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Chulabhorn Research Institute

INTERNATIONAL CENTER FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

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

THE KYOTO PROTOCOL — THE FIRST STEP IN A LONG JOURNEY

With Russia's ratification of the protocol in October 2004, the treaty signed in December 1997 is due to finally come into force in January 2005, since the treaty becomes effective 90 days after ratification.

The Kyoto Protocol is a legally binding international agreement to reduce the greenhouse gas emissions causing climate change.

The agreement commits industrialized countries to reduce emissions of six greenhouse gases by 5% by 2012. Rather than placing a specific target on each of the gases, the overall targets for all six will be translated into "CO_2 equivalents" to produce a single figure.

The agreement specifies that all Parties to the Protocol must follow a number of steps including:

- design and implementation of climate change mitigation and adaptation programs
- preparation of a national inventory of emissions removals by carbon sinks
- promotion of climate friendly technology transfer
- fostering partnerships in research and observation of climate science, impacts and response strategies

Developing countries are not legally bound to emissions reduction targets as yet, because these countries have historically been responsible for only a small portion of the global greenhouse gas emissions.

Emissions Reduction Targets:

Requirements to achieve the 5% group target:

- 8% emission cuts by Switzerland, most Central and East European states, and the EU (which will meet its target by distributing different rates among its member states.)
- 7% emission cut by the US
- 6% emission cuts by Canada, Hungary, Japan, and Poland

(Continued on page 2)

THE KYOTO PROTOCOL — THE FIRST STEP IN A LONG JOURNEY

(Continued from page 1)

- Russia, New Zealand, and Ukraine are to stabilize their emissions
- Norway may increase emissions by up to 1 %
- Australia may increase emissions by up to 8%
- Iceland may increase emissions up to 10%

A provision in the agreement allows a nation to meet its reduction quota by reducing emissions from power plants and automobiles. Developed countries may also achieve their commitments by deducting the greenhouse gas emissions absorbed by carbon sinks from their gross emissions in the commitment period. This provision includes emissions absorbed or emitted by certain land-use changes and forestry activities, such as reforestation.

Why the Kyoto Protocol is Just the Beginning

Every year we release almost 7 billion tons of carbon into the atmosphere, where it will remain for around a century, raising the level of carbon dioxide in the atmosphere and trapping more of the sun's heat.

Before the industrial age, the CO_2 level was steady at around 280 parts per million. When the Kyoto Protocol was drawn up in 1997, the CO_2 level had reached 368 ppm. In 2004, it hit 379 ppm.

Most predictions of soaring temperatures, flood, droughts, storms and rising sea levels are based on a concentration of 550 ppm. On current trends, this figure is likely to be reached in the second half of this century. Even if levels rose no higher, this would just be the start. Time lags in natural systems such as ice caps and ocean circulation mean that changes will continue for millennia after the CO_2 level stabilizes.

The bottom line is that only drastic cuts in global emissions of CO_2, of two-thirds or more, can stop the concentration of the gas rising ever higher and stave off ever more severe climate change. The more quickly the world can make such cuts, the lower the level at which concentrations will eventually stabilize.

The Kyoto Protocol, however, involves only very modest reductions of less than 5%. The US does not support it, developing nations do not have to make any cuts, and it expires in 2012. Perhaps most crucially, it does not provide a blueprint for where we want to end up and how we intend to get there.

However, activation of the Kyoto Protocol is nonetheless highly significant as it now frees negotiators to begin to discuss what to do next.

Climate scientists say politicians must move on from Kyoto-style piecemeal negotiations on individual national targets to a global plan to cap concentrations of critical greenhouse gases, especially CO_2. Most would like to see CO_2 concentrations in the atmosphere kept below 450 ppm, but many accept that 550 ppm is more realistic.

This would still lead to substantial climate change, with the temperature rising by 2°C to 5°C and the sea level rising by 0.3 to 0.8 metres by 2100, and by 7 to 13 metres over the next millennium. But a 550 ppm ceiling would stave off even more severe changes. It would also address the international commitment made at the Earth Summit in Rio in 1992 to prevent "dangerous" climate change.

One option being discussed is to propose a ceiling on atmospheric CO_2 that would set a firm and scientifically coherent benchmark to measure the success of future negotiations.

Agreeing on a CO_2 ceiling would be the easy part. Any ceiling effectively puts an absolute limit on global emissions over the coming century, and the tricky part will be deciding who is entitled to make those emissions.

