



**CRI/ICEIT
NEWSLETTER**

VOL. 4 NO. 3 – July 1994
ISSN 0858-2793
BANGKOK, THAILAND

Chulabhorn Research Institute

INTERNATIONAL CENTRE FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

CRI's ICEIT has been designated as a
"UNEP Centre of Excellence for Environmental and Industrial Toxicology".

New Technologies Improve Environmental Performance in the Chemical Industry

The development of technologies that allow efficient production with little or no environmental damage is the goal of environmental technology, the branch of engineering concerned with improving the environmental performance of industry.

A survey conducted by Chemistry & Industry gathered information on examples of novel environmental technologies applied in recent years in the chemical industry. The survey instances technologies for removal of hydrocarbons from water, recovery of sulfuric acid, recovery of antimony, reduction of hydrocarbon emissions and benzene waste reduction as technologies that both improve environmental performance and the industry's profitability margin.

Removal of hydrocarbons from water

Akzo Nobel of Japan has developed a technology in which macroporous polymer particles (MPP) are used to remove hydrocarbons from ground and process water. The type of particles needed for water purification depends on the type of hydrocarbon to be removed.

Hydrocarbon droplets can be absorbed in the pores of polymer particles, or particles containing a suitable extraction liquid can be applied to absorb dissolved hydrocarbons.

Among the advantages of MPP systems are the effective removal of dispersed hydrocarbons, the low effluent

concentrations produced, the removal of non-volatile hydrophobic components, low energy consumption and lower investment and operational costs.

Recovery of sulfuric acid

A new production system installed in December 1993 by Asahi Chemical Industry's Kawasaki Works in Japan involves closed system production of the methyl methacrylate monomer without a waste stream. This has been made possible by a new facility for recovering spent sulfuric acid. The recovery process adopted is a "wet process" based on the WSA process for sulfuric acid production licensed from Haldor Topsoe A/S of Denmark.

Conventional sulfuric acid production processes are generally "dry" processes, requiring cooling and dehydration of the combustion gas and therefore involving a long process train, high energy consumption, and secondary waste water. In the wet process, the combustion gas is fed directly to the de-dusting and oxidation steps, eliminating prior cooling and dehydration and resulting in a 30% shorter process train and 50% lower energy consumption.

(Continued on page 2)

New Technologies Improve Environmental Performance in the Chemical Industry

(Continued from page 1)

Recovering antimony

At Ciba's Schweizerhalle Works in Basel, Switzerland, a new process has been introduced to deal with the problem of waste water that contains considerable quantities of antimony as well as traces of some other heavy metals. (Such waste water results from the process of making additives for the production of polymer plastic.)

The new process involves cementation: converting the antimony using iron in the form of shavings or filings so that the antimony dissolved in the water precipitates as solid metal and the unproblematic iron dissolves.

The development of the process involved optimising the reaction conditions so that the antimony precipitated in as pure a form as possible, little iron or other aids were needed, the hydrogen gases formed by secondary reactions were kept to a minimum and, at the same time, the amount of antimony remaining was so small as to allow the waste water to be released into the sewer system.

The tests carried out so far have shown such success that the process can now be described as ready for industrial use.

The results show great advantages over the previous process from both an economic and an environmental point of view.

Reduction of hydrocarbon emissions

As a result of project at two of DSM polymers division's high density polyethylene plants in Geleen, The Netherlands, the plants' hydrocarbons emissions and catalyst discharges have been virtually eliminated.

In the final processing sections of the two plants, trace hydrocarbons are removed from the polyethylene powder in a fluidised bed. Until recently, the hydrocarbons were discharged to the atmosphere with the fluidisation air.

In the DSM method, the air leaving the fluidised bed, from which the polyethylene dust has been removed with the aid of large double filters, is sent to a combined heat and power (CHP) generator via a plastic pipeline. In the CHP unit this air is mixed with heated air supplied to the furnace for the generation of steam and power. The overall effect is a reduction in environmental impact to virtually zero, elimination of four emission sources and fuel savings in the CHP unit.

