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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".

CRI Launches Regional Capacity Building Project

The Chulabhorn Research Institute has launched a regional project to build capacity in environmental toxicology and management to promote sustainable development in Southeast Asia and in other developing countries in Asia and the Pacific. This project, which will be carried out over a three year period, is supported by the Royal Thai Government and the United Nations Development Programme. Under the project, the human resources development program for the region will train approximately 600 trainees, primarily from Southeast Asian countries, with the organization of 13 training courses, seminars and workshops scheduled over a period of three years from 1998. The project aims to build self sufficiency in the expertise needed to ensure safe and effective use of chemicals and biotechnology to assist in the sustainable development of the region.

Activities in the program will be organized at the Chulabhorn Research Institute in Bangkok, Thailand, for all Southeast Asian and some other developing countries in Asia and the Pacific. In parallel with activities organized in the regional program, in-country training will be organized in Vietnam and Indonesia in order to train a critical number of personnel as subsequent trainers at the national level and to ensure that the training provided is appropriate to the local situation.

The Chulabhorn Research Institute will establish linkages for scientific cooperation with leading institutes in developed countries as well as with international organizations to facilitate technology transfer and the exchange of scientists and information.

The project builds on the Chulabhorn Research Institute's extensive experience over the last eight years in initiating and implementing training programs in environmental and industrial toxicology. It has been found that the lack of well qualified technical personnel is a critical problem in governmental as well as industrial sectors throughout the region. Generally, existing personnel are not specifically trained in environmental toxicology and, in many cases, are mobilized from related disciplines. Extension training is therefore needed to enable key personnel to exercise the safe use, control and management of potentially toxic chemicals, as well as to assess and manage health and environmental risks.

The regional capacity building project will be implemented by the Chulabhorn Research Institute with a project director and a working committee consisting of senior institute staff and a group of world-renowned toxicologists from North America and Europe.

Health Impacts of Indoor Air Pollution

A growing interest in the health impacts of indoor air pollution in the home, as well as in the workplace, has led to increasing research activities and heightened awareness among regulators and policy makers. There are important implications for those involved in the manufacture and supply of building materials, paints, furniture, carpets and other consumer products used in indoor environments.

For a number of pollutants found indoors, the main sources are from outside, since car exhaust gases and fine particles, for example, can readily infiltrate into the home.

It is still common in epidemiological studies to use outdoor air pollutant levels and equate these with health effects in the population. Increasingly, however, the need is being recognised to take much more account of indoor exposures and to understand the importance of personal behaviour patterns.

A large number of natural and man-made chemicals can be identified in the air inside a typical home as well as particulate material and potent allergens. Some of the most important indoor pollutants currently recognised are radon; oxides of nitrogen (NO_2); carbon monoxide (CO); tobacco smoke; formaldehyde; volatile organic compounds (VOCs); chlorinated organic compounds; dust and particulates, PM_{10} (small particles less than 10 µm in diameter); house dust mite allergen; cat allergen; fungi and fungal spores; bacteria; pollen; and asbestos fibres.

Levels of NO_2 in homes reflect the concentrations outdoors but are normally lower unless there is an indoor source. The most important indoor source is cooking with gas, but high mean and peak levels of NO_2 in homes can result from use of other unflued combustion appliances such as butane gas or kerosene heaters. Very high levels of NO_2 (even exceeding those found, for example, during outdoor pollution episodes) can regularly occur in kitchens while cooking with gas.

Volatile organic compounds (VOCs) in the indoor environment originate from a number of sources including furnishings, furniture and carpet adhesives, building materials, cosmetics, cleaning agents and DIY materials. VOCs also originate from fungi, tobacco smoke and fuel combustion. Between 50 and 300 different compounds may occur in a typical non-industrial indoor environment, including aliphatic and aromatic hydrocarbons, halogenated compounds and aldehydes.