Developing countries insist they can only accept quotas based on population and suggest extending the Kyoto plans for emissions trading to smooth the transition. Industrialized countries such as the US, which emits eight times as much CO_2 per head of population as China and 18 times as much as India, reject such suggestions, but are having difficulty finding a fair alternative.

Assuming agreement can be reached on emissions quotas, the next step will be achieving them. Stabilizing at 550 ppm would mean ensuring global emissions peak no later than 2025, according to the Intergovernmental Panel on Climate Change. Simple measures such as improving energy efficiency would help, but they will not be nearly enough. To ensure we add no more carbon to the atmosphere than we take away will require major structural changes to the global energy industry.

How much this will cost is unclear. Some economists say such changes will be hugely expensive, while companies with a competitive lead in alternative technologies see only profits. Big changes will be necessary whatever happens, as oil and natural gas supplies dwindle, though coal is still available in huge quantities.

Then there is the question of exactly what changes to make. The relative contribution of renewable energy sources such as wind and solar power, the role of the hydrogen economy and whether fission power has a role to play are still fiercely debated.

In the US many insist that research into better technologies is more important than premature, expensive measures to cut emissions, and that may be right. But time is running out. It took 150 years for CO_2 concentrations to rise from 250 ppm to 330 ppm; it has taken just 30 years to get from 330 ppm to 380 ppm.

Source: Newscientist.com, October 2004.

The Effect of Cigarette Smoking on the Association Between Ingested Arsenic and Lung Cancer

Cigarette smoking has been found to be a major cause of lung cancer during the past 50 years, and it was estimated that quitting cigarette smoking may prevent more than 90% of lung cancers. A meta-analysis of studies on occupational arsenic exposure via inhalation found a synergistic effect of cigarette smoking and arsenic on lung cancer, and 30% to 54% of lung cancer cases were attributable to both exposures. A population-based case-control study reported an odds ratio (OR) of 32.0 (95% CI, 7.2-198.0) for cigarette smokers who had an ingested arsenic exposure level of 200 µg/L or higher compared with nonsmokers exposed to an arsenic level of less than 49 µg/L. The OR was much higher than that for cigarette smoking alone (OR, 6.1; 95% CI, 1.3-39.2; for cigarette smokers compared with nonsmokers) and elevated arsenic exposure alone (OR, 8.0; 95% CI, 1.7-52.3; for arsenic exposure of ≥200 µg/L compared with <49 µg/L).

This study combined 2 study cohorts recruited from southwestern and northeastern Taiwan with 10591 residents who had been followed up for an average of 8 years in an effort to elucidate the dose-response relationship between ingested arsenic exposure and lung cancer risk. The larger number of study participants, longer period of follow-up with more incident lung cancer cases, and wider range of arsenic exposure levels provided a unique opportunity to further investigate the modifying effect of cigarette smoking on the association between ingested arsenic and lung cancer.

The results indicated that the lung cancer risk among those with the highest exposures to cigarette smoking and arsenic could be as high as 11-fold when compared with nonsmokers with the lowest arsenic exposure. Approximately 32% to 55% of lung cancer cases were estimated to be attributable to the combined effect of cigarette smoking and ingested arsenic, depending on the levels of both exposures. The synergy indices ranged from 1.62 to 2.52, which were all above 1, indicating a synergistic effect under an additive scale. This finding was consistent with a meta-analysis of occupational arsenic exposure via inhalation and cigarette smoking, with 30% to 50% of lung cancer cases attributable to both exposures.

The study provides evidence of a synergistic relationship between cigarette smoking and ingested arsenic on the risk of lung cancer. The reductions in cigarette smoking would likely reduce the lung cancer risk accompanied by exposure to arsenic, and similarly, reductions in arsenic exposure would reduce the lung cancer risk among cigarette smokers. Appropriate public health interventions, such as cigarette smoking cessation programs and reduction in arsenic concentration of drinking water, are warranted. Furthermore, it is essential to take cigarette smoking into consideration in the risk assessment and the determination of the maximal contamination level of arsenic in drinking water.

Source: JAMA, Vol. 292, No. 24, December 2004.