Benzene waste reduction

With the promulgation of the new organic toxicity characteristic regulations (OTC) in 1988, Union Carbide's Seadrift olefins unit in Texas needed to reduce the benzene in its unit wastewater steam before sending it to the plant wastewater treatment system. The unit stream contained about 0.2% benzene, 1.1% methanol, and other aromatic hydrocarbons, and was the source of over 150,000lbs/a of secondary emissions as the result of volatilisation of organic constituents from the wastewater, of which benzene was 113,000lbs/a.

The source of the wastewater was a condensed process stream, which separated as the process gas was compressed and cooled. Molecular sieve beds (furnace gas dryers) downstream of the C4/C5 splitter

removed the last traces of moisture before low temperature processing in the distillation train. Methanol was continuously injected into the C4/C5 splitter to prevent the formation of hydrates in the condenser section. Most of the methanol and water left with the kettle product from the C4/C5 splitter. The water/hydrocarbon mixture was decanted, but the water layer was saturated with benzene and methanol. All of the contained benzene and methanol had to be treated in the plant waste treatment ponds.

Five proposals were studied for benzene reduction. Four of the proposals continued to use methanol to control hydrates, requiring recovery or replacement or disposal of the contaminated methanol. The other proposal was to relocate the furnace gas dryers from downstream to upstream of the C4/C5 splitter.

After significant review, the furnace gas dryer relocation project was chosen. An additional dryer was required to handle the larger water load, and other equipment was needed for regenerating the dryers, and automating the regeneration process. The project cost \$5.5 million. Facilities were made operational in December 1991. Methanol injection to the C4/C5 splitter was stopped at the end of February 1992. Subsequently, methanol and hydrocarbon loading on the wastewater treatment system has been reduced by over 2.1 Mlbs/a (290,000lbs/a benzene and 1.8 Mlbs/a methanol), and the resulting secondary emissions have been reduced by over 150,000lbs/a (113,000lbs/a benzene and 24,000lbs/a methanol).

Source: Chemistry & Industry 11: 414-418, 1994.

HEAVY LEAD FOUND IN SOME FRENCH RED WINE

Dangerous organolead has been discovered in French red wine reports the July 7th issue of Nature. This particular wine was made from grapes that grew in vineyards bordering French autoroutes A7 and A9. The study of 19 vintages revealed levels of organolead concentrations of 10 to 100 times those of drinking water. The researchers are concerned with these findings because organolead is ex-

tremely dangerous to the body. It can easily be absorbed by the brain, is not easily eliminated, and can accumulate in the liver and the kidneys.

The researchers claim that organolead is absorbed by wine because it can bind to ethanol during the fermentation process. The researchers conclude that the use of leaded gasoline should be further regulated.

Health Risks From Incinerators Through Noninhalation Pathways

Although hazardous waste incinerators are designed to eliminate organic compounds with a high degree of efficiency, metals can be detected in stack emissions resulting in increased metal soil concentrations due to the metal laden particulates from ambient air in the vicinity of incinerators.

Previous investigations of potential public health risks associated with stack emissions from hazardous waste incinerators were limited to exposure due to the inhalation of toxicants; however, a recent study reported in Environmental Health Perspectives Volume 102, Supplement 2, June 1994 evaluates the potential health effects associated with arsenic, cadmium, lead, and mercury emissions due to human exposure through noninhalation pathways. The assessment of risk employed in this study involves an evaluation of human exposure to toxicants that could migrate into surface and groundwater, shallow soils, crops livestock and aquatic species, thus entering the food chain.

The emissions from nine incinerators in the United States were studied. The evaluation for each incinerator was conducted by assuming that it was located in a rural agricultural setting. In addition, potential effects on human health associated with the levels of metals in ambient air were evaluated.

Estimates of human exposure to metals due to the movement from soil into affected foods were based on estimating the changes from baseline dietary intake measured in U.S. Food and Drug Administration's (FDA) total diet studies. Baseline intakes of cadmium, arsenic and mercury were based on adult values since sensitive toxic effects are associated with long-term low levels of exposure, while the baseline levels of exposure to lead were based on the lead intake of preschool children since this age group appears to be highly sensitive to the adverse effects of lead. The results of the study indicate that in certain circumstances, incinerator emissions could tangibly contribute to aggregate human exposure to specific metals; however, the increase in soil metal concentration does not necessarily result

in an exposure that would adversely impact human health.

