CRI/ICEIT
NEWSLETTER

VOL. 7 NO. 2 – April 1997
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".

ATMOSPHERIC DUST AND ACID RAIN

For the past several decades, scientists have been studying acid rain and how it affects the environment. As the harmful consequences of acidic air pollutants became increasingly clear, governments in North America and Europe began to regulate emissions of these compounds. Countries in the European Union enacted a variety of laws to control the release of sulfur dioxide and nitrogen oxides; the Clean Air Act imposed similar regulations in the U.S. Policymakers expected these reductions to rejuvenate forests, lakes and streams in many regions.

But the problem of acid rain has not gone away. Recent findings suggest that acid rain is a much more complex phenomenon than previously thought. Results from several studies point to the unexpected but critical role of chemicals in the atmosphere known as bases, which can counteract the effects of acid rain by neutralizing acidic pollutants.

The attention given to acidic compounds in the atmosphere has obscured the fact that emissions of bases have also decreased. A number of factors seem to be diminishing the level of these atmospheric bases and in the process aggravating the ecological effects of acid rain. Ironically, among these factors are some of the very steps that governments have taken to improve air quality.

Acids and bases are measured by what is known as the pH scale: solutions with a pH of less than 7 are acidic; those with a pH greater than 7 are basic; those with a pH of 7 are neutral. Common acids around the home include vinegar, orange juice and beer; ammonia, baking soda and antacid tablets are all bases. Most of the bases in the atmosphere can be found in airborne particles referred to as atmospheric dust. These dust particles are rich in minerals such as calcium carbonate and magnesium carbonate, which act as bases when they dissolve in water.

Atmospheric dust particles originate from a combination of sources. Fossil-fuel

(Continued on page 2)

ATMOSPHERIC DUST AND ACID RAIN

(Continued from page 1)

combustion and industrial activities such as cement manufacturing, mining operations and metal processing, generate particles that contain bases. Construction sites, farms and traffic on unpaved roads also contribute. Sources such as forest fires and erosion caused by wind blowing over arid soils with little vegetation are considered natural yet can still be linked to human activity.

In the air, dust particles can neutralize acid rain in a manner similar to the way antacids counteract excess acid in an upset stomach. In a sense, when an acid and a base combine, they cancel each other out, producing a more neutral substance. Neutralization in the atmosphere takes place as dust particles dissolve into acidic cloud-water droplets or combine directly with acidic gases such as sulfur dioxide or nitrogen oxides. These reactions also generate so-called base cations – a term used to describe the positively charged atoms of elements such as calcium and magnesium that arise when mineral bases dissolve in water.

In addition to lowering the acidity of precipitation, atmospheric base cations also neutralize acid rain once they reach the ground – although the chemistry is a bit different than in the atmosphere. Small particles of clay and humus (decayed organic matter) in soil bear negative charges and thus attract positively charged cations, such as calcium and magnesium; as a result, soils contain a natural store of base cations attached to these particles. As acidic rainwater drains into the ground, the base cations give up their places to the positively charged hydrogen ions found in acids, which bind more tightly to the soil particles. Because these particles sequester hydrogen ions, the acidity of the water that flows through the soil stays low. In some soils the process becomes more complex: acid rain triggers the dissolution of toxic aluminum ions that also displace the base cations.

As long as the soil has an abundant supply of base cations, this buffering system, known as cation exchange, protects forests from the harmful effects of acid rain. But the natural reserves of base cations can become depleted if soils that are

(Continued on page 8)

Effects of the Green Aid Plan

The Green Aid Plan was launched in 1991 by Japan's Ministry of International Trade and Industry to help Asian countries overcome their domestic pollution problems. It has become increasingly focused on China's rapidly expanding power and steel industries.

Across East Asia, fast economic growth, out-dated industrial plants, and lack of environmental regulations are creating record levels of air pollution. The region now accounts for more than a third of the world's output of sulphur oxide gases. The northeastern city of Shenyang in China is responsible for emission of 200,000 tonnes of sulphur oxide a year, equivalent to the total emissions of the whole of Japan.

The problem is the high sulphur coal that is China's main source of energy. The large amounts of sulphur dioxide produced as the coal burns dissolve in rainwater and fall to the ground as sulphuric acid. Old, inefficient technology means that China must burn 50 million tonnes of coal more than a developed country would for the same amount of energy. Fifty million tonnes of Chinese coal produce 1.4 million tonnes of sulphur dioxide.

