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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 SIGNING OF A MEMORANDUM OF UNDERSTANDING BETWEEN THE CHULABHORN RESEARCH INSTITUTE AND THE KOREA INSTITUTE OF SCIENCE AND TECHNOLOGY MAY 2005

The close relations that have been established over a number of years between the Chulabhorn Research Institute (CRI) and the Korea Institute of Science and Technology (KIST) were further enhanced when on 17 May 2005 a Memorandum of Understanding was signed between the two institutes with HRH Princess Chulabhorn, President of CRI, and Dr. Youseung Kim, President of KIST, taking part in the signing ceremony in Seoul.

Under the terms of the Memorandum of Understanding both institutes will explore opportunities for mutually beneficial joint research and development in areas of common interest including environmental toxicology, natural products, organic synthesis and medicinal chemistry.

The agreement to cooperate in furthering scientific and technological advancement is most appropriate since both CRI and KIST are pioneers in designing and launching innovative postgraduate programs that emphasize the importance of multidisciplinary studies linking the life sciences and environmental technology.

In January 1998, CRI in collaboration with Asian Institute of Technology (AIT) and Mahidol University (MU) opened two inter-university postgraduate programs at Master's and PhD levels in Environmental Toxicology, Technology and Management (ETTM). These innovative multi-disciplinary programs combine health sciences, biotechnology and environmental engineering. And KIST, in 2001, inaugurated the International Research and Development

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Academy (IRDA) offering students a multi-disciplinary hands-on approach to learning, and thus extending opportunities to pursue individual research activities.

The present Memorandum of Understanding provides a framework for the exchange of technical personnel, exchange of information, promotion of joint research and academic cooperation which will be to the mutual benefit of both institutes.

Air Pollution and ST-Segment Depression in Elderly Subjects

Associations of acute increases in particle levels with increased risk of cardiac morbidity and mortality have been demonstrated in a number of studies in which efforts have been directed toward understanding mechanisms for these associations.

In a study carried out in Helsinki, Finland, both carbon monoxide (CO) and particulate mass <2.5 μg/m^3 ($PM_{2.5}$) were associated with increased risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease in 45 adults with stable coronary heart disease. $PM_{2.5}$ was believed to be the primary source of this association, but because of correlation with CO, the Finnish researchers reported that independent effects were difficult to separate. It may be that black carbon (BC) is a more precise measure than $PM_{2.5}$ of the portion of participle mass related to traffic. Thus a recent study carried out in the United States has examined whether there were independent associations of the ambient traffic-associated pollutants BC and CO with ST-segment depression before and after submaximal exercise in a community-based repeated-measures study of elderly adults from Boston, Massachusetts.

The study included 269 observations on 24 active Boston residents 61-88 years of age, each observed up to 12 times from June to September 1999.

The protocol involved continuous Holter electrocardiogram monitoring including 5 min of rest, 5 min of exercise outdoors, 5 min of recovery, and 20 cycles of paced breathing. Pollution-associated ST-depression was estimated for a 10th – to 90th-percentile change in BC. The average ST-segment level, referenced to the P-R isoelectric values, was calculated for each portion of the protocol. The mean BC level in the previous 12 hr, and the BC level 5 hr before testing, predicted ST-segment depression in most portions of the protocol, but the effect was strongest in the postexercise periods. During postexercise rest, an elevated BC level was associated with –0.1 mm ST-segment depression (p = 0.02 for 12-hr mean BC; p = 0.001 for 5-hr BC) in continuous models. Elevated BC also predicted increased risk of ST-segment depression ≥ 0.5 mm among those with at least one episode of that level of ST-segment depression. Carbon monoxide was not a confounder of this association. ST-segment depression, possibly representing myocardial ischemia or inflammation, is associated with increased exposure to particles whose predominant source is traffic.

Source: Environmental Health Perspectives, Vol. 113, July 2005.

Health Problems Caused by Chronic Exposure to Cement Dust

Cement dust causes health problems in cement workers. In an industrial setting, inhalation and skin contact are the most important routes of entry of chemicals present in the dust.