While the findings of this study indicate that the stack emissions from most incinerators would not significantly contribute to human exposure to metals through noninhalation pathways, siting of the incinerating facility

elsewhere may result in different findings. The methodology used in this study should facilitate future assessments.

Source: Environmental Health Perspectives 102 (Suppl. 2): 105-112, 1994.

A HIGH FLYING FIX FOR OZONE LOSS

Alfred Wong a UCLA plasma physicist believes his findings in a chemistry lab may be able to help him design a scheme which will restore the stratospheric ozone.

In their experiment, Wong's group monitored a "simplified stratospheric brew," which contained CFCs (chlorofluorocarbons) and low levels of ozone molecules. Ultraviolet light was used to create the ozone molecules which were subsequently depleted by the CFCs. Electric charges were then injected into the coffin-sized chamber. The result was a sharp increase in the ozone levels. It was suggested that the electric charges produced inactive chloride ions by ionizing the active chlorine atoms, resulting in stoppage of the ozone destroying process.

Wong's plan would involve flying 10-20 balloon propelled platforms carrying the ozone restoring panels into the antarctic and arctic stratospheres. These panels would then have many of their electrons dislodged by ultraviolet light. The resulting electron charges could then convert chlorine atoms into virtually inert chloride ions which would then

easily be collected by other, positively charged surfaces.

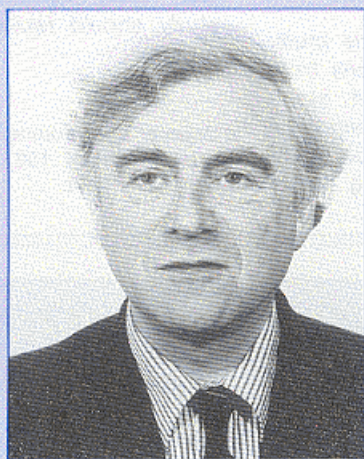
While most atmospheric chemists believe in Wong's lab findings, few believe that the results of the experiment can be projected to a large scale scheme which would restore the ozone layer in the environment. Wong's critics point out that 20 times as many charges as were needed to deactivate the chlorine atoms had to be injected into the tank in the controlled experiment. These critics wonder what other molecules in the environment might absorb these extra electrons and how this would affect the environment. Other critics believe that even if inactive chloride ions were formed, they might in turn form chlorine molecules which could be blasted apart by solar radiation producing ozone depleting chlorine atoms once again.

Remediation of the stratosphere is extremely important and lab proven experiments like Wong's offer hope, but most experts believe we are a long way from accomplishing the task.

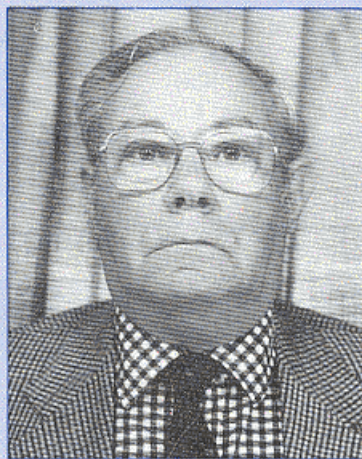
Source: Science 264: 1401-1402, 1994.

Environmental Toxicology – A Model for Research Management

*John H. Duffus and Morrell H. Draper
The Edinburgh Centre for Toxicology
Heriot-Watt University
Edinburgh, Scotland, U.K.*



John H. Duffus



Morrell H. Draper

Since the 1970's when the recognition of the dangers of the indiscriminate release of chemicals into the work, domestic and general environments began to be appreciated, there have been enormous developments in the scientific and technical approaches to the health problems posed by chemicals in the environment. The realization that many relatively simple chemicals, some widely used and in very large quantities (e.g. vinyl chloride), could pose serious health risks, such as cancer, brought about a demand for the testing of all dubious chemicals, particularly newly synthesized chemicals entering the market place. It was soon recognized that this was an enormous task that could only be attempted by an international collaborative effort. International agencies were created to avoid duplication of testing efforts and to ensure quality of test data, so that data generated at any recognised centre would be internationally acceptable. To this end a series of protocols were developed by the Or-

ganization for Economic Co-operation and Development (OECD) to ensure "Good Laboratory Practice" and "Quality Assurance Auditing". These protocols must be applied to any data submitted to a regulatory agency.