By far the greatest potential for exposure to VOCs occurs during home decorating using solvent-based paints. Glues are another important source of high peak levels. The main indoor sources of formaldehyde are pressed wood (chipboard) flooring and furniture, carpets and urea-formaldehyde foam insulation. It is also found as a preservative in a wide range of other household materials and products.

Formaldehyde can produce sensory and airway irritation. Concentrations above 0.1 mg/m^3 may cause transient sensory effects in some individuals, including eye and throat irritation, but most people do not experience any effects at or below this level. Exposure to levels above 0.07 mg/ m^3 has been associated in one study with increased incidence of asthma and chronic bronchitis in children. On the other hand, chamber studies on volunteer asthmatics have shown no effect on lung function of 3.6 mg/m^3 formaldehyde exposure over 180 minutes.

House dust mites occur in virtually all homes, preferring warm, moist microenvironments, including mattresses, pillows and carpets. House dust mites are potentially one of the most important indoor problems because of the role they may play in the incidence and prevalence of asthma.

Although exposure to house dust mite allergen represents a proven hazard to health, the exposure-response relationship between measured allergen levels and development of allergic sensitisation remains poorly defined. The public health benefits of a marginal reduction in mite allergen exposure are thus uncertain, but could be very important. It is therefore sensible to initiate actions and policies to achieve a general reduction in mite allergen exposure in homes. Lower indoor humidity could contribute to reducing house dust mite numbers and hence allergen exposure. More research is needed to establish better exposure-response relations and to evaluate the cost-benefits of possible mitigation strategies.

Many different species of bacteria and fungi can be found in homes, associated with various forms of organic matter such as surface coating of walls, wood, fabrics and foodstuffs. Some species are particularly associated with dampness in buildings and several health effects (other than infections) have been attributed to the bacterial and fungal flora of the indoor environment. There is certainly consistent evidence of an association between damp and mouldy homes and reports of respiratory illness in children, but the causal interpretation of these findings remains unclear and the relationships cannot, at the present time, be attributed to specific fungi or bacteria in the air.

Mould and dampness are often associated with poor ventilation, which tends to increase exposure to other contaminants as well as to microbiological products. Improved ventilation should reduce indoor dampness and mould growth and lower pollutant levels generally. There is a dearth of information on the toxicity of fungi and bacteria and their metabolites, which needs to be addressed, and the general issue of damp housing and health similarly requires further study.

While the main focus of public attention may, for a while at least, remain on outdoor air quality, notably traffic pollution, it is important that the responsible government departments, agencies and research councils continue to devote sufficient attention to the indoor environment.

Within the scientific community too, there is the requirement to consider fully the role of indoor pollution. It is no longer appropriate, for example, to conduct epidemiological studies on the health effects of nitrogen dioxide assuming that exposure is only from outdoors and ignoring the enormous confounding of indoor sources or to assume generally that personal

(Continued on page 8)

MEASURING AIR QUALITY

The Office of Air Quality Planning and Standards of the US Environmental Protection Agency has now made available on the Internet its Pollutant Standards Index (PSI) which has been developed to provide information about daily levels of air pollution.

The EPA uses the Pollutant Standards Index to measure five major pollutants for which it has established National Ambient Air Quality Standards under the Clean Air Act. The pollutants are particulate matter (soot, dust, particles), sulfur dioxide, carbon monoxide, nitrogen dioxide, and ozone*. For each of these pollutants, EPA has established air quality standards protecting against health effects that can occur within short periods of time (a few hours or a day). For example, the standard for sulfur dioxide – that is, the allowable concentration of this pollutant in a community's air – is 0.14 parts per million. For ozone, the hourly average concentration permitted under the standard is 0.12 parts per million.

The PSI converts the measured pollutant concentration in a community's air to a number on a scale of 0 to 500. The most important number on this scale is 100, since this number corresponds to the standard established under the Clean Air Act. A 0.14 ppm reading for sulfur dioxide or a 0.12 ppm reading for ozone would translate to a PSI level of 100. A PSI level in excess of 100 means that a pollutant is in the unhealthful range on a given day; a PSI level at or below 100 means that a pollutant reading is in the satisfactory range.

