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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".

Presentation of EMS Hollaender Award to Professor Dr HRH Princess Chulabhorn

30 April 2002

HRH Princess Chulabhorn receives the EMS Hollaender International Award from Dr. David M. DeMarini, President, EMS

HRH Princess Chulabhorn was honoured for her outstanding contribution to the field of environmental mutagenesis with the presentation of this year's Environmental Mutagen Society (EMS) Alexander Hollaender International Fellow award at the 33rd annual meeting of the Society held from April 27 to May 2, 2002 at the Hilton Hotel, Anchorage, Alaska. The award presentation ceremony was part of the program of the EMS 2002 International Conference: Frontiers Beyond the Human Genome. The award is conferred by the Alexander Hollaender Committee for outstanding contributions in the field of environmental mutagenesis in research, training and promotion of international activities.

In her acceptance speech delivered to an audience of eminent scientists, HRH Princess Chulabhorn described how her own vocation as a scientist had been greatly influenced by the work of her father King Bhumibol whose design and oversight of the royally initiated projects had contributed so significantly to improvements in the quality of life of the Thai people. With her increasing concern with the humanitarian applications of science and technology, Her Royal Highness' own research interests have in recent years experienced a shift from organic chemistry, in which she received her initial scientific training, to the areas of toxicology and carcinogenesis where research applications address issues of immediate human concern.

Sources of pesticide exposure in children

In many countries, particularly in the industrialized world, children can be exposed to pesticides through a range of means including carpets, house dust and chemically treated lawns and gardens. Another potential source is pets treated with parasite control products. In the United States, dogs are the most popular household pets. Together with other pets and domestic animals commonly treated for insect pests, they could be a source of insecticide exposure to the millions of children who come into direct contact with them.

Parasite control products often contain carbamate, pyrethroid, or organophosphate insecticides. One such substance, the organophosphate chlorpyrifos, used until recently in the United States in commercial dips to treat dogs and cats for fleas and ticks, has been the focus of research carried out by scientists at Mississippi State University. The study set out to estimate the amount of transferable residues that children could obtain from their treated pets. The scientists found that young children could receive chlorpyrifos doses nearly equal to the U.S. Environmental Protection Agency (EPA) reference dose, or the amount a person can be exposed to daily without risk of adverse health effects over a lifetime. Although chlorpyrifos has since been withdrawn from the domestic use market, human exposure to other flea- and tick-control pesticides from pets and livestock could occur in much the same way.

Twelve dogs of similar breeds and weights were dipped with a nonprescription commercial flea dip, according to the manufacturer's guidelines, for four consecutive treatments at three-week intervals, with no shampooing in between. Another 12 dogs were shampooed in between dips. Chlorpyrifos samples were taken from the dogs' fur before and after dipping by rubbing the animals with cotton gloves.

Samples collected at 4 hours and at 7, 14, and 21 days after treatment from dogs that were not shampooed averaged 971, 157, 70, and 26 µg chlorpyrifos, respectively. Samples from dogs that were shampooed averaged 459, 49, 15, and 10 µg, respectively. Most of the transferable chlorpyrifos residues dissipated after 3 weeks, with the sharpest decrease after 1 week.

Humans are therefore at the greatest potential risk of exposure to chlorpyrifos shortly after treatment. Children, particularly, spend much time with pets and may be more sensitive to pesticide exposures than adults, so plausible estimates of exposure levels are needed to calculate risk.

Currently, there is no perfect technique for measuring human exposure to flea- and tick-control pesticides from pets and livestock and assessing the attendant risk. Given the large number of pesticides in use, however, the cotton glove dosimeter model could be a quick, reliable, and useful tool for evaluating potential pesticide exposure.

Source: Environmental Health Perspectives Vol. 109 November 2001.

Chronic Low-Dose Pesticide Exposure and Cognitive Impairment

In a recent study, French researchers have carried out tests on agricultural workers in vineyards in the Bordeaux region, most of whom use fungicides, to measure their cognitive well-being and see how it related to the amount of pesticides they had been exposed to over the years.

Previous studies had shown that high-dose pesticide poisoning can cause acute human health effects such as motor skill damage, impaired intellectual functioning, and memory loss. In this study, the first to assess long-term neuropsychologic effects of chronic, low-level pesticide exposures in a large sample of workers, the French researchers found many examples of impaired cognitive functioning among exposed workers.