HEALTH EFFECTS OF LOW-LEVEL LEAD EXPOSURE

Many studies have reported impaired renal function and kidney disease at high levels of lead exposure, as estimated mainly through concentrations of serum creatinine (SCr) and rates of creatinine clearance from the body. However, lower-level lead exposure has not been correlated with renal effects as conclusively, perhaps because blood lead reflects relatively recent exposure, and therefore is not an adequate measure of total body burden. Now however, researchers at the Harvard School of Public Health have undertaken a prospective study to examine changes in renal function during 6 years of follow-up in relation to baseline lead levels, diabetes, and hypertension among 448 middle-age and elderly men.

Lead levels were generally low at baseline, with mean blood lead, patella lead, and tibia lead values of 6.5 µg/dL, 32.4 µg/g, and 21.5 µg/g, respectively. Six percent and 26% of subjects had diabetes and hypertension at baseline, respectively. In multivariate-adjusted regression analyses, longitudinal increases in SCr were associated with higher baseline lead levels but these associations were not statistically significant. However, significant interactions of blood lead and tibia lead with diabetes in predicting annual change in SCr were observed. For example, increasing the tibia lead level from the midpoints of the lowest to the highest quartiles (9-34 µg/g) was associated with an increase in the rate of rise in SCr that was 17.6-fold greater in diabetics than in nondiabetics (1.08 mg/dL/10 years vs. 0.062 mg/dL/10 years; $p < 0.01$). Significant interactions of blood lead and tibia lead with diabetes in relation to baseline SCr levels (tibia lead only) and follow-up SCr levels were also observed as well as a significant interaction of tibia lead with hypertensive status in predicting annual change in SCr. The researchers concluded that longitudinal decline of renal function among middle-age and elderly individuals appears to depend on both long-term lead stores and circulating lead, with an effect that is most pronounced among diabetics and hypertensives, subjects who likely represent particularly susceptible groups.

Source: Environmental Health Perspective, Vol. 112, No. 11, August 2004.

TRAINING PROGRAM FOR CAPACITY BUILDING IN ENVIRONMENTAL TOXICOLOGY FOR CHEMICAL SAFETY MANAGEMENT AND ENVIRONMENTAL PROTECTION IN LAO PEOPLE'S DEMOCRATIC REPUBLIC

Executive Seminar on Environmental Toxicology for Sustainable Development
November 8 – 10, 2004, Vientiane, Lao, P.D.R.

It is well recognized that environmental problems, although existing at country level, usually have global impacts. At present, in Laos, environmental damage due to toxic chemicals and hazardous wastes from industrial processes has not yet reached the levels encountered in many industrialized countries. If immediate action is taken there still exists an opportunity to develop a proper program for regulating and managing environmental toxicological problems to prevent long-term degradation and irreparable damage.

A successful and effective environmental management system requires appropriately trained personnel as well as the cooperation of government, industrial academic and public sectors. Unfortunately, in Laos, there appears to be a critical shortage of well-trained and qualified personnel in this subject area. The majority of existing technical personnel are not specifically trained in toxicology, and in many cases are mobilized from related disciplines. They therefore need extension training to be able to exercise the safe use, control and management of toxic chemicals as well as to assess and manage health and environmental risks. Without the appropriate level of human resource development, the sustainability of the recent rapid development of the country will be seriously compromised.

Since 1963, the Thai Department of Technical and Economic Cooperation (DTEC) has been responsible for the technical cooperation which Thailand provides to other developing countries, as well as mutual assistance programs organized among developing countries. Many of these activities are funded entirely by the Thai government. However, some are paid for by foreign governments. At present, Thailand provides technical cooperation to other developing countries under the scheme entitled "The Thai International Cooperation Program" (TICP).

Activities performed under the TICP banner include development projects, missions, awarding of fellowships and other forms of training, and the dispatch of experts and equipment. Projects focus mainly on priority sectors (agriculture, education and public health), which have been identified by Thailand's cooperation partners, and are designed to help Thailand's partners reach their development goals.

Currently there are very few academic or research institutes in Southeast Asia that are in a position to offer training specifically tailored to regional needs or to carry out the necessary research to support training programs. The Chulabhorn Research Institute (CRI) has built up its own infrastructure capacity with regard to both physical plant and equipment and the establishment of linkages with a number of renowned institutes, universities and international organizations. As a result, it is in a position to assist the countries in this region in human resource development to support environmental management for sustainable industrial development. Past activities and track record of CRI's International Center for Environmental and Industrial Toxicology (ICEIT) has earned recognition from the United Nations Environmental Programme (UNEP) which, in 1990, designated the CRI's ICEIT as "UNEP Centre of Excellence in Environmental and Industrial Toxicology".

