impact of Phytoremediation Technologies

(Continued from page 2)

Mercury also is strongly bound to soil and is therefore not readily available to plants; indeed no accumulators are known. However, in research being carried out at the University of Georgia, researchers have put a modified bacterial gene for an enzyme that reduces mercury ions into Arabidopsis thaliana (thale cress), and the transformed plants can reduce mercuric ions into elemental mercury.

This research demonstrates the feasibility of manipulating metal chemistry to achieve decontamination via phytotransformation. However, although the use of metal hyperaccumulator plants to clean up polluted sites is certainly feasible, there are a number of problems that need to be addressed before phytoremediation technologies can be widely applied.

Plants with hyperaccumulator properties are usually located in sites of naturally high metal content. There is a need to multiply these plants and to protect any genetic variations that give rise to patently useful traits.

There are opportunities to improve further the agronomic and biochemical traits of metal hyperaccumulator plants, either by conventional breeding or by genetic engineering. Many of the European hyperaccumulator plants are relatively small and slow-growing. The selection of faster growing strains and the production of greater biomass would seem to be obvious targets to increase metal uptake. Growing larger plants would also assist the harvesting process, by making it easier for it to be done by machinery. Even more important than biomass, however, is the need to achieve metal concentrations in the plants that are orders of magnitude greater than in ordinary plants.

Factors limiting metal bioavailability and uptake need to be addressed to predict and guide future applications. This will involve careful site characterisation, including analysis of soil features that affect metal ionic form and mobility. The complex interactions and especially the transport processes that occur at the soil/microbial/root interfaces and within the general soil ecosystem will require detailed modelling, especially when phytoremediation is scaled up from the laboratory to the field. Further practical considerations will be influenced by the knowledge that most contaminated sites will have multi-metal rather than single metal toxicity problems.

The environmental impact of phytoremediation technologies must not be overlooked. For example, livestock must be prevented from eating vegetation that has high concentrations of toxic metals, and harvesting must be timed to minimise the return of shed leaves with their toxic metals in bioavailable form to the soil. The growth of non-palatable forms is likely to reduce the chances of food chain transfer during phytoextraction.

Consideration must also be given to disposing of the harvested metal-rich plant biomass. This could be dried and put in landfill or burned to produce a more metal-rich ash. Little work has been done on methods for recovering metals from biomass of ash so far. One option is to use controlled, low temperature ashing to concentrate the metals further, followed by electrolytic metal recovery. Alternatively, methods to remove the metals that are present in predominantly soluble form in the tissues could be considered.

Many practical and research challenges remain to be met before phytoremediation can be used on a large scale to decontaminate metal-polluted soils.

It is, however, an approach that carries exceptional promise for cleaning up polluted sites, and perhaps even more importantly for safeguarding soil fertility.

Source: Chemistry & Industry, 16 November 1998.

Announcement

CRI Project: Capacity Building in Environmental Toxicology and Management to Promote Sustainable Development in Asia and the Pacific

Work Plan – 1999

There is one change to the dates announced in the January 1999 issue of the Newsletter.

This change applies only to the *Training Workshop on Risk Assessment and Management in Biotechnology* which has been rescheduled as follows:

Date	Course	City/Country	Registration Fee
December 3-9	Training Workshop on Risk Assessment and Management in Biotechnology	Bangkok/ Thailand	US$ 400

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