The team interviewed 917 men and women, aged 43-58, between February 1997 and August 1998. Of the study participants, 528 had been directly exposed to pesticides through mixing or spraying over a mean of 22 years, another 173 had been indirectly exposed by contact with treated plants, and 216 had never been exposed. The pesticides used were primarily fungicides.

Workers who were either directly or indirectly exposed performed worse on tests of memory, selective attention, verbal fluency, and abstraction compared with nonexposed workers. On a test of both selective attention and working memory, directly exposed workers were 3.5 times more likely to score low compared with nonexposed subjects. On a similar test of selective attention and mental flexibility, the exposed individuals were 3.1 times more likely to score low. The exposed men and women processed information less quickly than nonexposed colleagues, although performances of exposed workers were similar to those of the nonexposed if the tasks were slowed.

The results of the study point to possible effects on functions of workers exposed to pesticides involving selective attention, working memory, associative memory, verbal fluency, and abstraction. These effects were strongest in workers directly exposed to pesticides compared to the nonexposed and were not explained by current exposure. Further research is needed to provide toxicologic explanations.

Source: Environmental Health Perspectives, Vol. 109, August 2001.

SARIN EXPOSURE AND MEMORY LOSS

On 20 March 1995, members of the Aum Shinrikyo religious cult released sarin, a deadly military nerve gas, in the Tokyo subway, using umbrellas to puncture newspaper-wrapped bags of the gas as they left the trains. Twelve people were killed in the incident, and more than 5,500 required emergency medical treatment.

Following this attack, a group of Japanese researchers carried out an investigation into the long-term physical and psychiatric effects of acute poisoning by sarin.

The team examined rescue workers and police officers who had been dispatched to the scene and were exposed to sarin in the course of performing their duties – a group of subjects with similar occupational, socioeconomic, and educational backgrounds. The study included 56 exposed subjects from the Tokyo fire and police departments, who were subdivided into high- and low-exposure groups, and 52 nonexposed subjects of similar backgrounds from the same departments. The research was conducted three years after the exposure.

To assess neurobehavioral effects, the researchers administered five tests designed to measure psychomotor function and memory function. A significant causal relationship was discovered between exposure to sarin and memory disturbance in rescue team staff members and police officers 2 years and 10 months to 3 years and 9 months after exposure to sarin in the Tokyo subway attack.

However, further research is needed to determine whether the memory disturbance observed in this study is in fact caused by the direct neurotoxicity of sarin. At present the mechanism behind the disturbance remains unclear.

Source: Environmental Health Perspectives, Vol. 109, November 2001.

CURRENT RESEARCH INTO PARKINSON'S DISEASE

Parkinson's disease (PD) is one of many similar disorders grouped together under the name 'parkinsonism'.

Sufferers may have tremor in their limbs, jaw, or face, and rigidity in their limbs and torso. They may move slowly with jerky motions and suffer impaired balance and coordination. PD is a progressive disorder that most often begins after age 55. It is incurable. However, symptoms can sometimes be treated with drugs such as L-dopa or with treatments such as deep brain stimulation, in which an electrode is implanted in the brain in order to standardize the electric current fueling the brain's different processes.

PD symptoms are caused by the loss of cells in the substantia nigra, a part of the brain that controls movement. Nigral cells produce the neurotransmitter dopamine, and dopamine controls signaling to the motor cortex, which initiates smooth movement. Once dopamine has been depleted by about 80%, the jerky, uncontrolled movements characteristic of PD begin to appear. It is not yet known whether PD patients are born with less dopamine, whether dopamine loss accelerates with age, or whether loss is secondary to some other process.

Postmortem examinations also reveal that PD patients typically develop Lewy bodies, microscopic structures containing the protein α-synuclein, in the substantia nigra.

Although ageing has been claimed to be the only unequivocal risk factor for PD, several environmental factors also correlate with incidence of the disorder. These include exposure to hydrocarbon solvents, viral infections such as encephalitis, high dietary fat, high iron levels, depression, and head injury. Exposure to both single metals such as manganese and to mixtures of metals including lead, copper, and iron has also been implicated.

Also, pesticides have long been suspected of having a particularly strong link with PD, and recent studies have shown a 70% increased risk of the disorder among users of in-home insecticide and garden herbicides.

