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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".

Her Royal Highness Princess Chulabhorn Opens Hanoi Training Course

12 November 2001

HRH Princess Chulabhorn presided at the opening of a training course on the Detection of Environmental Pollutants and the Monitoring of Health Effects jointly organized by the Vietnamese Department of Science Technology and Environment and the Chulabhorn Research Institute. The training course was held in Hanoi from 12 to 23 November 2001

In her opening speech, HRH Princess Chulabhorn stated that the course was the final activity of a highly successful training program under a UNDP supported project for Human Resource Development in Environmental Toxicology for Vietnam.

This program has been successful largely because of the close cooperation between the Department of Science Technology and Environment (DOSTE) and the Chulabhorn Research Institute. As a result of the program a total of 300 Vietnamese scientists have received training in courses held both in Hanoi and Ho Chi Minh City. In addition, 57 trainees from Vietnam have attended training courses organized in Bangkok. Faculty teaching on the present course included Dr. N. Frank and Dr. B. Spiegelhalder of the German Cancer Research Center.

ARSENIC AND BLADDER CANCER: TAIWANESE STUDIES

Significant associations between ingested arsenic and urinary tract cancers were reported in earlier studies made in an arseniasis-endemic area in southwest Taiwan. These studies showed that long term exposure to arsenic through water consumption increases the risk of cancers of the bladder and kidneys. However, because the number of wells shared by residents living in the villages in the study area was small, median arsenic levels in the well water were used to derive individual exposures to ingested arsenic. Thus cumulative arsenic exposure was estimated in an ecologic way which might result in nondifferential misclassification of individual exposure. Now, a new study has been carried out in an arseniasis-endemic region in the northeast of Taiwan where each household has its own well for obtaining drinking water. Since there was found to be a very wide range of arsenic concentrations in any given village (from undetectable levels to several hundred micrograms per liter), residents living in the same village had significant differences in levels of exposure to ingested arsenic. This was considered an important factor in the assessment of cancer risk associated with ingested inorganic arsenic.

The findings of the study showed that among residents of this arseniasis-endemic area in the northeast of Taiwan, there were significantly increased risks of developing cancers of the urinary organs and transitional cell carcinoma with standard incidence ratios of 2.05 and 2.82 respectively. However, no association was observed for the duration of well water drinking, indicating that it was the arsenic concentration in well water rather than duration of drinking the water which detemined the risk of cancer.

Source: American Journal of Epidemiology, Vol. 153, No. 5, March 2001.

Urinary levels of Arsenic in residents of villages in southern Thailand

The problem of environmental arsenic contamination in Ronpibul district of Nakhon Si Thammarat province in southern Thailand emerged in the late 1980s. Urinary levels of arsenic in residents of a village of the district were studied in the period 1999 to 2000 in a pilot study, in which 539 villagers participated. Urine specimens (single urine) were collected and tested for total arsenic. From the data obtained the means, medians and standard deviations of the urinary levels of arsenic were calculated.

Although detailed speciation of arsenic was not done in this study, urinary levels of inorganic arsenic have been estimated and were found to be quite high. Inorganic arsenic is considered to be more harmful to human health. If 50 per cent of the total arsenic is assumed to be organic, the average level of urinary inorganic arsenic will be 83 µg As/g creatinine. This is similar to the level of 88 µg As/g creatinine reported from a study of 17 people exposed to 0.014 mg As/L in Mexico. Thus, it is suggested that exposure to environmental arsenic is quite high and may account for human pathology including dermatological disorders, internal malignancies, and diabetes.

The present study underlines the need for further study of the association between arsenic and related diseases.

Source: Journal of Environmental Medicine, Vol. 3, No. 2, 2001.

The Fight Against Groundwater Contamination

Removal of contaminated water in polluted groundwater systems is often not possible because of the volume of water that needs to be pumped out and treated. A further problem is that contaminants continue to leach out after the contaminated water has been extracted and pollute more groundwater.

Barriers can be placed in the subsurface of polluted groundwater to treat the contamination *in situ,* but this is only economically viable in shallow and restricted areas of contamination.

A more feasible approach to the problem is a process called *in situ* bioremediation in which microorganisms degrade, detoxify, or immobilize contaminants in the groundwater system.