ICEIT has regularly organized training, seminars and conferences from which participants from Thailand and other developing countries in Asia and Pacific have benefited. It is through ICEIT's activities that Science Technology and Environment Agency (STEA), Department of Environment, Laos has established cooperation and has successfully initiated awareness in Environmental Toxicology in government and academic sectors. Training has been provided to a small number of governmental personnel through the support of ASEAN foundation from 2000 – 2003. Recognizing the importance of environmental toxicity for sustainable development, Laos therefore requests a sustainative training program for future trainers to meet the urgent need of Laos in human resources development assistance under the framework of Thailand – Lao Technical cooperation Program.

PROGRAM FOR IMMEDIATE NEEDS (FROM 2005-2006)

A training program for governmental personnel through a series of training courses on environmental toxicology has been designed and organized by CRI as a training center. The program will be implemented by CRI in cooperation with STEA.

Training will be organized in 3 categories:

1. Organization of Executive seminar in environmental toxicology for sustainable development in Laos. The seminar is designed for senior governmental officials and senior management industrial personnel to make them recognize the negative impacts of chemicals and the importance of toxicology in the protection of human health and the environment. At the same time, the group of resource persons together with the participants will conduct a consultative meeting to survey the status of human resources and specific training requirements in Laos.

2. Implementation of a training program with a series of training courses organized at CRI, Bangkok, Thailand for Laos participants.

Structure of the Training Program:

The training program consists of a series of 5 courses to provide background knowledge from fundamental bases to applied ones. The following are brief descriptions of courses, presented in sequential order.

1. Integrated Life Science (Prerequisite course): January 4 – February 10, 2005

This course aims at providing fundamental knowledge for trainees with no background in biomedical sciences. The course will cover integrated structural and functional organization of life processes at the molecular and cellular levels. The course comprises basic ecology, cell biology, biochemistry, physiology and microbiology. In addition, trainees will be provided with basic concepts and techniques employed in cell biology.

2. Principles of Toxicology: January 10 – February 28, 2005

This course presents the basic concepts of toxicology, including dose-response relationships; types of harmful effects; mechanisms involved in chemical actions from the entrance of chemicals into the body until excretion; toxicokinetics; activation and detoxification of carcinogenesis and mutagenesis; the principles of testing for toxic effects; epidemiology and concepts of risk assessment.

3. Environmental Toxicology: July 18 – 28, 2005

This course will provide the trainee with a background of major groups of toxic substances encountered by man and animals through food and environment and through exposure at the workplace. These toxicants include mycotoxins, naturally occurring toxins, N-nitroso compounds, solvents, plastics, pesticides, pollutants and radiation (UV, electromagnetic, ionizing). The course focuses on the chemistry, fate and distribution of chemicals in the environment, mechanisms of their actions, toxic manifestation in living organisms as well as toxic syndrome in human beings.

4. Environmental and Health Risk Assessment and Management: November, 2005 (dates to be announced)

Emphasis will be placed on potential adverse health effects of human exposure to environmental hazards. This course, which is an integration of science and policy, covers the fundamental basis of the risk assessment and management process that starts from identification of hazard, assessment methods, the inherent uncertainties in each step and the relationship between risk assessment and risk management, and the need for open, transparent and participating acceptance procedures and credible communication methods. The course also teaches the practical application of risk assessment methods to various problems, e.g. hazardous waste site release. The application of environmental impact assessment procedures for identifying and assessing risk is included in the course.

5. Detection of Environmental Pollutants, Testing and Screening of Toxicity: January 23 – February 3, 2006

This is a course of lectures and laboratory exercises on chemical, physical and biological methods currently used to detect chemical pollutants in the air, water and soil, and to detect their biological effects on organs or important changes in the health of people exposed to environmental chemicals. Testing and screening of toxicity will also be included.

The primary objective is to develop human resource in Environmental Toxicology for Laos through the capacity building program in Environmental Toxicology, Technology and Management to cope with the increasing use of chemicals for rapid economic expansion industrialization.

Such human resources are necessary for the enhancement of capacity and endogenous capabilities of Laos for the management of environmental and industrial toxicological problems resulting from chemical use, to promote sustainable development and protection of environment and human health.