Cement dust is a gray powder manufactured from clay and limestone. Molecules of primary importance in cement are CaO; SiC_2, Al_2O_3; SO_3, and Fe_2O_3.

In the presence of water, SiO_2 becomes hydrated to form silanol groups (-SiOH). Silica dust reacts with lung cells, leading to peroxidation of membrane lipids and damage to cell membranes.

Silica dust stimulates the generation of reactive oxygen species (ROS) from alveolar macrophages, which overwhelms antioxidant defenses of the lung and causes lipid peroxidation and cell damage. Such damage may lead to scarring or destruction of alveolar septa. It is believed that the high reactivity of crystalline silica to biologic membranes is due to the unique properties of these surface – SiOH groups.

In a new study carried out in Turkey, researchers investigated plasma malondialdehyde (MDA) levels as an index of lipid peroxidation and erythrocyte reduced glutathione (GSH, non-enzymatic antioxidant) levels as an index of antioxidant status in cement factory workers. Also correlations were made between SiO_2 levels and respiratory function tests and plasma MDA, erythrocyte GSH levels.

Subjects selected for this study were 48 non-smoker volunteer male workers in a cement factory in Afyon, who have been working there for more than 10 years. They were aged between 24 and 55 years.

Worksite pulmonary function test results; forced expiratory volume in 1 second (FEV_1), forced expiratory volume in 1 second % (FEV_1%pred), forced midexpiratory airflow 25-75 (FEV_{25-75}), forced midexpiratory airflow 25-75% ($FEV_{25-75\%}$) were measured from each worker by using methods that adhered to the recommended techniques of the American Thoracic Society.

Working conditions were evaluated in all workplaces. Free silica fractions in dust samples were determined by using an infrared spectrophotometer. First, dust and silica content were measured in respirable air. Personal Dust Sampler consists of two parts, one of which is a measurement unit carried on a worker, to which a dust collecting reservoir is attached via a transparent tubing. The workers carried the measurement unit for 1 h on themselves. The amount of dust was measured by gravimetric analysis. Second dust analysis was performed from aggregates collected from the floor of working places. For these measurements X-ray diffraction method was used.

The study found that respiratory function tests were reduced in cement-dust exposed workers. A negative correlation was observed between plasma MDA and FEV_1 and plasma MDA and FEV_1% but no correlation was observed between erythrocyte GSH and these parameters.

Based on the updated use of prophylactic and therapeutic strategies it is unlikely that a single strategic approach will suffice for adequate treatment of the whole spectrum of mineral particle-induced pulmonary inflammation, fibrosis, and carcinogenesis. Hence, additive and possibly synergic effects between a number of intervention strategies need to be considered in the hope of finding the best possible intervention strategy for lung fibrosis and cancer. Perhaps the future of prophylactic and therapeutic interventions lies in the combination of treatments with different agents which act by various mechanisms. Furthermore, diets rich in fruits and vegetables combined with pharmaceutical interventions with drugs which counteract inflammation, oxidative stress and synthesis may offer the greatest promise in counteracting particle induced pathogenicity. These treatments should be on an ongoing basis, since mineral particles which are not cleared are durable and persist within the lung for long periods of time.

Source: Toxicology 207, February 1, 2005.

BLOOD LEAD LEVEL AND RISK OF ASTHMA

Lead poisoning is a serious environmental health hazard for many children in the United States, particularly children from backgrounds of low socio-economic status and children of minority status. The epidemiology of pediatric asthma and that of lead poisoning are strikingly similar. Both are prevalent among minority children, and elements in the physical environment increase risk of disease.

Published analyses suggest that lead exposure may result in alterations to immune system components known to be associated with asthma. Lead has been associated with the increased production of total immunoglobulin E (IgE), which is also observed in atopic and nanotopic individuals with asthma.

It has been hypothesized that differential risk of lead poisoning among urban minority children may contribute to increased risk of asthma in this population. On the basis of this hypothesis a recent study has used encounter and claims data to examine relationships between blood levels (BLL) and development of asthma by race in a study population of enrollees of a large, non-profit managed care organization (MCO) in southeastern Michigan, served by physicians in a staff model medical group.