The widely used paraquat, maneb, and heptachlor have been especially targeted as pesticides implicated in PD, and paraquat and maneb in combination have been shown in mouse studies to cause greater locomotor disturbance than either pesticide by itself.

The United States National Institute for Environmental Health Sciences (NIEHS) currently supports 24 PD-related research projects through its extramural Environmental Health Sciences Centers Program and the institute has now announced its intention to establish a consortium to study gene-environment interactions in PD.

By learning more about how environmental exposures and genetic predisposition work together to cause PD, scientists can begin to develop effective approaches to disease prevention, diagnosis, and treatment.

Source: Environmental Health Perspectives Vol. 109 December 2001.

CRI and WHO Co-organise an International Conference on Environmental Threats to The Health of Children: Hazards and Vulnerability Bangkok, Thailand, 3-7 March 2002

A growing number of diseases in children are linked to the unsafe environments in which they live, learn, play and grow. Children may be exposed during periods of vulnerability, to high levels of pollutants in air, water, food and soil, and to chemicals present in household products and contaminated consumer goods. These problems are magnified in the developing countries, where air pollution, lack of access to safe water and sanitation, misuse of chemicals and other environmental risks are determinants of a large burden of disease in children. It has been estimated that about one third of the global burden of disease (GBD) can be attributed to environmental risk factors, and that 43% of the environmental disease burden falls on children under years of age, who constitute 12% of the world population.

The environment is a key factor in determining the healthy development of children, and WHO recognizes that efforts should be undertaken to enable countries to assess their environmental risks and establish appropriate prevention and monitoring mechanisms. The health and environment sectors should be able to exchange experience and knowledge, promote research, disseminate information and inform the community on how to protect children from environmental threats.

Such was the context for a conference that attracted delegates from 40 countries. The objectives of the meeting were threefold:

(a) To present and discuss recent knowledge, new research results and methodologies used to identify the effects of environmental threats to children's health, including the effects of specific hazards (e.g. pesticides, persistent toxic pollutants, lead, arsenic, radiation, noise and others).

(b) To increase the awareness of health and environment professionals about the effects of chemicals on children's health and development, and motivate action required.

(c) To provide guidance on how to prepare and develop actions required to promote the protec-

tion of children's environmental health at country level.

The conference program included plenary, focus and poster sessions as well as field trips to a health-promoting school, Kanaratbamrung Pathum, and to Ban Saladang Village which has a community-based approach to water and waste management. On the final day of the conference there was a round-table presentation on the topic Building Partnerships: Research, Capacity Building and Interventions by Dr. Ruth Etzel of the American Academy of Pediatrics, Dr. Rahmat Awang, Asia Pacific Association of Medical Toxicology, Dr. Vanich Vannapruks of Phramongkutklao College of Medicine, and Dr. Peter van den Hazel, of the International Network on Children's Health, Environment and Safety. An important outcome of the conference was the drafting of the Bangkok Statement, a pledge to promote the protection of children's environmental health throughout the world.

In her concluding report on the conference, Dr. Mathuros Ruchirawat, Vice-President of CRI, stated: The priorities for action that we have heard articulated in the "Bangkok Statement" provide the basis for hope in achieving a cleaner environment in which children will no longer be in jeopardy due to the health threats of industrial pollution and the many other sources of pollution and disease that spring from the imbalances in our global community.

THE BANGKOK STATEMENT

WE AFFIRM

That the principle *"children are not little adults"* requires full recognition and a preventive approach. Children are uniquely vulnerable to the effects of many chemical, biological and physical agents. All children should be protected from injury, poisoning and hazards in the different environments where they are born, live, learn, play, develop and grow to become the adults of tomorrow and citizens in their own right.

That all children should have the right to safe, clean and supportive environments that ensure their survival, growth, development, healthy life and well-being. The recognition of this right is especially important as the world moves towards the adoption of sustainable development practices.

That it is the responsibility of community workers, local and national authorities and policy-makers, national and international organizations, and all professionals dealing with health, environment and education issues to ensure that actions are initiated, developed and sustained in all countries to promote the recognition, assessment and mitigation of physical, chemical and biological hazards, and also of social hazards that threaten children's health and quality of life.