The rapid increase in the demand for electricity in Asia has caused coal consumption in the region to grow by an average of 5.5 percent a year over the past decade.

Japan is deeply concerned about pollution from China, especially from the rapidly industrializing northeast. The problem is most apparent in China itself, with more than 40 percent of the country thought to be affected by acid rain; but pollution in China has already had a serious effect on southern Japan, and the effect is spreading.

Researchers at Japan's National Institute for Environmental Studies have set up a computer model to predict what effect China's future emissions of nitrogen and sulphur dioxides will have on Japan. It is sophisticated enough to take account of chemical reactions between pollutants in the atmosphere as well as their diffusion patterns, and simulates the spread of pollutants from China across northeastern Asia.

The imminent threat of pollution from China has become a major issue for Japan. China is being told either to take measures to prevent pollution or risk losing Japanese aid for building power stations. At the same time, the Green Aid Plan offers incentives and technical help to reduce toxic emissions.

Under the plan, a study group was set up in 1993 to investigate air and water pollution in China. One of its earliest projects was an experimental flue gas desulphurization plant at the Huang Tao power station in Shandong province. Mitsubishi Heavy Industries, the Japanese company that built the plant, has since won contracts for three more.

In September 1996, the Chinese government announced that it intended to spend up to 320 billion yuan in the next five years to reduce pollution. It claims to already have shut down some 57,000 dirty factories, mainly paper and chemical plants.

China is sensitive to Western criticism of its environmental record and claims that it has done more to curb pollution than developed countries did in their early days of industrialization.

Source: New Scientist, 15 February 1997.

Particulate Air Pollution

After fifty years of research on air pollution, the problem of what controls the toxicity of the ambient particulate aerosol is still being discussed.

Recent studies on particles can be divided into two categories: cohort studies in towns and cities of differing pollution levels, and time-series studies of variations of concentrations of pollutants on health.

The time-series studies provide evidence that suggests day-to-day variations in already low concentrations of particles have a damaging effect on health. This effect does not appear to be much dependent on the composition of the particles and there is no apparent threshold of effect.

Demonstration of the effects on health depends on modelling the relationship between day-to-day rates of events such as deaths or asthma attacks and a range of known or suspected causal factors. These include season, day of the week, pollen levels, temperature, relative humidity, concentrations of gaseous pollutants such as SO_2, NO_2, O_3 and CO and concentrations of particles.

Only when the multiple linear regression models have been built and the effects of these factors added or removed does the effect of particles appear. The effect revealed is not large: an increase in concentration of particles equivalent to an increase in PM_{10} (particles of less than 10 micrometers diameter) of 10 micrograms/m^3 is associated with only a 1 percent increase in daily deaths and a 1 to 3 percent increase in other indicators. Although the effect is not large, the cumulative impact may be considerable.

Support for the results of time-series studies has come from the large cohort studies which have been carried out in the United States for over twenty years. Such results are evidence of the need to undertake long-term and inevitably expensive public health research.

Source: Chemical Incident, Vol. 1, No. 4, October 1996.

Mortality among Workers Exposed to Carbon Disulfide

Studies from the United Kingdom, Finland and the United States have demonstrated increased mortality from coronary heart disease in viscose rayon workers who are exposed to carbon disulfide (CS_2).

The mechanism of toxicity for CS_2 is not known, but possibilities proposed have included a direct toxic effect on the myocardium or an acceleration of the atherosclerotic process. Carbon disulfide has been associated with increases in total cholesterol in some studies. It has been observed that relatively modest exposure to CS_2 may raise low-density lipoprotein (LDL) cholesterol concentrations, as well as diastolic blood pressure.

Another target organ appears to be the nervous system. Effects on the peripheral nervous system that lead to neuropathy have been reported in a number of recent studies. Cases of chronic CS_2 poisoning have been described in which the symptoms are very similar to those of cerebral arteriosclerosis of pre-senile origin, occurring at a relatively early age (from 42 to 55 years) after many years of exposure.