Until recently, practical applications of *in situ* bioremediation have focused mostly on aerobic microorganisms, which gain energy by oxidizing organic compounds to carbon dioxide, with oxygen serving as the electron acceptor. When oxygen is available in the subsurface, aerobes can clean up contaminated groundwater by oxidizing organic contaminants to carbon dioxide.

However, this approach has had limited success, not least because oxygen–an absolute requirement for aerobes–is scarce in many contaminated subsurface environments. The amount of oxygen dissolved in groundwater is low, and the rate of oxygen supply through diffusion from

(Continued on page 3)

ASTHENOSPERMIA AND EXPOSURE TO SOLVENTS: A CASE STUDY

A case study has been published of a 34 year-old laboratory worker in Taiwan who experienced elevated exposure to solvents resulting from the shutdown of the ventilation system in his laboratory during a nine month period. The subject reported that his wife had difficulty becoming pregnant.

The subject was married in December 1995. A complete fertility test was performed for the subject and his wife on 28 May 1996 in a local hospital. Results of tests for the subject and for his wife were within normal limits. The subject had normal semen appearance, volume, and sperm count. Ninety-two percent of sperm were normal in morphology. At 30 min after ejaculation, 95% of sperm were motile at a normal speed, and at 60 min, 30% were motile.

In June 1997, test results showed a reduction in sperm motility (asthenospermia) compared to the test from 1 year earlier.

This change occurred during the period in which the subject reported the shutdown of the ventilation system in the laboratory in which he worked.

The subject used infrared spectrophotometry (IR) to analyze the purity of petrochemical products. He received an average of 40-50 samples daily in 5-mL glass vials. Both before and after sample analysis, he was required to clean three types of IR specimen holders with isooctane, chloroform, and tetrahydrofuran (THF). The washing times for these three types of specimen holders were 25-60 sec with isooctane, 100-200 sec with chloroform, and 120-300 sec with THF, depending on the type of specimen holder used and the sample viscosity. The subject always wore gloves (polybutadiene latex) during this procedure. He also wore a respiratory mask equipped with a charcoal cartridge when he judged that the ventilation was not efficient. The charcoal cartridge was replaced on an irregular basis. However, the subject reported that he could still smell the organic solvents even when he wore the respirator. He began work at about 0830 hr, he took a break from 1130 to 1330 hr, and he worked until 1700 hr, a workday of approximately 6.5-7.0 hr.

Normally, the task of cleaning with solvents should be done in an exhaust ventilation hood. The subject started this job in October 1994, but due to pump failure during August 1996-April 1997, the ventilation system was shut down. The subject performed his routine procedures under the same ventilation hood, with the hood door open wide, and he used surrogate exhaust ventilation (a wall fan) beside the hood during the ventilation shutdown period.

Before the shutdown of the ventilation system, the subject's semen analysis was normal. Except for the ventilation shutdown, no other occupational or environmental hazards that were associated with the sudden reduction in the subject's sperm motility could be identified. Drugs, drinking alcohol, smoking tobacco, or surgery probably did not cause the condition because these were unchanged during the period of May 1996-July 1997,

After the ventilation system was repaired, the subjects sperm motility was improved. Because chloroform is known to damage sperm, the possibility of chloroform causing significant reduction of sperm motility cannot be ruled out. The shutdown of the ventilation system may be the explanation for the reduction of the subject's sperm motility. To protect workers from potential reproductive hazards, further investigation is needed to determine chloroform effects on sperm motility at various dose ranges, including dose levels near the permissible limits. Further human epidemiology studies or animal assays are needed to verify this hypothesis.

Source: Environmental Health Perspectives, Vol. 109, No. 7, July 2001.

The Fight Against Groundwater Contamination

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overlying unsaturated soils is slow. In subsurface environments polluted with organic contaminants, such as petroleum or leached materials from landfills, aerobes dutifully oxidize the contaminants to carbon dioxide, consuming the available dissolved oxygen in the process.

The scarcity of oxygen in many contaminated subsurface environments has raised interest in the *in situ* bioremediation potential of anaerobes, which grow in the absence of oxygen. Anaerobes also oxidize organic compounds to carbon dioxide but use electron acceptors such as nitrate, sulfate, or Fe^{3+} oxides instead of oxygen.

The diverse metabolic capabilities of anaerobes thus represent a potentially potent force in the fight against groundwater contamination.

Source: Science, Vol. 293, No. 5534, August 2001.