Collaborative efforts were necessary because the quantity of data needed by regulatory bodies greatly exceeded the available testing capacity of even the most advanced industrial nations. The many scientific disciplines involved in testing programmes also meant that very few research institutes or university departments were sufficiently well endowed to offer even a partial programme of test procedures. Further, few such laboratories were interested in routine testing activities. Hence there was in the 1980's a big expansion in commercial institutions that were devoted primarily to the analysing and testing of chemicals.

The commercial supplementation of the toxicological resources of governmental and university institutes has served reasonably well for

assessment of the potential health hazards of individual chemicals and of exposure to such chemicals in the industrial, domestic or general environments. However it has not been satisfactory in meeting the growing concerns about general environmental pollution hazards. Here the basic problem is that exposures are always to complex mixtures of substances, many of which may not have been fully characterised chemically. Assessment of such environments is in its infancy and the sophistication and complexity of the techniques required for this purpose are now beyond the resources of any single institution.

The key science in assessing chemical hazards in the environment is environmental toxicology which is essentially a multidisciplinary science without clear boundaries. At its heart is toxicology, but toxicology in its widest sense encompassing all living organisms and their ecological interactions. Adverse effects

considered must include effects on the environmental media - air, water, soil or sediments. Thus meteorology, geology, hydrology, and geography become important considerations in addition to the more obvious subjects of physics and chemistry which have always been of importance to toxicologists. However, even this is not a sufficient background of knowledge, because most of our environment is in its present state as a result of human activities such as agriculture and industry and reflects the consequences of our environmental management decisions influenced by politics, law, economics, culture and other social forces. Considered in this way, it can be seen that it is impossible for any single institute to cover all the necessary expertise for the broadly based research needed for full assessment and understanding of a problem in environmental toxicology.

Faced with the problems outlined above, it is clear that the traditional concept of specialized institutes working independently is not the best basis for tackling environmental problems. An institute of environmental toxicology must, like other institutes, have a group with expertise in both breadth and depth but it must also have the management skills needed to integrate contributions to its research from a wide range of collaborators. Since such contributions must be given appropriate financial support, the institute of environmental toxicology becomes to some extent a funding body and must incorporate the correct financial management as part of its activities. A model may be found for this in the research the Edinburgh Centre for Toxicology has been carrying out on the cause of lung and nasal cancers at the Clydach Nickel Refinery.

The Clydach Nickel Refinery was associated with a very high incidence of lung and nasal cancers, initiated by exposures during the first quarter of this century. The nature of these exposures was never determined and in assessment of the epidemiological data it was assumed that the carcinogenic materials present must be nickel itself, nickel oxides and/or nickel salts. In hindsight, it seems strange that in industrial situations where the products of the refinery, such as nickel metal,

nickel powder, or nickel salts, were used, often in large quantities, there was and is no evidence of the presence of any powerful carcinogen. It is also of note that those recruited at Clydach after 1924 to the refinery where the epidemic occurred were clearly at greatly reduced risk in spite of the fact the exposures to nickel metal, nickel oxide and soluble nickel salts continued for decades.

In order to investigate the Clydach epidemic further, Dr Morrell Draper, our Chief Medical Consultant, visited Clydach and made contact with Dr Lindsay Morgan, the physician responsible for occupational health at the INCO refinery. He was given access to all the relevant records, and some samples of process materials relating both to a year when cancer initiation was at its peak and to a year when initiation was low were discovered. These samples were characterized by laser ionization microprobe analysis in the Department of Chemistry at Heriot-Watt University, by X-ray crystallography at the Department of Chemistry at the University of St Andrews and the Department of Geology, University of Edinburgh, and by elemental analysis carried out by a commercial laboratory. Study of the spectra by Dr Park in the Edinburgh Centre for Toxicology revealed a hitherto unsuspected material, a nickel arsenide complex in the form of the mineral orcelite, to be a major component of the process material associated with cancer initiation. Currently, Dr L. Levy of the Institute of Occupational Health at the University of Birmingham is investigating the mutagenic properties and transforming potential of this material in his laboratory.