Levels above 100 may trigger preventive action by State or local officials, depending upon the level of the pollution concentration. This could include health advisories for citizens or susceptible individuals to limit certain activities and potential restrictions on industrial activities. The 200 level is likely to trigger an "Alert" stage. Activities that might be restricted by local governments, depending on the nature of the problem, include incinerator use, and open burning of leaves or refuse. A level of 300 on the PSI will probably trigger "Warning", which is likely to prohibit the use of incinerators, severely curtail power plant operations, cut back operations at specified manufacturing facilities, and require the public to limit driving by using car pools and public transportation. A PSI level of 400 or above would constitute an "Emergency", and would require a cessation of most industrial and commercial activity, plus a prohibition of almost all private use of motor vehicles. If air pollution were to reach such extremely high levels, death could occur in some sick and elderly people, and restrictions on normal activity may be required even in healthy people. Before determining which stage is to be called, officials examine both current pollutant concentrations and prevailing and predicted meteorological conditions.

Significant seasonal variations can occur in PSI-reported values. In winter, carbon monoxide is likely to be the pollutant with the highest PSI levels, because cold weather makes it much more difficult for automotive emission control systems to operate effectively. In summer, the chief pollutant in many communities is likely to be ozone, since emissions of volatile organic compounds and nitrogen oxides form ozone much more rapidly in the presence of heat and sunlight.

The PSI places maximum emphasis on acute health effects occurring over very short time periods – 24 hours or less – rather than chronic effects occurring over months or years. By notifying the public when a PSI value exceeds 100, citizens are given adequate opportunity to react and take whatever steps they can to avoid exposure. The approach EPA follows is conservative, because (1) each standard has built into it a margin of safety that is designed to protect highly susceptible people, and (2) the public notice is triggered as soon as a single sampling station in the community records a PSI level that exceeds 100.

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*Note: Ozone at the ground level can be a health and environmental problem, but ozone is beneficial in the stratosphere (6-30 miles above the earth) where it shields the Earth from the sun's harmful ultraviolet radiation. EPA has programs to reduce chlorofluorocarbons and related substances to protect the stratospheric ozone layer. The PSI relates only to ground-level ozone, a major component of smog.

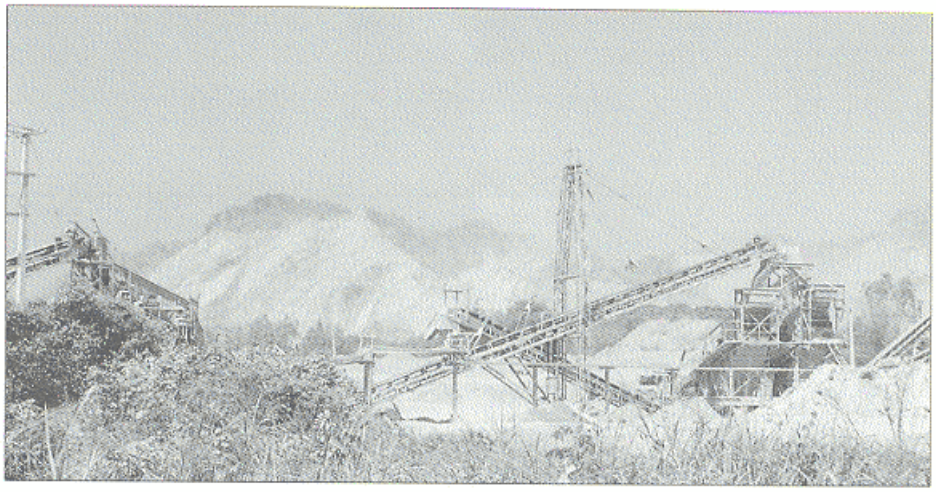
Source: Website – http://www.epa.gov/airprogm/oaqps/psi.html.