Training Workshop on Environmental Health Risk Assessment and Risk Management of Toxic Chemicals

3-11 December 2001

at Chulabhorn Research Institute, Bangkok, Thailand

The great majority of the participants at this training course came from overseas, with the countries represented being Cambodia, China, Indonesia, Mongolia, Myanmar, Philippines, Vietnam and Thailand.

This strongly regional participation reflects the emphasis CRI gives in its training program to the needs of the whole region, and to appropriate training in the skills essential to ensure sustainable development in all countries participating in the UNDP funded project for human resource development and capacity building in environmental toxicology technology and management. The program of the course covered 7 days of lectures and practical assignments based on case studies relevant to the situations of the participants in their fields of operation.

The topics included: Toxic Effects of Chemicals and Toxicity Testing; Risk Perception and Communication; Human Data and Epidemiology; Dose-response and Exposure Assessment; Risk Characterization, Management and Reduction; and Ecological Risk Assessment.

The case-studies presented by the teaching faculty were as follows:

Cancer risk assessment cases

Information for environmental health decision making

Dietary risk assessment cases

Persistent organic pollutants

Derivation of air quality

Guidelines/standard setting

Endocrine disruptors

The group of international experts comprising the teaching faculty were:

- Professor Herman Autrup

  Department of Environmental Occupational Medicine, Aarhus University, Denmark

- Professor Martin van der Berg

  Institute for Risk Assessment Sciences (formerly Research Institute of Toxicology), University of Utrecht, The Netherlands

- Dr. Kersten Gutschmidt

  International Programme on Chemical Safety (IPCS), Department of Protection of the Human Environment, World Health Organization (WHO), Switzerland

- Professor Leonard Ritter

  Department of Environmental Biology, University of Guelph, Canada and

- Dr. Maged Younes

  International Programme on Chemical Safety (IPCS), Department of Protection of the Human Environment, World Health Organization (WHO), Switzerland

The course co-ordinator was Dr. Mathuros Ruchirawat, Vice President for Research, CRI

TRAINING FELLOWSHIPS AVAILABLE

Training Course on Principles of Environmental Toxicology and Pollution Control 23 September – 4 October 2002, Bangkok, Thailand

A limited numbers of training fellowships (airfare and per diem) are available for trainees from ASEAN COUNTRIES. Candidates (belonging to governmental agencies or academic institutions) must have background in chemistry, biological/medical sciences, pharmacology or environmental engineering and related areas with work experience/responsibilities related to the control and use of chemicals in industry or agriculture or engaged in teaching related subjects. Candidates should also be fluent in English in all skills, particularly in writing and listening comprehension.

Written application and full curriculum vitae (CV) should be submitted by **31 July 2002** to Chulabhorn Research Institute, Office of Academic Affairs Vipavadee-rangsit Highway, Lak Si, Bangkok 10210, THAILAND Tel: (66-2) 574-0622 ext. 1602 Fax: (66-2) 574-0616, 247-1222 E-mail: vina@tubtim.cri.or.th

CONTENTS:

- Introduction to Environmental Toxicology
- Principles of Toxicology
- Target Organ Toxicity
- Pesticides and Industrial Chemicals
- Chemical Carcinogenesis
- Toxicological Basis for Regulating Chemical Exposure
- Health and Environmental Risk Assessment of Chemicals
- Ecotoxicology, Application to Risk Assessment
- Risk Management and Communication

A CENTURY OF DEVELOPMENT OF AGENTS OF BIOLOGICAL WARFARE

There is an increasing concern within both the scientific and security communities that the ongoing revolution in biology has the potential to be misused in offensive biological weapons programs.

Although it is not widely appreciated by the biomedical community, the historical record clearly shows a series of offensive biological weapons programs carried out by major states such as Germany, France, Japan, the UK, the USA, the USSR and Iraq during the 20th century.

These programs may be reasonably divided into three generations. (i) the relatively unscientific programs created during the First World War and between the world wars (ii) the science-based, effective programs created during and after the Second World War and (iii) the programs carried out toward the end of the Cold War, in which the USSR began making use of new genetic engineering techniques while the USA de-emphasized bioweapons. This history suggests that if the process is not halted, we will see a fourth generation of programs in the new century that will increasingly use knowledge gained from the genomics revolution.