A recent study of the possible effects of carbon disulfide on cerebrovascular disease was made at a plant in Ontario, Canada, that produced viscose rayon, with carbon disulfide as a main raw material. Work-history records for 279 deceased workers at the plant (plant A) were obtained and compared with those for 511 deceased workers at a pulp and paper plant in the same city (plant B). In a proportional mortality analysis, using as a reference the general population of Ontario, at both plants there were fewer deaths from ischemic heart disease than expected (the proportional mortality ratios [PMRs] were 83 at plant A and 95 at plant B) but more deaths than expected from cerebrovascular disease (PMRs were 115 at plant A and 149 at plant B). In a subgroup of plant A workers who had been employed in high-carbon-disulfide exposure areas, deaths from ischemic heart disease were less than expected, particularly among those who worked in these areas for more than 5 years. Most deaths occurred among those aged 65 years or more. Mortality from strokes, however, was greater than expected; the excess was confined to workers who died at age 65 years or older. Proportional mortality from strokes was also increased in the pulp workers among those who died at age 65 years or older. In a case-control analysis, the risk of ischemic heart disease at plant A was slightly less than at plant B, with no association between risk and years worked in high-carbon-disulfide areas. Among those who died at age 65 years or older, the risk of stroke in the high-exposure subgroup was (a) increased significantly, compared with other plant A workers; and (b) increased slightly, compared with plant B workers. These results suggested an unusually low risk of strokes among other plant A workers. The risk of stroke was associated with years in high-carbon-disulfide areas. The observed increase in proportional mortality from strokes may represent a chance finding, but a causal role for exposure cannot be excluded.

Source: Archives of Environmental Health, Vol. 51, No. 3, May/June 1996.

BIOLOGICAL MONITORING OF CADMIUM EXPOSED WORKERS

The human health effects associated with cadmium (Cd) exposure in air include multiple organ system insults. However, principal health effects of concern are kidney dysfunction manifested primarily as tubular proteinuria and lung cancer.

Recognizing these health risks, the US Occupational Safety and Health Administration (OSHA) published final cadmium standards in 1992 covering general, maritime, and agricultural industries, with the second standard covering the construction industries for occupational exposure to cadmium.

A significant use of cadmium in the United States is in nickel-cadmium (Ni-Cad) battery manufacture. OSHA has estimated that approximately 3,000 workers are exposed to cadmium in their work environment in the six major US manufacturers of Ni-Cad batteries.

As part of a settlement agreement with OSHA involving exposure to cadmium, a battery production facility provided medical surveillance data to OSHA for review. Measurements of cadmium in blood, cadmium in urine, and beta2-microglobulin in urine were obtained for more than 100 workers over an 18-month period. Some airborne Cd exposure data were also made available. Two subpopulations of this cohort were of primary interest in evaluating compliance with the medical surveillance provisions of the Cadmium Standard. These were a group of 16 workers medically removed from cadmium exposure due to elevations in some biological parameters, and a group of platemakers. Platemaking had presented a particularly high exposure opportunity and had recently undergone engineering interventions to minimize exposure.

Studies of the effect on three biological monitoring parameters of medical removal protection in the first group and engineering controls in platemakers reveal that both medical removal from cadmium exposures and exposure abatement through the use of engineering and work practice controls generally result in declines in biological monitoring parameters of exposed workers.

Classical industrial hygiene control elements have been successfully employed in this work environment to decrease employee exposure to cadmium. Workplace air sampling data demonstrate exposure concentration declines with engineering interventions. Additionally, biological monitoring data complement workplace sampling results and document declines in worker CdB (cadmium in blood) and CdU (cadmium in urine) values, illustrating the efficacy of engineering and work practice controls, as well as the administrative control of medical removal.

Source: American Industrial Hygiene Association Journal, Vol. 57, November 1996.

Mercury Exposure from Breast Milk

Compared with placental transport, little attention has been directed to lactational transport of mercury compounds. A recent study, therefore, attempted to evaluate postnatal exposure to mercury via breast milk. The study group comprising 30 lactating women from a community in the north of Sweden participated in a monitoring study on metal levels in breast milk. Twelve of the women had delivered their first child and the remainder had breast-fed between one and four children previously. In addition to breast milk, 5 infants also received infant formula. Hair samples were collected at the time of delivery from 26 of the 30 women. Samples of antecubital vein blood were taken six weeks after delivery in heparinized, mercury-free glass tubes. At the same time, approximately 80 ml of milk was collected from the mothers by an electrical milk pump, two to three hours after feeding the infant.

At six weeks after delivery each woman answered a questionnaire about breast feeding as well as a

questionnaire that explored fish consumption after delivery.