Clinical Cases of Silicosis in Thailand

Silicosis, a fibrotic disease of the lungs caused by inhalation of dust containing crystalline silicon dioxide or silica, is of increasing concern in Thailand where the stone-grinding industry has expanded vigorously in recent years.

There are now approximately 6,700 stone grinding factories in Thailand employing more than 1.7 million workers.

In a study of 33 stone-grinding factories in Saraburi, a province 100 km North of Bangkok, 31 of the factories have levels of either total dust or respirable dust exceeding the hygienic limit values, the average levels of total dust and respirable dust being 24.3± 34.6 and 2.4±1.6 mg/m^3 respectively. The prevalence of silicosis in this study was 9%.



The three clinical forms of silicosis – chronic, accelerated, and acute – are determined primarily by the intensity and the length of exposure to silica. Long-term exposure to low concentrations of silica is likely to be associated with a slow progressive nodular infiltration on both lungs, predominantly in the upper lobe. This radiographic abnormality of chronic silicosis usually appears more than 15 years after the onset of exposure. The lesion usually marks a progressive disease, even in the absence of further exposure to silica, and will frequently develop into progressive massive fibrosis. Most patients with chronic silicosis have no symptoms. Among symptomatic patients, dyspnea is common but is usually severe only if there is progressive massive fibrosis.

In accelerated silicosis, exposure to high concentrations of silica over a period of as little as four years results in a more rapidly progressing form of silicosis. The symptom of breathlessness presents early and the patient's condition rapidly deteriorates to hypoxic respiratory failure. This type of silicosis is common in certain occupations, e.g. sandblasting and stone masonry.

Acute silicosis can develop within a few weeks or months of exposure to very high concentrations of silica. The lungs show a ground-glass appearance, similar to that of pulmonary edema. The lesion may consolidate into appearances more characteristic of massive fibrosis over a short period of time. The clinical presentation of this group is usually rapidly progressive dyspnea, cough and weight loss. Death occurs within a short period of time despite intensive treatment.

At present, silicosis continues to be an occupational disease of considerable importance in Thailand. Over the past five years, the number of claims for compensation of silicosis has been increasing. These findings indicate that silicosis is a serious social and economic problem in Thailand.

Source: Asian-Pacific Newsletter on Occupational Health and Safety, Vol.4, No.2, September 1997.

New Ways to Monitor Herbicide Pollution

At the European Congress on Biotechnology held in Budapest in August 1997, researchers reported on developments that could lead to new ways to monitor herbicide pollution.

Genetically modified plants, or plantibodies, could provide a cost effective way of cleaning up water and soil contaminated with herbicides. Researchers at Aberdeen University have genetically engineered tobacco plants to produce antibodies that specifically latch onto the herbicide atrazine. The research team plans to test the plants to see how much atrazine they can tolerate before transferring them to contaminated sites. The first commercial development is likely to be monitoring herbicide pollution in ground and surface water. The plantibodies can detect 100 parts per trillion of atrazine. They could be fixed to molecules that emit fluorescent light when atrazine binds; in this way, the more herbicide that binds, the stronger the signal of emitted light.

(News Item)

Prevention of Silica Dust Hazards in the Workplace

Exposure to silica, the term given to minerals that contain silicon dioxide (SiO_2), is associated with the development of silicosis in workers in a number of industries.

The percentage of crystalline silica in a mineral dust mixture is an important factor in evaluating the respiratory hazard associated with breathing the mixture. Generally, the silica dose to the lungs is highest with exposure to fine dust mixtures containing high percentages of silica. The percentage of crystalline silica found in various materials used during different processes can vary greatly.

In order to determine workers' occupational exposure to silica, the respirable crystalline silica content of the ambient air at the workplace must be quantified. These measurement results can be compared against internationally recognized levels of respirable silica which are considered safe for the average health worker. Quantitative exposure assessment information is also needed to select appropriate engineering controls and respiratory protection. Monitoring of the ambient air at the workplace can be used to evaluate the effectiveness of control measures.