The MCO has a stable population of > 250,000 with a mean enrollment of 9.3 years. The MCO enrollment database and the associated laboratory database (all lead screens are sent to a single central laboratory) were linked to identify children born on or between 1 January 1994 and 31 December 1997, enrolled

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SPECIAL ANNOUNCEMENT

The Chulabhorn Research Institute, Ministry of Natural Resources and Environment, and Ministry of Public Health

will organize the following event

Scientific Conference on Asia Pacific Environmental Health

– Significant, Emerging and Current Challenges, Research and Capacity Building Opportunities, Collaborative Response Needs –

supported by

WHO, UNEP, ADB and UNDP

December 8 – 11, 2005
at the Convention Center, Chulabhorn Research Institute, Bangkok, Thailand

Conference programme

The Conference will identify and discuss ***significant and emerging environmental health*** issues that are current, emerging and expected in the horizon to provide an early alert. It will also provide the forum for discussing and determining the critical linkages of environment, health and development, which give rise to the significant issues.

The Conference sessions will also address the special problems and challenges faced by vulnerable groups, such as, children, the elderly and pregnant women.

Current scientific knowledge and the gaps for research, education, training and capacity building will cut-across the issues reviewed and discussed.

The Conference structure will be interactive and deliberative, with presentations of scientific papers and round tables.

Participants will be from Ministries of Environment, Health, Development and Planning, international organizations, public and private sectors, research institutions, and academia, from ASEAN and East Asian Countries as well as international experts of renown.

Keynote Lectures by

Prof. Dr. Her Royal Highness Princess Chulabhorn
Dr. Klaus Toepfer *(UNEP Executive Director)*
Dr. Paul Crutzen *(Nobel Laureate in Chemistry)*

Invited Speakers (partial list)

Herman Autrup *(University of Aarhus, Denmark);* **Martin van den Berg** *(IRAS and University of Utrecht, The Netherlands);* **John Clemens** *(International Vaccine Institute, Republic of Korea);* **Stephen J. Connor** *(Columbia University, USA);* **Maxx Dilley** *(UNDP, Geneva, Switzerland);* **John Essigmann** *(MIT, USA);* **Yannick Glemarec** *(GEF/BDP/UNDP, NY, USA);* **John Groopman** *(Johns Hopkins University, MD, USA);* **Nasir Mohd. Hassan** *(Universiti Putra Malaysia);* **Nay Htun** *(Imperial College of Science, UK);* **Andrew Hudson** *(International Waters, GEF/BDP/UNDP, NY, USA);* **Michael McElroy** *(Columbia University, USA);* **Tony McMichael** *(Australian National University, Australia);* **Jenny Pronczuk de Garbino** *(WHO, Geneva, Switzerland);* **Twisuk Punpeng** *(Ministry of Public Health, Thailand);* **Khunying Mathuros Ruchirawat** *(CRI, Thailand);* **Ram Sasisekharan** *(MIT, USA);* **Surendra Shrestha** *(UNEP);* **Kirk Smith** *(University of California, Berkeley, USA);* **Jeff Spickett** *(University of Technology, Western Australia);* **Jin Yinlong** *(Institute for Environmental Health and Related Product Safety, China).*

Conference Sessions

Emerging Environmental Health Challenges

- Climate Impacts on Environmental Health
- Transboundary Air Pollution and Environmental Health
- *Luncheon Lecture:* Vaccines R&D and Water Borne Diseases
- Ecosystem Change and Human Health
- International Risk Assessment and Management of Chemicals with Focus on Persistent Organic Pollutants
- An Overview of Exposure Assessment and Biomonitoring
- An Overview of Special Needs of the Vulnerable Groups in Environmental Health

Significant Environmental Health Challenges

- Challenges in Rural and Peri-Urban Settings
- *Luncheon Lecture:* Life Sciences and Environmental Health Translation Research
- Challenges in Urban Settings
- Research, Education, Training and Financing for Significant and Emerging Environmental Health Challenges
- ROUNDTABLE: Opportunities and Modalities for Increasing Regional Cooperation In Environmental Health
- Synthesis Summary and Highlights

Registration* – *Registration Fee – US$ 500

Registration fee includes accommodation and full board.