Information on the number, size and location of amalgam teeth fillings was obtained from 27 of the 30 women. The mean number of amalgam fillings was 12 ± 4 (range = 3 – 17).

In the group under study, the total mercury concentration was 0.6 ± 0.4 ng/g and the range was 0.1 – 2.0 ng/g. Mean total mercury concentrations in whole blood and hair were 2.3 ± 1.0 ng/g for blood and 0.28 ± 0.16 µg/g for hair. The levels of organic mercury were 0.3 ± 0.2 ng/g in milk and 1.7 ± 0.7 ng/g in blood.

The inorganic mercury fraction in milk averaged 51 percent of the total mercury in milk. The levels of inorganic and total mercury were highly intercorrelated. In addition, there was a significant correlation in blood: an average 26 percent of the total mercury in blood was present as inorganic mercury.

Total mercury concentrations in blood and milk (but not in hair) correlated with the numbers of amalgam fillings, with the total mercury concentration in blood increasing 0.1 ng/g and 0.05 ng/g in milk, for each filling. Also, there were significant correlations between the levels of inorganic mercury in blood, as well as in milk, and the number of amalgam fillings.

The data from the study showed that inorganic mercury was transported from amalgam fillings to breast milk via blood. Furthermore, the intake of methylmercury via fish was reflected by increased mercury levels in blood and hair, but not in milk.

The excretion of mercury in milk after exposure to methylmercury or inorganic mercury is not understood well. Neither are the adverse effects of early postnatal exposure to inorganic mercury. Breast milk offers a unique and complete nutrition source to the infant. Therefore, all efforts should be made to prevent its contamination with environmental pollutants.

Source: Archives of Environmental Health, Vol. 51, No. 3 May/June 1996.

POTENTIAL HEALTH HAZARDS FROM UNCOATED METALLIC LEAD

Metallic lead is widely used as radiation shielding in research and development, nuclear medicine and radiology. Although the density of lead dust is high, it may still become airborne, contaminate floors and other work surfaces, and be inadvertently inhaled or ingested if protective equipment is not properly used and if personal hygiene practices are poor.

Since lead is an important shielding material for gamma and x-rays, it is in general not feasible to eliminate or substitute it. Other means must be employed to minimize potential health hazards from dispersable lead dust.

One solution is to encapsulate lead sheeting where possible. If coatings are used, however, careful attention should be given to their composition. Metallic additives could become activated and produce a residual radiation hazard if the shielding is later used in an environment with sufficient photon or particle energies.

Even with an ideal encapsulant, it is extremely labor-intensive to coat sheeting after shielding is mounted.

The following precautions should be observed whenever handling or storing uncoated lead shielding:

- ☆ Recognize that metallic lead releases lead dust. Minimize the handling of lead shielding, require that impervious gloves be worn during such work, and encourage hand washing when the work is completed. Extended handling may also create an inhalation hazard and warrant respiratory protection and disposable coveralls.
- ☆ Although lead is known for its resistance to corrosion, significant oxidation can occur in humid environments. Do not store lead shielding outdoors since, even when wrapped in plastic, condensation will occur and promote surface oxidation.
- ☆ Purchase or prepare individually encapsulated lead bricks; alternatively, consider *in situ* encapsulation or enclosure. Vinyl, plastisol, polyurethane, epoxy, and thermosetting acrylics are recommended for protecting metals from abrasion, marring, and physical impact. Identify the intended uses of the shielding to avoid a coating that could become activated in the future.
- ☆ Work areas near large lead shielding structures should be regularly cleaned and in a manner that minimizes the generation and resuspension of particulate matter, i.e., wet mopping followed by high-efficiency particulate air (HEPA)-filtered vacuuming for a comparison of cleaning methods on carpeted, bare wood, and linoleum-covered floors.

Source: American Industrial Hygiene Association Journal, Vol. 57, December 1996.

HORMONE TREATMENT CANCER RISK

According to a report published in the Lancet, many women who take hormone treatments for menopause may be getting insufficient amounts of one of the hormones they need to reduce their risk of endometrial cancer.

To lower this risk, many oestrogen replacement regimens are now combined with a progestagen for at least a few days a month. This opposes the effect of oestrogens on endometrial cells. Researchers have found that when compared with women who had never taken hormone replacement therapy for more than 6 months, women who had taken oestrogens without progestagens had a four-fold increase in the risk of endometrial cancer. In contrast, in women who took a progestagen with their oestrogen therapy, there was little or no increase in risk for endometrial cancer. However, this protective effect depends on how many days a month the women took the progestagen. It was found that for women who took progestagen for 10 to 21 days each month, there was no significant increase in risk. However, for those who took progestagens for fewer than 10 days a month, the risk of endometrial cancer was three-times higher compared with non-users.