Exposure to silica in the workplace can be controlled in a number of ways. Isolation is the process of enclosing a process in order to eliminate or significantly reduce workers' exposures. In general, isolating involves building an enclosure around a dusty work process. The enclosure must be built as air-tight as possible. It should be properly ventilated to eliminate the build-up of contaminants inside the enclosure. Natural ventilation is sufficient occasionally, but a more complex installation may require forced-air ventilation. One form of enclosure is a cab equipped with efficient supply-air filtration and air conditioning.

Modification of a manufacturing process is one way of reducing workers' exposure. A good example of this is the introduction of water to control dust levels. Wetting down is one of the simplest methods for dust control; its effectiveness depends on the use of proper techniques to wet down the dust. Tremendous reductions in dust concentrations have been achieved by forcing water through the drill bits used in rock drilling operations and by wet sawing of materials containing silica. These methods control the hazards presented by exposure to silica at the source, protecting adjacent workers from exposure. When possible, castings can be cleaned with water rather than with sand blasting, and the addition of moisture to moulding sand decreases airborne silica exposures.

Ventilation is one of the most effective methods available for preventing hazardous substances from entering the workroom atmosphere. Systems are often used ineffectively, however; either they do not provide the required degree of control, or their function and use are not properly understood.

Personal protective devices must be used when neither engineering methods nor work practices are feasible to control or avoid exposure. However, respirators are the least desirable method for controlling workers' exposure. The principles guiding proper selection and use of personal protective equipment at work are embodied in national and international standards. Individual anthropometric and physiological characteristics, as well as respirator comfort, are of vital importance.

Source: Asian-Pacific Newsletter on Occupational Health and Safety, Vol.4, No.2, September 1997.

Smoke Alarm from Forest Fires

In the last three months of 1997, forest fires in Brazil and Indonesia, exacerbated by the dry conditions resulting from El Niño, have raised global awareness of the health effects of breathing smog. Physicians in the remote city of Alta Floresta in central Brazil concluded that half the local population was suffering from respiratory illness caused by smoke from forest fires. In the Amazonian city of Manaus, some 900 kms northwest of Alta Floresta, very significant increases in the number of patients hospitalized with bronchitis were reported.

The atmospheric effects of the Indonesian forest fires have been felt over a similarly wide area of Southeast Asia. The situation in Sarawak has been particularly severe with visibility reduced to tens of meters. At one stage a state of emergency was declared and the 1.9 million people in Sarawak were advised to stay indoors with windows and doors shut. It is thought that one way smog might cause illness is by absorbing ultraviolet light, specifically the band known as UV-B, which is known to kill bacteria and viruses. Readings taken of UV-B in Alta Floresa over periods of dense concentrations of smoke were less than one tenth of the levels on a clear day, and on occasions the UV-B level reached zero. On reduced UV-B days, airborne bacteria that lack internal pigmentation became more common relative to pigmented bacteria. Because most pathogens are nonpigmented it is likely that bacteria and viruses could become more of a health threat under conditions of smog.

(News Item)

Carcinogenetic Effects of Diesel Emissions

Researchers from the National Institute of Public Health in Tokyo and the Kyoto Pharmaceutic University have identified a compound in the exhaust fumes of diesel engines that may be the most strongly carcinogenic ever analysed.

The compound, 3-nitrobenzanthrone, produced the highest score ever reported in an Ames test, a standard measure of the cancer-causing potential of toxic chemicals.

3-Nitrobenzanthrone is a nitrated polycyclic aromatic hydrocarbon (nitro-PAH). It is produced during reactions between ketones, which are by-products of burning fuel, and airborne nitrogen oxides that take place on the surface of hydrocarbon particles in diesel exhaust.

The researchers used the Ames test to measure the number of mutations the compound caused in the DNH of standard strains of bacteria. In a test with a strain of *Salmonella typhimurium,* 3-nitrobenzanthrone recorded over 6 million mutations per nanomole, which compared to a score of 4.8 million for the previously most powerful known mutagen 1,8-dinitropyrene.