(Places will be allocated on a first come first served basis)

Additional information:

Scientific Conference on Asia Pacific Environmental Health
Chulabhorn Research Institute
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EFFECTS OF FETAL EXPOSURE TO DIESEL EXHAUST ON ADULT RATS

A study has been conducted to examine possible defects in Sertoli cell differentiation in mature rats following the previously reported defect at gestational day 20 caused by prenatal diesel-exhaust exposure. Dose-response studies of diesel- and filtered-exhaust were also conducted to determine the no-observed adverse-effect level and the type of agents that have toxicity on the differentiation process. Serum hormones were measured to determine whether the function of the hypothalamo-pituitary complex unit was permanently depressed by prenatal exposure to diesel exhaust.

Thirty-six pregnant Fischer rats were divided into five groups. Group 1 was exposed to high dose total diesel-engine exhaust (total), group 2 was exposed to high dose filtered exhaust without particles (filtered), group 3 was exposed to low dose total diesel-engine exhaust (low-total), group 4 was exposed to low dose filtered exhaust (low-filtered) and group 5 was exposed to clean air (control). Exposures began on day 7 (day of impregnation = day 0) and continued until delivery. The exposure period was 6 h daily.

The number of pregnant rats for groups 1, 2, 3, 4 and 5 were 7, 7, 7, 7 and 8, respectively. Litter sizes ranged from 6 to 11. The total number of pups (male:female) from the mothers of each group were 57 (35:22), 57 (29:28), 55 (31:24), 64 (28:36) and 66 (33:33), respectively. Pups were kept with their mothers until they were weaned at 22 days of age. At the same time, the young rats were divided into groups of males and females; male rats were randomly assigned to groups of 6-8, housed in a single cage.

At the end of the experiment, body weights were measured and blood samples were used to measure the titer of serum hormones collected from the abdominal aorta under ether anesthesia on day 96 after birth.

Serum testosterone and estradiol levels of rats were determined using Enzyme Immunoassay Kits. Serum levels of luteinizing hormone (LH) and follicle stimulating hormone (FSH) were determined using a rat LH enzyme immunoassay system and a rat FSH enzyme immunoassay system respectively.

The present study provides evidence for the first time that mature male rats exposed to diesel exhaust during the fetal period showed a decrease in the daily production of sperm due to an insufficient number of Sertoli cells.

The number of Sertoli cells in the rats prenatally exposed to diesel exhaust was smaller than that in the control group on day 96 after birth. It is thought that the peak of cell division of Sertoli cells following differentiation occurs on day 20 of gestation, and the population of Sertoli cells in the adult animal is determined by 3 weeks after birth.

Regarding which substances were responsible for the reduced number of Sertoli cells, filtered exhaust might have been responsible, because total-exhaust exposure and filtered-exhaust exposure had almost the same effects. Filtered exhaust contains the remainder of diesel exhaust after filtration, including gases and ultrafine particles measuring less than 0.1-0.2 µm. Recently, the toxicity of ultrafine particles less than 0.1 µm has been reported, including the direct invasion of ultra fine particles through the lung, the translocation of deposited ultrafine particles to extrapulmonary tissues, and the toxicity of co-pollutants adsorbed to ultrafine particles. Thus it cannot be ruled out that ultrafine particles inhaled or transferred from the mother to the fetus act as toxicants.

Source: Science Direct Toxicology Letters 155, January, 2005.