The researchers who carried out this study caution that their results are preliminary. They note, moreover, that the protective effect of progestagens may diminish over time. The study does not provide information on women who take the combined therapy every day of the month.

Source: The Lancet, Vol. 349, No. 9050, February 1997.

Cancer Chemopreventive Activity of Resveratrol

The results of several epidemiological studies have suggested that coronary heart disease mortality can be decreased by moderate consumption of alcohol, especially red wine.

Now, researchers at the Department of Medicinal Chemistry and Pharmacology at the College of Pharmacy, University of Illinois, have found that resveratrol, a phytoalexin present in grapes and other food products, demonstrates cancer chemopreventive activity in assays representing three major states of carcinogenesis.

Resveratrol was found to act as an antioxidant and antimutagen and to induce phase II drug-metabolizing enzymes (anti-initiation activity); it mediated anti-inflammatory effects and inhibited cyclooxygenase and hydroperoxidase functions (antipromotion activity); and it induced human promyelocytic leukemia cell differentiation (antiprogression activity). In addition, it inhibited the development of preneoplastic lesions in carcinogen-treated mouse mammary glands in culture and inhibited tumorigenesis in a mouse skin cancer model.

The results of this work suggest that resveratrol merits further investigation as a cancer chemopreventive agent in humans.

In the light of the adverse health effects of long-term alcohol consumption, however, foods and non-alcoholic beverages derived from grapes should be considered as alternative dietary sources of resveratrol.

Source: Science, Vol. 275, No. 10 January 1997.

OXIDATION RETARDING PROPERTIES OF CHOCOLATE

A question that has puzzled scientists is why unrefrigerated chocolate bars do not turn rancid in the way so many other high-fat foods do? The answer given by food chemists from the University of California, who have been conducting research into the properties of chocolate, is that cocoa, the main ingredient of chocolate products, contains a number of potent flavonoids-pigments and other polyphenols that retard oxidation, which can make fats deteriorate.

In the body, oxidation transforms lipid-rich low-density lipoproteins (LDLs) in the blood into the foam cells that create artery clogging plaque. Cocoa powder extract, however, protects LDLs – the so-called bad lipoproteins – from oxidation. Its polyphenols, at a concentration of 5 micromoles per liter, cut oxidation of human LDLs by 75 percent in test-tube experiments.

The extract's antioxidant potency approximately matches that of gallic acid, another potent antioxidant, and according to recent studies, it appears to outperform the flavonoid blend in red wine.

Sources: The Lancet, Vol. 348, September 21, 1996.

Science News, Vol. 150, October 12, 1996.

Sunlight and Skin Cancer

Recent studies have provided a new insight into the link between sun exposure and nonmelanoma skin cancer, furnishing information about events occurring between the time of initial sun exposure and skin cancer years later.

The multistage theory of carcinogenesis is based on experimental studies in rodents and has been proposed as a general model for environmental carcinogenesis. In the first stage, initiation, a carcinogen mutates a target gene. This is followed by promotion, a process in visibly normal skin in which the single damaged cell expands to form a clone of damaged cells. These changes progress, leading to pre-cancerous clinically abnormal skin and then to cancer. A number of experimental studies have been designed to dissect the cellular and molecular mechanisms involved in this process. These studies involve investigations of DNA repair, eicosanoid and proteinase production, cytokine activation and immune suppression, and specific tumor-suppressor genes including patched and p. 53.

A picture emerges from these studies of the role of p53 in sunlight-induced skin cancer. Sun exposure of normal skin results in many p53 mutations that serve as an initiation process that start the cells on the path toward cancer.

Sun exposure also serves as a tumor promoter. Normally functioning p53 serves as a type of monitor. Cell damage results in increased stabilization of p53 protein that slows down the cell cycle to permit repair of DNA damage and turns cells sustaining unrepaired DNA damage toward apoptosis (programmed cell death) rather than the normal squamous differentiation. This is a protective mechanism that rids the skin of severely damaged cells.