The researchers found in a further experiment that the compound caused considerable chromosomal aberrations in the blood cells of mice. This suggests that it may have similar effects on other mammals, including humans.

A noteworthy finding of the Japanese study is a remarkable increase of 3-nitrobenzanthrone in the exhaust emission from a diesel engine working under high loading conditions. The possible connection between engine loading and concentration of nitroarenes in emission suggests a need for greater regulation over the load limit of diesel trucks, and is an area of study in which more research is needed.

Source: Environmental Science & Technology, Vol. 31, No. 10, October 1997.

Dangers of Repeated Exposures to HCFCs

A recent study indicates that there is an urgent need to develop safe alternatives to a mixture of two hydrochlorofluorocarbons, HCFC 123 and HCFC 124, chemicals now commonly used in airconditioners, cleaning agents and solvents as ozone-friendly substitutes for chlorofluorocarbons (CFCs). The World Health Organization reported in 1992 that repeated exposure to HCFC123 caused liver damage in rats, but said that there was no evidence that HCFC 124 is toxic.

However, a study carried out by the Industrial Toxicology and Occupational Medicine Unit of the Faculty of Medicine at the Catholic University of Louvain, Belgium, into the causes of an epidemic of liver disease in nine industrial workers who had repeated accidental exposure to a mixture of HCFC 123 and HCFC 124 indicates that repeated exposure of humans to these chemicals can result in serious liver injury in a large proportion of the exposed population.

Although the exact mechanism of hepatoxicity of these agents is not known, the results of the Belgian study suggest that trifluoroacetyl-altered liver proteins are involved.

The current production of HCFCs 123 and 124 is estimated to be several kilotonnes per year but, because of the ban on CFCs in the Montreal Protocol, is expected to become more widespread during the next few years.

It is, therefore, a matter of the greatest urgency, to ensure that strict measures of containment are implemented to prevent exposure to these chemicals. Medical personnel should also be aware of the potential toxicity when dealing with cases of accidental exposure, not only in the workplace but also in the general environment.

The marked hepatoxicity of HCFCs in the human population as well as their possible carcinogenicity raise concern about their widespread use. There is an urgent need to develop safer alternatives to CFCs.

Main Source: European Chemical News, Vol.68, No.1779, September 1997.

Water Chlorination and Cancer

Water chlorination is one of the major disease prevention achievements of the twentieth century and has become the principal means of effectively reducing water-borne enteric diseases such as typhoid fever, cholera, and dysentery.

However, it is known that chlorine may react with humic substances in water, producing large numbers of halogenated organic byproducts. One of these byproducts, 3-chloro-4-(dichloromethyl)-5-*hydroxy*-2*(5H)*-furanone, or MX, has come under increased scrutiny as a potential cancer risk. A study by researchers in Finland has reported that MX is a potent carcinogen in male and female rats, causing statistically significant increases in tumor incidence in seven different tissues at doses not overtly toxic to rats.

In the study, the researchers gave MX to Wistar rats in drinking water for a period of 104 weeks at average daily doses of 0, 0.4, 1.3 or 5.0 mg/kg for males and 0, 0.6, 1.9 or 6.6 mg/kg for females. The results of the study showed that MX caused the most dramatic increases in the prevalence of follicular adenoma and carcinoma in thyroid glands, and cholangioma in liver. It also caused lymphomas and leukemias and increased the frequency of other endocrinologic tumors.

The data obtained from the study indicate that MX is a definite multisite carcinogen in both male and female Wistar rats. Judging from the number of animals affected, the thyroid glands were the main target organ of the tumorigenic effects of MX in both males and females. The liver was the other tissue in which a strong carcinogenic response was manifested. The most prevalent MX-induced tumor was cholangioma, an epithelial tumor of the bile duct.