CALENDAR OF ACADEMIC EVENTS
Year 2005-2006

INTERNATIONAL TRAINING COURSES IN BANGKOK

Date	Activities	Venue
Jul. 18 – 28, 2005	Environmental Toxicology	CRI
Nov., 2005 (dates to be announced)	Environmental and Health Risk Assessment and Management	CRI
Jan. 23 – Feb. 3, 2006	Detection of Environmental Pollutants, Testing and Screening of Toxicity	CRI

Course in Environmental Toxicology
July 18-28, 2005

Closing Date for Applications:

June 15, 2005

Participants' Qualifications and Requirements:

- The course requires basic knowledge in chemistry, biological sciences and medicine
- Education: Equivalent to a bachelor degree from university/technical college
- Language: English

Program:

- Chemicals in the environment, fate and distribution of chemicals in the environment, prediction of environmental distribution and fate of chemical substances, exposure to chemical hazards
- Water pollutants, soil pollutants
- Environmental carcinogens, environmental carcinogenesis
- Methods of evaluating chemical exposures; biomarkers
- Toxicity of industrial chemicals, e.g. metals, fibers, organic solvents, gases etc.
- Controls for industrial exposures, industrial chemical toxicity, prevention and management of industrial chemical poisonings, prediction of environmental concentration, environmental fate from knowledge of release
- Occupational exposure standards, occupational health problems of thermal stress
- Toxicants in foods, food additives
- Pesticides
- Elimination, transformation and dispersion processes
- Relevant comparative physiology (bio-accumulation and bio-magnification through food webs)
- Testing strategies for the aquatic and the terrestrial environment, testing strategies for bio-accumulation and secondary poisoning
- Assessment of predicted no-effect levels in the main environmental compartments
- Air pollution, possible approaches to assessment of atmospheric effects
- Radiation

Venue:

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Teaching Faculty:

1. **R.C. Shank** Professor and Director, Environmental Toxicology Program and Chairman, Department of Community and Environmental Medicine, College of Medicine, University of California at Irvine, USA
2. **J.H. Duffus** Professor and Director, The Edinburgh Centre for Toxicology, UK
3. **L. Ritter** Professor, Department of Environmental Biology, University of Guelph, CANADA
4. **R.A. Walk** Director, Scientific Affairs and Communications, Philip Morris, USA

USE OF PESTICIDE PRODUCTS IN THE HOME: RISK OF NON-HODGKIN LYMPHOMA IN WOMEN

The U.S. Environmental Protection Agency estimates that approximately 1.2 billion pounds of pesticides were used in the United States in 1999, which was equivalent to 4.4 pounds per capita in the U.S. population. Of these pesticides, 76% were used in agriculture, 11% in other industries, and 13% in homes and gardens; also, they were used by 77% of U.S. households and 1.2 million certified professional applicators. Despite a recent decline in overall usage after a marked increase in the 1950s and 1960s, and despite the fact that registrations of some pesticides found to have unacceptable toxicity have been canceled, there has been a concern about their long-term effects on human health, because some pesticides persist in human tissues, soil, foods, and the home environment.

One of the major health concerns is carcinogenicity. More than 30 pesticides or groups of pesticides have been identified as possible carcinogens to humans by several national and international institutions. Pesticides may increase the risk of cancer through various mechanisms. Some are known to be genotoxic (mutagenic) or tumor promotive, whereas others possess hormonal, immunotoxic, or hematotoxic properties. Furthermore, it has been reported that exposure to certain pesticides synergistically increases the mutagenicity of diet-derived heterocyclic amines. Higher frequencies of chromosome aberrations, sister chromatid exchanges, and micronuclei have been observed in peripheral lymphocytes of pesticide applicators and certain groups of farmers. Because of these chromosome abnormalities, cancers in the hematolymphoid tissues [e.g., non-Hodgkin lymphoma (NHL), Hodgkin lymphoma, multiple myeloma, and leukemia] have been a central issue in the evaluation for potential health consequences of pesticide exposure. Particularly, NHL has received research attention because the recent rapid increase in its incidence parallels an exponential growth in pesticide use with a few decades of lag.

There have been extensive reviews on cancer risk associated with farming and pesticide exposure as well as a number of more recent articles on specific types of cancer and specific classes of pesticides. However, the vast majority of those studies have focused only on occupational exposures, except for some childhood cancer studies in which parental exposures in and around the home were assessed. Because of the widespread use of these chemicals in and around the home and because of the longer time spent at home than at work, especially among women, information about pesticide use around the home is critical to obtain a better picture of the overall effects of pesticides in the general population.