Dr Draper has analysed the medical data in depth and, in a metademographic study, has reconstructed the work histories of those who died of cancer in relation to the layout of the refinery at the time of cancer initiation, taking into account the exact processes in operation, the throughput and the nature of the raw materials and products at all stages. This detailed analysis has been dependent on input from Dr Morgan above and Dr Metcalf, the chief refinery chemist at Clydach and has had support from NIPERA, the Nickel Producers Environmental Research Agency. Assistance with epidemiological analysis of the data has been obtained from Dr Raymond Agius in the Department of Public Health at the University of Edinburgh.

It will be seen that a total of eight separate units has contributed to the research on the Clydach epidemic and it may be that we shall have to recruit further partners as we investigate further in depth. We believe that this is a model of the way in which environmental toxicology research must inevitably develop because of the breadth of knowledge, skills and facilities involved. Thus, the key is the establishment of a management unit with senior personnel of considerable experience and the ability to define precisely questions that need to be answered and to identify and establish collaboration with units which can provide the answers to these questions. Collaboration will involve the issue of contracts for specific studies to the collaborating bodies. Such contracts will define objectives, costing and deadlines. Thus, cost-effectiveness can be assessed and controlled, even for tackling the most complex problems. The final task of the management unit is to produce an overall conclusion to the study. This approach means that tackling a problem is not restricted by the limitations of an individual institute or even of a single country. Good laboratory practice (GLP) and quality assurance audit (QA) requirements mean that there can be confidence in results from all approved institutions.

Management units require access to good libraries, information technology, and communication facilities, which means that they are likely to be most economically effective if associated with major universities where these exist. Otherwise they require only suitable accommodation. Once established, with good personnel, they can ensure, by intelligent contracting, optimal use of toxicological and related facilities wherever these are available and needed for the tasks on hand. Without the development of a management approach of this kind to utilise all the relevant facilities wherever they exist, environmental toxicology can never succeed in providing the broad based research platform essential for good decision making for the preservation of environmental health and for all that depends upon this. In fact, a similar approach is needed to solve any problem that has an ecological dimension.

Environmental Estrogens Stir Debate

A number of studies have been reported which have suggested that chemical pollutants could be damaging the reproductive systems of humans and wildlife. Recently, however, many of these studies have been called into question as they undergo careful scientific scrutiny. As a result, a heated debate has developed; some scientists believe that there is great reason to think that chemical pollutants could be damaging the reproductive systems of humans, while others believe the concern has been blown out of proportion.

The results of a number of studies which suggest that chemical pollutants could be damaging human reproductive systems are now being questioned. One study which implied the seriousness of chemical pollutants on the reproductive systems of humans caused great alarm because it showed that sperm counts decreased from 113 million per milliliter in 1940 to 66 million per milliliter in 1990. Reexamination of the data however (when considering 88% of the men in the study) shows that since 1970 sperm counts have actually increased. Another widely publicized study which claimed to link breast cancer to high levels of DDE (a breakdown product of the estrogenic pesticide DDT) has been questioned since additional studies have failed to confirm the findings.

The debate is not a new one. DES (diethylstilbestrol) which was used to prevent miscarriages in the 50's and 60's produced the first evidence that hormone-modulating chemicals could cause a problem to human health. DES was banned in 1971 because daughters of mothers who took DES were found to have a high incidence of vaginal cancer. Numerous synthetic and natural chemicals have since been found which go through the estrogen receptor to negatively affect the endocrine system.

One reason there is so much debate over the seriousness of the problem is that much of the evidence for this biological activity comes from animal studies where animals have been exposed to high levels of estrogenic pollutants. Many scientists doubt that the results of these studies can be projected to suggest what might happen in the natural environment.

Wildlife studies however seem to offer more convincing evidence. In one such study a 90% decline in the birth-

rate of alligators and possible decreased penis size has been linked to a DDT spill at Florida's Lake Apopka. Other wildlife studies suggest similar effects of hormone-modulating pollutants on a number of different species. A number of experts believe that since many of the animals in these wildlife studies have reproductive systems similar to the human reproductive system, humans exposed to hormone modulators are at risk for fertility problems.