The data showed that MX caused lymphomas and leukemias in rats and that, although the numbers of animals with these tumors were low, the tumor frequency in females increased in a dose-dependent fashion and, in males, only MX-treated animals developed leukemias and lymphomas, which were evenly distributed between dose groups.

MX increased the frequency of several other endocrinologic tumors, such as cortical adenoma in adrenal glands, Langerhans' cell adenoma in pancreas, and adenocarcinoma and fibroadenoma in mammary glands.

The mechanisms of the tumorigenesis remain to be elucidated. However, on the basis of its known genotoxic activity and the target organs of tumorigenesis in rats, MX evidently caused direct genotoxic carcinogenicity.

These observations, therefore, identify MX as a potential risk factor for cancers that may be induced in humans who consume chlorinated drinking water.

Potential risks from MX must, however, be weighed against the proven benefits of chlorination, and the US Environmental Protection Agency is currently evaluating chemical and microbial risks of various disinfection processes.

Source: Journal of the National Cancer Institute, Vol.89, No.12, June 1997.

Surveillance Tests for Benzene Exposure in the Workplace

A study undertaken by researchers at the Institute for Medical Research and Occupational Health, Croatia, investigated the effects of benzene on forty-nine female workers in the shoemaking industry exposed to a solvent containing benzene, compared to a control group of twenty-seven workers who were not exposed to the solvent.

Forty-nine female workers in the shoemaking industry, exposed to a solvent mixture containing benzene and twenty-seven non-exposed controls, were investigated. Concentrations of benzene and toluene in the working atmosphere, as well as benzene and toluene in blood and phenols in pre-and post-shift urine as parameters of biological monitoring, were determined. In order to assess hematotoxic risk, a complete blood cell count with differential, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, reticulocytes, serum iron, alkaline phosphatase in neutrophils and red blood cell glycerol lysis time were determined in all subjects.

Benzene concentrations in the workplace atmosphere at the shoemaking factory ranged from 1.9 to 14.8 ppm (median = 5.9). Significant differences in benzene in blood and phenol in post-shift urine between exposed workers and controls confirmed exposure to benzene. Hemoglobin level and mean corpuscular hemoglobin concentration in the shoe workers were lower, and band neutrophils ($p = 0.005$) and mean corpuscular volume higher than in controls. Red blood cell glycerol lysis time was significantly higher in shoe workers than in controls and showed a significant correlation with exposure biomarkers.

Most benzene-related hematologic effects have been reported in humans occupationally exposed to relatively high concentrations, while the capacity of benzene to cause hematopoietic damage in low concentrations remains controversial. Data are lacking concerning a potential threshold concentration below which adverse effects do not occur in exposed individuals. Hence, adequate health monitoring of early adverse effects have been questioned.

The Croatian study found that in workers exposed to a benzene concentration below 15 ppm, there were no cytopenic effects, while lower hemoglobin and mean corpuscular hemoglobin concentration and higher mean corpuscular volume and band neutrophils were found. Also, an increase in resistance to the hemolytic action of glycerol, as a possible early sign of benzene hematotoxicity, was observed. In combination with mean corpuscular volume, red blood cell glycerol lysis time could distinguish benzene induced hematotoxicity from other hematologic diseases. Therefore, red blood cell glycerol lysis time could be suitable as a workplace surveillance test. The results of the study suggest that potential threshold concentration for hematologic effects of benzene is below 15 ppm.

Source: Toxicology and Industrial Health, Vol.13, No.4, 1997.

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exposure relates directly to concentrations measured at outdoor sampling stations. There has been considerable debate about the role of fine particles (PM_{10}) and health, stemming largely from epidemiological studies using outdoor measurements. There must be much to learn about this relationship by studying indoor and personal exposures.

Importantly, our accumulating knowledge about sources, levels and health impacts of indoor air pollutants needs to be put to good effect to ensure that the home provides the healthiest possible environment.

Source: Chemistry & Industry, No.17, September 1997.

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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