A recent population-based case-control study in upstate New York has been undertaken to determine whether pesticide products used in the home as well as at work are associated with increased risk of NHL among women.

The study involved 376 cases of NHL identified through the State Cancer Registry and 463 controls selected from the Medicare beneficiary files and state driver's license records. Information about history of farm work, history of other jobs associated with pesticide exposure, use of common household pesticide products, and potential confounding variables was obtained by telephone interview. Odds ratios (ORs) and 95% confidence intervals (CIs) were estimated using an unconditional logistic regression model. The risk of NHL was doubled (OR = 2.12; 95% CI, 1.21-3.71) among women who worked for at least 10 years on a farm where pesticides were reportedly used. When both farming and other types of jobs associated with pesticide exposure were combined, there was a progressive increase in risk of NHL with increasing duration of such work ($p = 0.005$). Overall cumulative frequency of use of household pesticide products was positively associated with risk of NHL ($p = 0.004$), which was most pronounced when they were applied by subjects themselves. When exposure was analyzed by type of products used, a significant association was observed for mothballs. The associations with both occupational and household pesticides were particularly elevated if exposure started in 1950-1969 and for high-grade NHL. Although the results of this case-control study suggest that exposure to pesticide products may be associated with an increased risk of NHL among women, methodologic limitations related to selection and recall bias suggest caution in inferring causation.

Source: Environmental Health Perspectives, Vol. 112, No. 13, September 2004.

The Effects of Lead on Blood Parameters and Viscosity

In recent years, increasing attention has been given to the toxic effects of lead on human and animal health. Lead is an ubiquitous metal in the environment that induces a broad range of physiological biochemical and behavioral dysfunctions.

Lead may have toxic effects on several organs, e.g. the hematopoietic system, the peripheral and central nervous system, the kidneys, the gastrointestinal tract, the cardiovascular system, and the reproductive system. Depending on the dose of lead taken up, acute or chronic toxic effects can be caused. In general, soluble lead compounds show more toxic effects than other lead compounds. Lead toxicity occurs at molecular and cellular levels. It has been demonstrated that lead affects various enzymatic systems. It reduces the activity of some enzymes by binding to sulfhydryl (-SH) groups or by replacing other metal ions.

The most obvious toxic effect of lead appears in the hematopoiesis process and the heme biosynthesis. It has been proposed that lead induces anemia in addition to the acknowledged shortened life span of erythrocytes and inhibition of hemoglobin synthesis.

The changes in the quantities of blood cells (erythrocytes, thrombocytes and leukocytes) cause variations in the friction between the blood layers and this friction determines viscosity.

A recent study has investigated the effects of lead on blood parameters and blood viscosity in female rats. In the study 14 female 180-day-old Wistar Albino-type rats weighing 190-220g, were used as experimental animals and divided into a control and a lead exposed group. Both groups were fed with the same standard food, but lead acetate was added to the drinking water of the experimental group for 5 weeks. At the end of the experimental period, blood samples were drawn from the abdominal aorta of the anaesthetized animals. Hematocrit (Hct %), hemoglobin (Hb), and the number of erythrocytes were determined, blood viscosity was measured with a rotational viscometer, and the lead concentration in blood was analyzed by means of flame atomic absorption spectrometry. The erythrocyte count, Hb and Hct % of the lead exposed group were found to be significantly lower than in the control group ($p<0.01$). The blood viscosity level was significantly higher compared to the control group ($p<0.01$). The study concluded that increased lead concentrations in blood impair certain hemorheological mechanisms and increase blood viscosity.

Source: Journal of Trace Elements in Medicine and Biology, Vol. 18, 2004.

OBITUARY

Professor Davide Calamari

It is with the greatest regret that we learned of the passing of Davide Calamari on 31 December 2004 from an illness that he had fought bravely and tenaciously for a number of years. He showed notable dedication at all times in contributing actively and selflessly to our capacity building program for Human Resource Development in Environmental Toxicology and Environmental Management.

Professor Calamari had been a member of Chulabhorn Research Institute's cadre of international experts since 1988, making an important and significant contribution to the International Centre for Environmental and Industrial Toxicology (ICEIT) training program as well as the Post-graduate Program on Environmental Toxicology, Technology and Management.

His colleagues and students will remember him with gratitude and affection. His erudite knowledge of his field of expertise was matched by his excellence as a communicator and teacher. He will be greatly missed.

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