Experts on the other side of the debate do not agree. Stephen Safe, Texas A and M University toxicologist, and others believe that there are two reasons why hormone-modulating pollutants do not present significant risks to human health. First, they say that a look at basic pharmacology suggests the improbability of danger to humans. Most pesticides and environmental estrogens are very weak estrogens which bind to the estrogen receptor hundreds to thousands of times more weakly than does estradiol. The body's own estradiol would thus most likely attach to the estrogen receptor blocking the environmental estrogens from attaching to the estrogen receptor.

Humans also have a mechanism which is different than most other mammals which may help to protect them from estrogens. In humans, estrogens are disarmed by the attachment of a sulfate group (or other conjugate), whereas rats and mice have a conjugate which specifically binds estradiol. Another reason Safe and others doubt the seriousness of hormone-modulators to human health is the acid-base argument. This argument claims that just as an acid and a base can cancel each other out, estrogens may be canceled out by natural and synthetic anti-estrogens which are present in the natural environment.

Safe's claims, however, have been highly criticized. Richard Peterson, University of Wisconsin toxicologist, and others do not accept Safe's acid-base argument. For one thing they say, anti-estrogens themselves have the potential to be potent hormone-modulators. Another criticism of the acid-base argument is that the body's response to estrogen may be heightened or muted by dioxin (a hormone-modulating byproduct of industrial processes) depending on where the dioxin is located in the body.

While the debate continues, both sides agree that more research needs to be done to determine conclusively if there's a connection between hormone-modulators and human disease.

Source: Science 265: 308-310, 1994.

DIOXINS MEDDLE WITH KEY THYROID HORMONE

Another potentially harmful effect of dioxins has recently been reported. Researchers report that babies' psychomotor development may be affected as a result of dioxin exposure to their thyroid systems. The study published in the November, 1993 issue of *Environmental Health Perspectives* reveals that infants who are exposed to greater than "normal" doses of dioxins have a heightened concentration of an important thyroid hormone in the blood. While the infants appeared healthy at 6 months, the researchers were still quite concerned about the long range effects of the dioxins on the babies' thyroid systems because of the importance of the thyroid system on growth.

The researchers measured the concentrations of seven dioxins and 10 dibenzofurans which were present in the breast milk of 38 mothers. Thyroid hormone concentrations and

other indicators of thyroid function in the infants' blood were then measured at birth, at one week, and at 11 weeks of age. Babies that were exposed to greater concentrations of dioxins and dibenzofurans showed higher concentrations of T4 (the hormone most synthesized by the thyroid gland) at 1 week and 11 weeks of age. Higher levels of thyroid-stimulating hormone were also present at 11 weeks in the babies who were exposed to higher levels of dioxins.

The researchers believe that dioxins influence thyroid hormone concentrations in infants by interfering with the thyroid hormone regulatory system. The researchers also suggest that dioxin may encourage the release of T4 in the pituitary gland. They are concerned that these effects on the thyroid regulatory system may jeopardize normal psychomotor development of the central nervous system.

Possible Risk of Endometriosis for Seveso, Italy, Residents: An Assessment of Exposure to Dioxin

In a recent study, Bois and Eskenazi attempted to compare levels of TCDD (2, 3, 7, 8-tetrachlorodibenzo-p-dioxin) found in humans (after a chemical plant explosion in Seveso, Italy) to levels found in rhesus monkeys exposed to TCDD in a controlled laboratory experiment. The results of this study could then be used to determine if the humans exposed to TCDD might be at an increased risk for endometriosis as was found to be the case for the monkeys.

Bois and Eskenazi, School of Public Health, University of California, used a toxicokinetic analysis which took into account species and exposure differences in dose and timing between humans and monkeys. The fact that concentration might change with time was also taken into account. The

results were based on measured internal blood or fat concentration data. Exposure was estimated via AUC (area under time-concentration curve). It was reported that after 4 years of exposure to 25 ppt of TCDD, the whole-weight adipose tissue concentrations were between 245 and 812 ppt in four of the monkeys in the study. Multiple-dosing kinetics provided the AUCs for adipose tissue concentration for these monkeys over a fourteen year follow-up study. AUCs fell between 350,000 pptxday to 1,400,000 pptxday. In the human study, the AUCs for whole-weight adipose tissue concentration for 1976-1993 were estimated to be between 1,660,000 pptxday and 112,000,000 pptxday.