Microbiologists who have looked carefully at the possible applications of currently available technology to offensive biological weapons programs suggest that it might now be possible to, among other things, enhance the antibiotic resistance of biological agents, modify their antigenic properties or transfer pathogenic properties between them. Such 'tailoring' of classical biological warfare agents could make them harder to detect, diagnose and treat. It could, in short, make them more militarily useful and thus increase the temptation to pursue offensive programs.

It has been argued by some that the availability of the human genome sequence will facilitate the development of biowarfare agents targeted to specific ethnic groups or individuals. Although this may not be impossible, for a number of reasons it seems extremely unlikely. Though genetic susceptibility to infectious disease has been described, the reductionist view that the human genetic code is the sole determinant of disease susceptibility is unsubstantiated. Analysis of the human genome sequence to date has failed to reveal any polymorphisms that can be used to absolutely define racial groups. In fact, genetic diversity in human populations is low relative to other species, supporting the notion of the recent origin and small size of the ancestral human population. A number of recent studies of polymorphisms in mitochondrial, Y-chromosome and autosomal DNA have shown that most human genetic diversity exists within, rather than between, populations. A greater current concern must be the limited genomic variation in our staple crops and animals than makes agriculture such a vulnerable target for biological attack.

Source: Nature Genetics published online October 2001 http://genetics. nature.com .

Air pollution inside vehicles – health effects of traffic congestion

The health effects of sitting inside a vehicle in dense traffic for long periods remain poorly understood. Many of the pollutants in the air inside a vehicle are suspected to be human carcinogens, and others may affect the neurologic, immune and reproductive systems. Some may disrupt the endocrine system.

While pollutant concentrations inside vehicles are usually well within those levels found in outside air, health evaluations are difficult because there are no established standards for inside vehicle air.

In a 1994 investigation in South Korea, researchers measured concentrations of benzene, toluene, ethylbenzene, *m/p*-xylene, *o*-xylene, and total volatile organic compounds (VOCs) during 70 winter trips–evenly split between buses and saloon cars in the vicinity of Taegu, a metropolitan area of about 2.5 million people. The study found that with windows and vents closed in all vehicles (except when buses opened their doors for passengers), total concentrations of VOCs in urban settings were 63-93% higher than in the suburbs, and were about 33% higher in cars than in buses. The researchers speculate that the car-bus differential may occur because a car's air intake is closer to the ground and thus may draw in more pollutants, and because opening the doors of a bus may allow some inside VOCs to escape. The study did not compare in-vehicle concentrations with those for roadside areas.

In another earlier study, researchers analyzed a range of in-vehicle pollutants in and around the French capital. They measured carbon monoxide and six monocyclic aromatic hydrocarbons (MAHs), such as benzene and toluene, during 58 trips of about one and a half hours each. The researchers sampled air inside vehicles, buses, and subways, as well as from zones used by pedestrians and bicycle riders. The study found that someone driving a car all day in urban areas likely would exceed U.S. and World Health Organization (WHO) standards for exposure to carbon monoxide–the average measurement of 12 ppm was higher than both the WHO's eight-hour standard of 10 ppm and the EPA's eight-hour standard of 9 ppm.

In the Paris study, carbon monoxide and MAHs were 6-8 times higher inside cars than in the ambient air in the heart of the city. Compared to an earlier study in Raleigh, North Carolina, levels of benzene and other sampled MAHs were much higher for comparable carbon monoxide concentrations, possibly because catalytic converters weren't in wide use in France. Researchers also found that the air inside a vehicle was contaminated by the vehicle's own exhaust when it was idling.

Pedestrians and those riding bicycles, buses, or the subway generally were exposed to lower levels of pollutants than car or truck occupants. However, researchers speculate that someone exercising heavily, such as a bicycle rider, would presumably inhale larger volumes of air, and thus might have a total exposure similar to that of a car or truck occupant. Similarly, bus and subway riders, who tend to have a longer commuting time and thus longer exposures than a vehicle occupant, might also be expected to have a total exposure only somewhat lower than that of a car or truck occupant.

The Taegu, Paris, California, and 20 other in-vehicle pollution studies have been reviewed by the International Center for Technology Assessment (ICTA). The ICTA found similar trends in all the studies, with in-vehicle pollutant concentrations generally being significantly higher than those for both roadside and ambient air. Additional studies are currently under way.