In skin, cells appearing after UV exposure that have a histologic appearance commonly called "sunburn cells" have been demonstrated to be apoptotic cells. These sunburn cells are virtually absent in UV-exposed skin of mice with a homozygous knockout of the p53 gene, while heterozygous p53 knockout mice have a partially reduced response in comparison to normal mice.

Thus cells with a single p53 mutation are more susceptible to the tumor promoting effects of sun exposure. They have a diminished p53-mediated apoptotic cell death protective mechanism thereby permitting these cells to continue to survive in an area of skin in which surrounding cells with wild-type p53 are killed by apoptosis. Eventually, repeated UV insults to cells containing a single p53 gene mutation may lead to a second mutation in the other allele or to loss of a portion of the normal chromosome. This allelic loss is commonly seen in actinic keratoses and is correlated with the appearance of clinically and histologically abnormal skin.

However, only about 1 in 1,000 actinic keratoses develop into squamous cell carcinomas. Other factors such as mutations in tumor-specific genes may determine the outcome.

Source: Proc. Natl. Acad. Sci. USA, Vol. 94, January 1997.

ANNOUNCEMENT

Inter-university Post-Graduate Programs in Environmental Toxicology, Technology and Management by Asian Institute of Technology (AIT), Chulabhorn Research Institute (CRI) and Mahidol University (MU)

An innovative multi-disciplinary inter-university program between the Chulabhorn Research Institute (CRI), the Asian Institute of Technology (AIT) and Mahidol University (MU) combining health sciences, biotechnology and environmental engineering will open in January 1998. The degree granting institute is AIT.

The curriculum has been designed by a team of international experts with support from the United Nations Development Programme to train human resources capable of undertaking control and management of toxic chemicals as well as research and development in the areas of environmental toxicology, technology and environmental management. This curriculum supports both Master of Science and Doctor of Philosophy degrees. In the master's and doctoral degree programs, coursework and research thesis are required.

Admission Requirements:

To be eligible for admission to the master's degree program, a candidate must hold a bachelor's degree (normally from a four-year program) or its equivalent preferably in Biological Sciences, Chemistry, Engineering, Natural Sciences, Medical Sciences, Agriculture or in a related field. Candidates for the doctoral degree program should hold a master's degree or its equivalent from an institution of good standing, and should normally have a GPA of 3.50 at the master's level.

For further Information

Please contact:

The Dean
School of Environment,
Resources and Development
Asian Institute of Technology
P.O. Box 4, Khlong Luang
Pathumthani 12120, THAILAND
Tel: (66-2) 524-6069
Fax: (66-2) 524-6071
E-mail: *deanserd@ait.ac.th*
AIT Homepage: *http://www.ait.ac.th*

or

Chulabhorn Research Institute
Office of Academic Affairs
Vipavadee Rangsit Highway
Bangkok 10210, THAILAND
Tel: (66-2) 574-0615
Fax: (66-2) 574-0616, 247-1222
E-mail: *etmgrad@tubtim.cri.or.th*
CRI Homepage: *http://www.cri.or.th*

ATMOSPHERIC DUST AND ACID RAIN

(Continued from page 2)

naturally poor in bases are exposed to acid rain over decades, as has been the case in regions of Europe and North America.

As scientists have discovered the importance of bases in the atmosphere and, more recently, the link between emissions of atmospheric dust and nutrients in the soil, they have begun to paint a new picture of how forests respond to atmospheric pollution. This emerging view suggests that the effects of acid rain are more complex than expected and that the damage caused by the pollution is more serious than predicted.

It is entirely feasible that continuing acid rain, in combination with limited supplies of base cations, could produce environmental conditions to which many plant species, particularly in sensitive ecosystems, have never been exposed in the course of their evolution. Consequently, predicting how they will respond over the next several decades will be extremely difficult.

Base cations take years to build up in soils, and it may take decades or more for forests to recover their depleted pools of nutrients, even if levels of acidic air pollution continue to fall. In the meantime, researchers and governments must develop careful strategies not only for monitoring the current health of forests but also for predicting their stability in the next century and beyond. Simple solutions do not always work in complex ecosystems.

Source: Abridged from Scientific American, Vol. 275, No. 6, December 1996.

EDITORIAL BOARD

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

The ICEIT NEWSLETTER is published quarterly by the International Centre for Environmental and Industrial Toxicology of the Chulabhorn Research Institute. 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 UNIMAHI TH
Telefax: (662) 247-1222
Tel: (662) 247-1900