According to Bois and Eskenazi's analysis, the humans were exposed to

larger amounts of TCDD than the monkeys, suggesting that humans exposed to TCDD at the Seveso accident may be at an increased risk for endometriosis. The researchers also believe that since monkeys exposed to only 5 ppt had an increased risk of endometriosis, populations of humans exposed to much lower levels of TCDD than those at Seveso may be at risk for endometriosis. The researchers further suggest that given their levels of exposure, the Seveso population would serve as a good population for further study to determine whether or not exposure to dioxin leads can lead to an increased risk of endometriosis.

Source: Environmental Health Perspectives 102: 476-478, 1994.

NEW RESEARCH FUELS SMOKING DEBATE

As concern grows pertaining to the dangers of smoking toward both smokers and non-smokers, new research related to both the dangers and the possible benefits of smoking rapidly emerges.

One danger of smoking recently reported by the American Cancer Society is that female smokers face a heightened risk of fatal breast cancer. In a study which included 880 women who had breast cancer, it was found that women who smoked 2 packs of cigarettes per day or more had a 74% higher chance of developing fatal breast cancer than non-smokers. Duration of smoking also proved to be a factor. Women who smoked 40 or more years were 25% more likely to die of a breast tumor than women who smoked between 20 and 29 years. Since research has not shown a link between breast cancer and cigarettes, the researchers suggest that smoking may only reduce survival for women who develop breast cancer.

Another danger of smoking that has recently been reported is that children of parents who smoke have an irreversibly reduced lung capacity. In a study reported in the June American Journal of Respiratory and Critical Medicine, 8,700 children between the ages of 6 and 18 years of age were examined. The study revealed that children exposed to tobacco smoke before the age of 6 had a 2 to 3 percent decrease in lung function. A follow up study conducted 12 years later showed that this reduced lung function still existed. The study also suggests that children who grow up in homes where one or both parents smoke have an increased chance of developing shortness of breath, persistent wheeze, chronic cough and phlegm, and bron-

chitis. Another study reported in the June 1st issue of the American Journal of Epidemiology showed similar results. In a study of 2,994 children between the ages of 7 and 11, it was found that passive indoor smoke correlated with respiratory harm to children. In yet another study which was reported in the June 15th issue of the American Journal of Epidemiology in which 8,863 children ages 8 to 12 were studied, a weak correlation between parental smoking and first-trimester harm to the fetal lungs was found.

While a number of studies suggest serious dangers associated with cigarette smoke, at least one benefit of smoking has also been recently reported. Cigarette smoking may relieve the transient effects of idiopathic Parkinson's disease reports the June 15th issue of the American Journal of Epidemiology. The reported study looked at 58 men who had Parkinson's disease over a 26 year period. The study revealed a 27% reduction in Parkinson's risk for each 10 years a man smoked. The explanation for this effect is that nicotine can stimulate production of dopamine (Parkinson's sufferers produce an insufficient amount of dopamine) in the brain.

The rapidly growing body of research suggests how serious a health debate issue smoking has become.

ADVANCE NOTICE

International Conference on
"Biotechnology Research and Applications
for Sustainable Development"
(BRASD)

August 7-10, 1995
Bangkok, Thailand

Organized by
Chulabhorn Research Institute
International Center for Environmental
and Industrial Toxicology (ICEIT)

Provisional list of invited speakers:

Environmental Biotechnology

- Prof. A.M. Chakrabarty (U.S.A.)
- Prof. S. Silver (U.S.A.)
- Dr. V. de Lorenzo (Spain)
- Dr. S. Farr (U.S.A.)
- Prof. P. Loewen (Canada)

Microbial Biotechnology

- Prof. G.C. Walker (U.S.A.)
- Prof. B. Demple (U.S.A.)
- Prof. P.S. Lovett (U.S.A.)
- Prof. Mel Simon (U.S.A.)
- Prof. J.H. Miller (U.S.A.)
- Prof. W. Goeble (Germany)
- Prof. D. Dean (U.S.A.)
- Prof. E.W. Nester (U.S.A.)
- Prof. J. Mattick (Australia)