EFFECT OF THE ORGANOCHLORIDE INSECTICIDE CHLORDANE ON HUMAN ERYTHROCYTES

Organochlorine pesticides, residues and metabolites, are ubiquitous in the environment because of their wide-spread use. Thus, the risk for human exposure and contamination is considerable. Chlordane is a member of the persistent organic pollutants, characterized by extremely long residence in the environment after application. It has been used for more than 50 years as a broad-spectrum contact insecticide, mainly in non-agricultural crops and on animals. In the USA its use is now restricted to underground termite control, and in several other countries approved uses have been gradually withdrawn. Nevertheless, due to its long half-life of 10-20 years it is still detected in indoor and outdoor residences, field soils, edible roots, aerial tissues of several crops, and pasteurized milk. However, the main source of exposure of the general population is through residues in freshwater and marine food chain, particularly from fat of marine mammals. As a result, humans consuming diets high in this kind of food can ingest levels of chlordane as high as 0.32-0.43 µg/kg/day. Chlordane and its metabolites can be detected in a variety of human biological tissues such as blood, brain, adipose tissue, liver, breast milk, kidneys, and urine. It has also been reported to cause protracted neurotoxicity in humans. In cases of intoxication, chlordane blood concentrations have been found to be highly elevated. Still, human studies are contradictory and fail to support a consistent association between this agent and hemotoxicity.

In the course of in *vitro* systems search for the toxicity screening of chemicals, different cellular models have been applied to examine the averse effects of pesticides in isolated organs. The present study examines the interaction of chlordane with human erythrocytes as well as with molecular models of the erythrocyte membrane. The cell membrane is a diffusion barrier that protects the cell interior. Therefore, its structure and functions are susceptible to alterations as a consequence of interactions with foreign species.

Results of the present study indicate that chlordane interacted with the human erythrocyte membrane, inducing an alteration of its morphology from the normal discoid shape to a spherocytic form.

The fact that chlordane induced the formation of spherocytes indicates that this insecticide located in both the outer and inner monolayers of the red cell membrane. This conclusion is supported by the results obtained from the X-ray diffraction study of bilayers composed of DMPC and DMPE, which represent phospholipid classes located in the outer and inner monolayers of human erythrocyte membrane, respectively.

Although the precise molecular mechanism of this insecticide toxicity is still not known, the experimental results of this study confirm the important role played by the phospholipid bilayer of cell membranes.

Source: Food and Chemical Toxicology, May 2005.

Dietary Modulation of Parathion-Induced Neurotoxicity in Rats

Among nutrients, sugars as a class are preferred by most of the population because of their high palatability and hedonic properties. It has been estimated that 11% of energy intake in the US population and 16% in children 2-19 years of age come from "added" sugars in the diet. While the suggested sugar content in a 2200 calories/day diet is 57 g, the average daily intake of sugars by Americans in 1994 was about 182 g. On the average, approximately 45 g/day (≈25% of total) of sugars were consumed in the form of soft drinks. About 42% of children 6-11 and 60% of adolescents 12-19 consume non-diet soft drinks daily. The average 13-18 years old male consumes more than two 12-oz beverages a day, while 5% of this sub-population consumes five or more. High fructose corn syrup (HFCS) is the most commonly used sweetener in soft drinks and a variety of food products. The annual average consumption of HFCS among Americans was 1.5 pounds per capita in the early 1970s; by the mid-1990s however, the average consumption of this sugar source had increased to 56.9 pounds per capita per year.

Previous studies have indicated that dietary glucose (15% in drinking water) could markedly exacerbate the toxicity of parathion in adult rats. The present study evaluated the effect of consumption of the commonly used sweetener, high fructose corn syrup (HFCS), on parathion toxicity in adult and juvenile rats. Animals were given free access to either water or 15% HFCS in drinking water for a total of 10 days and challenged with parathion (6 or 18 mg/kg, s.c., for juveniles or adults, respectively) on the 4th day. Signs of cholinergic toxicity, body weight and chow/fluid intake were recorded daily. Acetylcholinesterase (AChE) activity and immunoreactivity (AChE-IR) in frontal cortex and diaphragm were measured at 2, 4, and 7 days after parathion. As HFCS was associated with significant reduction in chow intake, adult rats were also pair-fed to evaluate the effect of similar reduced chow intake alone on parathion toxicity. The results indicated that the cholinergic toxicity of parathion was significantly increased by HFCS feeding in both age groups. The excess sugar consumption, however, did not significantly affect parathion-induced AChE inhibition in either tissue or either age group. Enzyme immunoreactivity in frontal cortex was generally not affected in either age group while diaphragm AChE-IR was significantly reduced by parathion and HFCS alone in adult animals at 2 and 4 days timepoints, and more so by the combination of sugar feeding and parathion exposure in both age groups. Food restriction alone did not exacerbate parathion toxicity. While the mechanism(s) remains unclear, the study concludes that voluntary consumption of the common sweetener HFCS can markedly amplify parathion acute toxicity in both juvenile and adult rats.