In August 2001, the US Environmental Protection Agency announced the launch of a new study to measure air pollutant exposures and evaluate subsequent health effects among North Carolina highway patrol troopers. The results of this study are expected shortly and they should contribute significantly to a better understanding of the health effects of in-vehicle air pollution.

Source: Environmental Health Perspectives, Vol. 109, No. 9, September 2001.

Risk Analysis of Candle Emissions

A recent study has been carried out using generally accepted risk assessment methodology to evaluate the potential health risks associated with candle emissions of soot and benzene.

Previous studies that reported significant public health risks from candle emissions relied on unsubstantiated toxicity assumptions about candle soot. In these studies candle soot was assumed to exhibit the same cancer potency as diesel particulate matter (DPM). However, carcinogenic polycyclic aromatic hydrocarbons (PAHs), notable constituents of DPM, are present only at low levels in candle soot, rendering the prior assumption of equal potency invalid.

In the present study, a Monte Carlo uncertainly analysis was performed using reasonable and average candle burning and exposure times, median indoor air turnover rates and median room and house size estimates. The 95th percentile for theoretical cancer risk due to benzene exposure was between 3.24×10^{-7} and 1.15×10^{-6}, thus, the risk estimates associated with benzene emissions from candles are not distinguishable from other indoor air constituent risks. Further, these results indicate that the calculable public health risks from candle emissions, including soot, do not exceed the threshold (1 excess cancer in 100,000 individuals) for California Proposition 65 listing and labeling.

Source: Research Communications in Pharmacology and Toxicology, Vol. 6, Nos. 1&2, 2001.

Mechanisms of invasion of Bacillus anthracis

The existence of toxins of *B. anthracis* was postulated by Robert Koch in the nineteenth century, when he discovered the cause of anthrax. Since their subsequent discovery almost 50 years ago, the toxins (known as 'lethal' and 'oedema'), along with the capsule that surrounds the bacterium, have been recognized as the main components that enable anthrax to cause harm. However, the manifestations of anthrax infection are not solely due to the effects of the toxins, as is the case with diphtheria, tetanus or botulism.

Rather, in anthrax, the bacterium invades and grows to high concentrations in the host; the toxins act mainly by damaging defensive cells called phagocytes, causing the immune system to malfunction. Late in the infection, toxins may be present in large amounts in the blood and contribute directly to the death of the infected organism. Some studies, in non-human primates, suggest that lethal toxin by itself is not even particularly potent, requiring milligram quantities to cause death. But the results of other work in mice, imply that further non-toxin components contribute to virulence that have yet to be identified. So antibiotics constitute the mainstay of treatment, although anti-toxins have long been considered an essential 'adjunctive' therapy, and remain so.

The toxins are composed of three proteins: a cell-receptor binding protein, known as protective antigen; and two enzymes, lethal factor and oedema factor.

Lethal factor is a zinc protease, a type of enzyme that contains zinc and cleaves other proteins. Oedema factor belongs to a class known as adenylate cyclases. When combined with lethal factor, protective antigen constitutes lethal toxin; with oedema factor it makes oedema toxin. From cell-culture studies it seems that anthrax operates as follows. First, protective antigen is cleaved, and so activated, by a protease on the surface of the cell under attack. It then forms heptamers—aggregations of seven—and subsequently binds one or more molecules of lethal or oedema factor, or both. The complex passes into the cell through the receptor for protective antigen and on into an acidic compartment inside the cell. There the heptamer inserts into the compartment's membrane, releasing lethal and oedema factors into the cell body where they exert their toxic effects. The precise molecular targets remain unknown.

The structure of lethal factor will help in the identification of drugs that interfere with its binding, and maybe that of oedema factor, to protective antigen. Investigating inhibitors of the activities of the two factors themselves is another route, and will be aided by knowledge of the crystal structures. Therapies might include soluble toxin receptors and other drugs to prevent protective antigen from binding to its receptor. Non-toxic mutants of protective antigen have been shown to neutralize toxin, and inhibitors of the protease(s) that activates it might have the same effect. Here, detailed knowledge of toxin kinetics during infection will be required, and the timing of drug delivery is critical. Another tactic may develop from understanding how a recently discovered motor protein confers resistance to lethal toxin in some phagocytes.

In the early days of microbiology, anthrax was significant mainly as an economically damaging disease of domesticated animals. The world's scientific community addressed that problem and developed effective countermeasures. It is now necessary once again to focus on anthrax, along with other pathogenic microorganisms, this time as agents of biological terrorism and threats to civilization.