HYPOSPADIAS AND ENDOCRINE DISRUPTION

There is mounting evidence that hypospadias may be, at least in part, the result of exposure to synthetic and natural chemicals that can affect normal development of the male fetus. The fetus is especially sensitive to chemicals known as endocrine disruptors that can mimic or interfere with natural hormones that control development.

Hypospadias can be defined as an arrest in normal development of the urethral, foreskin, and ventral aspect of the penis. This results in a wide range of abnormalities, with the urethral opening being anywhere along the shaft of the penis, within the scrotum, or even in the perineum.

Reports of increasing prevalence of hypospadias have raised questions concerning etiology, treatment, and prevention. To date, there is no comprehensive understanding of the etiology of hypospadias that can inform primary prevention efforts and improve therapeutics. The etiology of many hypospadias is often assumed to be multifactorial, implicating some combination of genes and environment in the development of the anomaly.

A limited number of human epidemiologic studies have examined the risk of hypospadias in offspring of parents based on regional agricultural and industrial background exposure and lifestyle. A study in Minnesota found an increased risk of urogenital anomalies in the general population when crop regions were compared. The odds ratio was 1.56 in the corn/soybean region compared with 2.25 in the wheat/sugar beet/potato region of the state. The latter region was considered a high-use region based on poundage applied of fungicides and chlorphenoxy herbicides. Not only are offspring of farmers in this region at greater risk but children of nonagriculturally employed parents living in the same region are as well. Children conceived in the spring were at greatest risk for all birth defects *($p < 0.01$),* which coincided with the season of heaviest pesticide use.

A Canadian study comparing birth defects among four communities found a significant increase in urogenital defects between one community and three others that were not as industrialized. Hypospadias was the only end point that was significantly different among the communities. No association was made with a specific industry in the community. However, three industries in the high-risk community (producers of polyvinyl chloride, aluminum, and paper and pulp) are among those associated with the release of dioxin and dioxin-like compounds.

A Danish study looking at cryptorchidism and hypospadias in the offspring of farmers and women gardeners found no risk for hypospadias but an increase for cryptorchidism. A Norwegian study looking at 192,417 births between 1967 and 1991, where the parents were identified as farmers, revealed an odds ratio of 1 for hypospadias. However, between 1967 and 1971, the odds increased to 2.06 among tractor sprayers. Prevalence was greatest throughout the study for April-June conceptions and grain farming.

Over the past decade, rapid advances in integrated cellular, molecular, physiologic, biochemical, and toxicological research have revealed several stages of urogenital development that are vulnerable to endocrine-disrupting chemicals. To date, the activity of xenoestrogens and their feminizing effects on males do not explain hypospadias. However, since environmental antiandrogens were first reported in 1995, several stages of male urogenital development have clearly been revealed where specific synthetic chemicals can impede normal molecular and biochemical activity leading to frank expression of hypospadias. Despite these new discoveries, the lack of a putative causal agent for hypospadias in humans continues to pose a problem. This will become more of a problem if the list of antiandrogens continues to grow. It will also increase the difficulty of making personal and public health decisions about reducing exposure of reproductive age individuals.

Source: Environmental Health Perspectives, Vol. 109, November 2001.

Health dangers from contaminated coal

It has long been noted that a large number of the population of Guizhou Province in southwestern China suffer from arsenic poisoning, with symptoms ranging from freckled skin to squamous cell carcinoma, and many millions are afflicted by fluorosis, which can soften and disfigure teeth and bones. It is only recently, however, that the source of the problem has been discovered: contaminated coal.

In damp, cool autumn weather, it is impossible to dry corn, chili pepper, and other crops out of doors, so rural families bring such crops into their houses to dry them over coal-burning stoves. Geologists have found, however, that the coal contains abnormally high concentrations of arsenic and fluorine.

Wood was at one time the primary fuel for people living in this region, but the forests were largely denuded by the early 1900s and now most people who live there have no alternative to coal.