Source: Toxicology, June 2005.

Early Behavioral Effects of Lead Perinatal Exposure in Rat Pups

A recent research study has investigated the effects in early behavior of rats exposed to low lead levels during different phases of pregnancy. The study correlated blood lead levels and ultrasonic vocalizations (USVs) while checking motility, body temperature and weight of pups.

The study found that the perinatal exposure to lead produced a different behavioral effect depending on the developmental phase of the pups with a low rate of calling when a high behavioral outcome was expected, on the 7th postnatal day and a high number of calls of the 14th day old pups, when no vocalizations were expected.

These results suggest that there was a displacement of the normal vocalization drive in young pups by lead and, also, that ultrasonic vocalizations may represent an early indicator of neurotoxicity, correlated to blood lead levels, and sensitive to subtle functional biological changes as the ones produced by dopamine receptor blocking agents or aluminum, at dose levels below those associated with overt signs of neurotoxicity.

The results further suggest that even blood lead levels lower than 10 µg/dl could be deleterious to rat pups, since some mothers treated with the lower dose of lead had a lower blood level on the 10th day of pregnancy and their pups' vocalizations changed with some pups from groups of low lead dosing having low lead levels and vocalization changes.

In the clinical field, lead levels, between 10 and 20 µg/dl, are considered to be of concern and are here demonstrated to be highly correlated to important behavioral changes in infant rats. The acoustic analysis of human baby cry was seen to provide an early marker for specific brain damage produced by toxic agents and that it is a sensitive and selective index for measuring the effect of pre and perinatal lead exposure. The changes in cry parameters with age could represent maturation, in which various aspects of reflex immature cry behavior develop to be under willful control. Regardless of the origin of the effect of lead on infant vocalization, it may alter the infant-caretaker communication and may expose the infant to further risk of developmental delay due to its impact on the social interaction with the caretaker. This may also be another reason why the 14-day-old pups of this study had a change in vocalizations.

In sum, the findings in the present study suggest that ultrasonic vocalization may represent a useful test in detecting minor lead exposition during the developmental period of rats. These behavioral consequences were observed in rat pups at low blood lead levels for children and may be a red flag with respect to the lead range to be accepted in human toxicological evaluations.

Source: Toxicology 211, July 1, 2005.

PRENATAL PHTHALATE EXPOSURE CAN DISRUPT DEVELOPMENT OF REPRODUCTIVE ORGANS OF MALE INFANTS

A study carried out by scientists from the University of Rochester, the National Center for Environmental Health and other centers across the United States, has presented the first evidence that common chemicals used in products as diverse as cosmetics, toys, clingfilm and plastic bags may harm the development of unborn baby boys.

The researchers measured the levels of nine widely used phthalates in the urine of pregnant women and compared them with standard physiological measurements of their babies.

Tests showed that women with higher levels of four different phthalates were more likely to have baby boys with a range of conditions, from smaller penises and undescended testicles to a shorter perineum, the distance between the genitals and the anus. The differences, say the authors, indicate a feminization of the boys similar to that seen in animals exposed to the chemicals.

Phthalates have become ubiquitous in modern society. Some of these oily substances are used as solvents, but most serve as softeners, making rigid materials more flexible. Manufacturers worldwide produce an estimated billion pounds of phthalates annually.

Source: Science News Online, June 2005.