Agricultural Biotechnology

- Prof. M.V. Montagu (Belgium)
- Prof. R. Dixon (U.S.A.)
- Prof. J. Ryal (U.S.A.)
- Prof. F. Chumley (U.S.A.)
- Prof. M. Zenk (Germany)
- Dr. T. Katchun (Germany)
- Dr. R. Walden (Germany)
- Dr. D. Inze (Belgium)
- Prof. A. Kondorosi (France)
- Prof. R.B. Flavell (U.K.)
- Prof. N. Doke (Japan)
- Dr. H. Klee (U.S.A.)

New Technologies

- Prof. W. Sybalski (U.S.A.)

Scientific program:

A keynote address and plenary lectures will be given by distinguished scientists followed by concurrent sessions of lectures and short presentations. Several poster sessions will be organized.

Theme:

The conference will cover both basic and applied research. Emphasis will be on the role of biochemistry, microbiology and molecular genetics in develop-

ing new technologies in the areas of agricultural, environmental and microbial biotechnology.

Topics:

Topics related to basic or applied research in biotechnology will be covered. Special sessions will be arranged in the following areas:

1. Agricultural Biotechnology

Development of transgenic plants, molecular breeding and rapid mapping techniques (RELP, RAPD), molecular mechanisms underlying important physiological processes. Plant/microbe interactions and plant diseases.

2. Environmental Biotechnology

Molecular aspects of microbial detoxification. Engineering of new degradative pathways, Biosensors, Biosafety and application of new technologies.

3. Microbial Biotechnology

Molecular genetics of important physiological processes, genetic manipulation of potentially useful microbes, novel host vector/systems, regulation of gene expression.

4. New Technologies

Development of new technologies in either microbial or eukaryotic cells which will advance knowledge in the areas of biotechnology, environmental science and medicine.

Venue:

Central Plaza Hotel, Bangkok, Thailand

All enquiries should be directed to:
Dr. Skorn Mongkolsuk,
Secretary General,
Chulabhorn Research Institute
Vipavadee-Rangsit Highway,
Bangkok 10210, Thailand
Fax: (66-2) 247-1222
email scsmk@mucc.mahidol.ac.th

ANOTHER EMASCULATING PESTICIDE FOUND

Abnormal sexual and behavioral development of male animals can result when a chemical blocks the activity of male sex hormones in the womb, reports the June 1994 issue of Toxicology and Applied Pharmacology. In this study, pregnant rats were exposed to up to 200 milligrams per kilogram of body weight of vinclozolin (a systemic fungicide) from the 14th day of pregnancy through the third day following the birth of each rat's litter. One year old offspring showed a number of reproductive abnormalities including feminization. Additional effects such as undescended testes, a cleft phallus, infertility, and hypospadias were also present.

The researchers believe that the abnormalities developed because male hormone receptors in reproductive tissue were blocked by the attachment of vinclozolin. The research suggests that an increasing incidence of undescended testes, low sperm production, and hypospadias in men may be due to chemical pollutants which mimic estrogen.

EDITORIAL BOARD

Skorn Mongkolsuk, Ph.D.
Mathuros Ruchirawat, Ph.D.
Somsak Ruchirawat, Ph.D.;
Jutamaad Satayavivad, Ph.D.
M.R. Jisnusun Svasti, Ph.D.

The ICEIT NEWSLETTER is published quarterly by the International Centre for Environmental and Industrial Toxicology of the Chulabhorn Research Institute, supported in part by the Asian Development Bank. It is intended to be a source of information to create awareness of the problems caused by chemicals. However, the contents and views expressed in this newsletter do not necessarily represent the policies of ICEIT.

Correspondence should be addressed to:

ICEIT NEWSLETTER
Chulabhorn Research Institute
Office of Scientific Affairs
c/o Faculty of Science,
Mahidol University
Rama 6 Road, Bangkok 10400,
Thailand
Telex: 84770 UNIMAH TH
Telefax: (662) 247-1222
Tel: (662) 247-1900