Compounding the problem of the contaminated fuel is the fact that most houses have no chimneys and, as a result, volatilized elements from the coal collect indoors. U.S. and Chinese scientists have begun mapping the geologic distribution of fluorine and arsenic within regional coal deposits to determine the least harmful sites to establish quarries, and researchers are working on developing a field test kit that residents of Guizhou Province can

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Titanium dioxide – a weapon against air pollution

Titanium dioxide, used as a pigment for whitening teeth, can also assist in the fight against air pollution by incorporating the chemical in construction materials. Titanium dioxide acts as an anti-pollutant because of its efficient photocatalyst properties, accelerating the breakdown of water vapor by ultraviolet light. The results of this reaction are hydroxyl radicals, which attack both inorganic and organic compounds, and turn them into harmless molecules that can be easily washed away.

Concrete slabs coated with titanium dioxide have been found to remove up to 90 percent of nitrogen oxide, a contributor to air pollution and acid rain, emitted in vehicle exhaust, particularly from diesel engines. The exhaust pollutant breaks down when sunlight hits the coated concrete, releasing reactive oxygen that turns nitric oxide to nitric acid ions. The alkaline concrete neutralizes the ions, which are then washed away by rain.

However a major problem with this promising application is that titanium dioxide does not absorb sunlight very well and indeed it has been calculated that only 3 percent of sunlight falls into the range needed for the titanium dioxide to work. Although a recent advance developed by laboratories in Japan has boosted the efficiency to 10 percent, this is still only a small amount of the sunlight.

Source: Scientific American, Vol. 286, No. 2, February 2002.

Differences in Individual Reactions to Lead Exposure

The same level of lead exposure can cause widely varying symptoms of lead poisoning and organ dysfunction in different people.

Recent research carried out at Harvard University has revealed that an underlying cause for individual differences may be genetic polymorphism in the δ-amino-levulinic acid dehydratase *(ALAD)* gene.

The team studied *ALAD* polymorphism and its relation to altered concentrations of bone and blood lead among 726 middle-aged and elderly men who had been exposed to lead nonoccupationally. Given their ages, these men would have faced now-obsolete lead exposures such as inhaling combusted leaded gasoline and ingesting food from lead-soldered cans, as well as still-present exposures such as eating vegetables grown in lead-contaminated soil. The men were participants in the Normative Aging Study, a longitudinal study of aging begun in 1963. Middle-aged to elderly men are at the highest risk for the onset of certain chronic diseases such as hypertension, stroke, heart attack, and dementia, and the researchers believe that cumulative lead exposure may be a significant risk factor for these problems.

Bone lead measurements were taken at the thigh and the knee. These sites were chosen because they consist, respectively, of pure cortical and pure trabecular bone. Cortical bone has very slow turnover; lead that is deposited there persists for many decades and thus provides a good reflection of total lead exposure. Conversely, trabecular bone has a relatively rapid turnover and releases a good deal of lead into the blood. Trabecular bone is therefore a good reflection of bone lead stores that can be mobilized into circulation.

The results showed that patella lead was the major predictor of blood lead in this aging nonoccupationally exposed population and that *ALAD* polymorphism significantly affected this association. For example, when patella lead exceeded 60 µg/g, blood lead concentrations in carriers of the *ALAD 2* allele were higher than those in *ALAD 1-1* carriers. By contrast, when patella lead concentrations were lower than 40 µg/g, blood lead concentrations were higher in *ALAD 1-1* carriers than in *ALAD 2* carriers. On the basis of their results, the researchers suggest that when blood lead concentrations are relatively low-less than about 8 µg/dL, for instance-*ALAD 1-1* carriers will have higher blood lead concentrations than *ALAD 2* carriers.

Source: Environmental Health Perspectives Vol. 109 August 2001.

Health dangers from contaminated coal

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use to measure the concentrations of dangerous elements in the coal they excavate.

A low-tech test has already been designed that involves easy to follow procedures of crushing the coal, boiling it and adding certain chemicals. The chemical reactions produce arsenic gas, which reacts with a paper test strip inside a sealed bottle. The paper turns a shade of yellow, orange or brown, depending on the concentration of arsenic.

If these kits are successful, it is hoped to develop cheap test kits to measure fluorine levels as well.

Source: Scientific American, Vol. 286, No. 2, February 2002.

REPORTS ON DEATHS DUE TO BIO-TERRORISM-RELATED INHALATION OF ANTHRAX

Reports have been published on the case of two postal workers in the United States who died of inhalational anthrax as a result of handling a letter containing anthrax spores mailed to a US senator's office in Washington D.C. on 9 October 2001 and opened in the office building on 15 October.