Source: Nature, Vol. 414, No. 6860, November 2001.

DEFENSE AGAINST ANTHRAX ATTACKS

Anthrax is a disease of livestock that occurs almost everywhere in the world. It is particularly difficult to eradicate because it forms hardy spores that can lie dormant in the soil for many years.

Although researchers have worked with anthrax in animals, very few have studied anthrax in humans, particularly inhalation anthrax.

Thus the apparent series of attacks with anthrax that took place in the United States in September 2001 occasioned widespread alarm and moved biodefense to the top of the political agenda.

Research institutes throughout the United States are pooling their expertise on many fronts.

Researchers familiar with the organism's DNA are being called on to help "fingerprint" the samples that arrived by mail in the apparent October assaults in hope of identifying their origins. Others are looking at vaccines that can be administered conveniently. The old standby, the only anthrax vaccine licensed for use in the United States today, requires six shots and an annual booster. It is also in short supply, and the limited stocks are reserved primarily for military use. Still other researchers are developing better diagnostics to determine who is infected and who is not, as well as drugs that can block the anthrax toxin, which remains lethal even after antibiotics have killed the bacteria. All these requirements seem likely to get increased attention in the coming months.

However developing new drugs can often take many years. In the meantime simple generic solutions may offer the best line of defense from installing highly efficient air filters in large buildings to educating the public on what to do, and what not to do, in the case of an outbreak of anthrax poisoning.

Source: Science, Vol. 294, No. 5542, October 2001.

INHERITABLE GENETIC MODIFICATIONS: URGENT NEED FOR OVERSIGHT

Techniques developed to modify genes transmitted to future generations have the potential to bring about far-reaching changes in medicine and indeed in society. A dilemma posed by the rapid pace of biomedical research is that inheritable genetic modification (IGM) techniques developed initially for therapeutic purposes are also likely to be applicable to normal genes for purposes of enhancement of desirable characteristics.

The possible effect of such developments on future generations marks IGM out as an area in which public policy is urgently needed.

In the United States, the National Institutes of Health Recombinant Advisory Committee has declared explicitly that it would not entertain proposals for germ line alterations.

Outside the United States, laws, treaties and declarations, mostly originating in Europe, overwhelmingly proscribe germ line interventions in humans.

These positions rest on a concern for the risks involved with such a nascent technology and/or a need to consider the ethical, social, and human rights implications associated with these interventions. Among the exceptions are those where the germ line alteration is not the aim, but only a side effect of medical treatment and it may only be used if the risk entailed is outweighed by the anticipated benefits; and only on persons who are unable to have descendents. It is rare to find a clear definition of what is encompassed under that rubric of "germ line" beyond the stipulation that interventions be capable of being inherited.

It has thus become a matter of urgent concern to put in place a system of oversight at the national level in countries in which IGM research is ongoing, with authority over human IGM in both public and private sector.

If IGM research or applications were to proceed, oversight should include the scientific and ethical review of all protocols or procedures with IGM implications in the public and private sectors, and a process for monitoring the uses of IGM as they go forward.

For any system of public oversight, public safety must be paramount, especially where the genetic endowment of future persons will be affected.

Source: Science, Vol. 292, No. 5520, May 2001.

Chulabhorn Research Institute

Calendar of Training Program Year 2002

PLEASE NOTE THE CHANGE IN THE DATES PREVIOUSLY ANNOUNCED

Date	Activities	Country
24-28 June	Introductory Course: Executive Seminar in Environmental Toxicology and Management for Sustainable Development	Union of Myanmar
1-2 July	Introductory Course: Executive Seminar in Environmental Toxicology and Management for Sustainable Development	Cambodia
4-5 July	Introductory Course: Executive Seminar in Environmental Toxicology and Management for Sustainable Development	Lao People's Democratic Republic
23 September-4 October	* Training Course on Principles of Environmental Toxicology and Pollution Control *(Registration Fee: US$650)*	Thailand
12-20 December	Training Workshop on Environmental and Health Risk Assessment and Risk Management of Toxic Chemicals *(Registration Fee: US$650)*	Thailand

* **TRAINING FELLOWSHIPS ARE AVAILABLE for participants from <u>ASEAN countries.</u> Deadline for submission of written application with full CV is 31 July 2002.**

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