BLOOD LEAD LEVEL AND RISK OF ASTHMA

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in the MCO at time of birth, and with laboratory results for a lead screen performed between 1 January 1995 and 31 December 1998. When results existed for more than one lead screen for an individual child, the highest BLL recorded within the study ascertainment period was used as the baseline BLL. Usually this was the first BLL documented. The resulting data set was linked to the patient encounter database to obtain all ambulatory and inpatient visits, as well as demographic information, including child's date of birth, race, and residential address. The pharmacy claims database provided information on drug claims for asthma medications.

MCO enrollment and disenrollment dates were used to calculate the person-years that each child contributed to the cohort. Geographic information system (GIS; Mapping Solutions, LI.C, Laming, MI), a computer mapping and analysis technology capable of linking geographic with demographic information, was used in conjunction with patient address and census data. Each study participant was assigned the average income per person in the block group of residence (a subdivision of a census tract representing a city block).

The method for obtaining birth weight for children in this cohort was approved by the State of Michigan Division for Vital Records and Health Statistics in addition to the HFHS institutional review board. Identifiers for members of the study cohort were matched, at the State of Michigan Division for Vital Records and Health Statistics, to live birth records. Birth record identifier fields were not supplied to the researchers. Matches outside of the state were censored. The resulting match rate was 97.8%.

Blood samples were obtained by venipuncture, collected in ETDA tubes, and shipped at room temperature to the HFHS chemistry laboratory. Lead was measured in the blood using graphite furnace atomic absorption spectrophotometry, with detection limits of 1 µg/dL.

Lead poisoning and asthma jeopardize the health and quality of life of urban minority children in the United States. The present study sought to evaluate the contribution of BLL to the increased risk of asthma among African Americans. BLL was less a predictor of asthma than was race and did not affect the relationship of race to prevalent or incident asthma. Because lead poisoning and asthma share risk factors that are heavily influenced by SES, it is difficult to obtain an unbiased estimate of the true relationship. Previous studies have shown an association between BLLL and serum IgE, and because serum IgE is observed in both atopic and nanotopic asthma, it was of interest to determine whether a relationship between BLL and development of asthma could be demonstrated using secondary data sources.

An elevated risk of asthma was observed among children exposed to lead, although these associations were not always statistically significant and were observed only for certain subgroups. Three interesting findings can be garnered from this study. First, a trend toward elevated risk estimates for asthma was observed for BLL at a cut point lower than what is currently considered toxic. Second, in addition to a trend toward increased risk at lead levels ≥ 5 µg/dL, the elevated risk was observed consistently only for Caucasians. Although the risk of developing asthma was significantly increased for African Americans when compared with Caucasians with BLL < 5 µg/dL, the risk was not dependent on BLL; that is, African Americans with BLL < 5 µg/dL were also at increased risk of asthma. Third, although the results are inconclusive regarding a dose-response relationship for BLL and asthma, among African Americans BLL ≥ 10 µg/dL held a higher risk of asthma than did BLL ≥ 5 µg/dL. Among Caucasians, the adjHR for BLL and incident asthma increased as the asthma definition became more stringent. However, because BLL is an inadequate dosimeter of lead exposure, a dose-response relationship between BLL and asthma may not be observed in the data, if indeed such a relationship exists.

The racial differences observed are of interest. It was clear that African Americans were at a significantly higher risk of developing asthma when compared with Caucasians, regardless of BLL. The effect of BLL on the immune system of African-American children may be masked by more influential factors working to increase risk.

The data showed a trend toward an elevated risk of developing asthma in Caucasian children with evidence of BLL of < 5 µg/dL before the age of 3 years.

Assessment of the effect of BLL on IgE may provide a better understanding of the etiology and prevention of atopy and asthma. African Americans were at an increased risk of asthma when compared with Caucasians, but if there were any effects related to BLL, they were not observed. The racial differences observed in this study illustrate the need for further exploration of the role of race in the interrelationships between genetic susceptibility, socioenvironmental exposures, and risk of asthma.

Source: Environmental Health Perspectives, Vol. 113, July 2005.

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.

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