Both patients had nonspecific prodromal illnesses. One patient developed predominantly gastrointestinal symptoms, including nausea, vomiting, and abdominal pain. The other patient had a "flulike" illness associated with myalgias and malaise. Both patients ultimately developed dyspnea, retrosternal chest pressure, and respiratory failure requiring mechanical ventilation. Leukocytosis and hemoconcentration were noted in both cases prior to death. Both patients had evidence of mediastinitis and extensive pulmonary infiltrates late in their course of illness. The durations of illness were 7 days and 5 days from onset of symptoms to death; both patients died within 24 hours of hospitalization. Without a clinician's high index of suspicion, the diagnosis of inhalational anthrax is difficult during nonspecific prodromal illness.

Inhalational anthrax presents with nonspecific symptoms that cannot be distinguished from many more common diseases based on early clinical manifestations or routine laboratory tests. Both patients in this report sought medical care for apparently mild, nonspecific illnesses and were sent home. Only after the news media reported cases of inhalational anthrax involving 2 postal workers from the local mail facility did these patients' physicians consider the possibility that they could have inhalational anthrax. At that point, the patients had been ill for 7 days (patient 1) and 5 days (patient 2).

Despite aggressive medical therapy both patients developed rapidly progressive disease and died. They had received antimicrobial therapy at the discretion of their physicians before the Centers for Disease Control and Prevention released formal guidelines on 26 October 2001.

These guidelines recommend combination therapy for inhalational anthrax and complicated cutaneous anthrax. Ciprofloxacin or doxycycline are recommended in conjunction with another active antimicrobial drug, such as rifampin or clindamycin. Even though all isolates tested were susceptible to penicillin, β-lactamases were identified in these isolates, and penicillin monotherapy for treatment of systemic infection is not recommended. Susceptibility testing also revealed intermediate sensitivity to ceftriaxone and presence of a cephalosporinase. Cephalosporins, therefore, are not indicated for the treatment of *B anthracis* infection.

These 2 cases emphasize that in the event of serious outbreaks of infectious diseases, rapid communication of epidemiologic data to front-line medical care providers (especially emergency physicians and primary care clinicians) is essential so they may initiate appropriate diagnostic procedures and therapies. Efforts should be made to enhance communications systems between public health agencies and clinicians.

Source: JAMA, Vol. 286, November 2001.

Bioterrorism – A New Challenge for Medicine

On 4 October, 2001, it was announced that a 63-year-old man had been hospitalized in Palm Beach County, Florida, with inhalational anthrax. This was the first recognized case of inhalational anthrax in the United States since 1976, and the first in US history to result from an intentional human act. As such, it ushered in a new era for the United States, one in which the hypothetical threat of lethal bioterrorism has become a stark reality.

Since 4 October, the Centers for Disease Control and Prevention (CDC) has confirmed a total of 10 cases of inhalational anthrax and 7 cases of cutaneous anthrax. Five additional cases have been identified as being suspicious for cutaneous anthrax. All but 1 of these cases appear to have been directly linked to the US postal system. The epidemiologic link of the apparently isolated case of the 61-year-old Bronx resident and employee of a Manhattan hospital who died of inhalational anthrax remains a mystery. Clinical cases of cutaneous or inhalational anthrax have clustered in the Boca Raton, Fla, New York City/ New Jersey, and Washington, DC, areas. However, traces of anthrax spores, which likely are secondary contamination from identified primary sources of anthrax spores, have been found in distant locations such as Indianapolis, Ind, and Kansas City, Mo. More than 30000 people are estimated to have received antibiotics as a consequence of possible exposure to anthrax spores. The need for continual reevaluation of conventional wisdom regarding this disease as well as other potential bioterrorist threats has been made clear from these recent experiences. In this regard, the cross-contamination of mail and the special vulnerability of postal workers are two of the most unexpected epidemiologic findings thus far.

Source: JAMA, Vol. 286, November 2001.

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Correspondence should be addressed to:

ICEIT NEWSLETTER
Chulabhorn Research Institute
Office of Academic Affairs
Vibhavadee-Rangsit Highway
Bangkok 10210, Thailand
Tel: (66-2) 574-0622 to 33 ext. 1610
Fax: (66-2) 574-0616 or (66-2) 247-1222
CRI Homepage: <http://www.cri.or.th>