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"UNEP Centre of Excellence for Environmental and Industrial Toxicology".

A Special Celebration to Honor Professor Dr. Her Royal Highness Princess Chulabhorn on Receiving Two Prestigious Scientific Awards

On 18 January 2010, a special celebration was organized by Chulabhorn Research Institute at the Chulabhorn Convention Center to honor Her Royal Highness for the recognition of her scientific work at the very highest international level. Distinguished guests were invited to a banquet at which H.E. General Prem Tinsulanonda, President of the Privy Council and Statesman, delivered a speech of congratulations in honor of Her Royal Highness Princess Chulabhorn's achievements.

In 2009, Her Royal Highness Princess Chulabhorn was presented with two highly esteemed international academic awards for her outstanding contribution to science. These were the Ramazzini Award from the Collegium Ramazzini, Italy for her continuing

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A Special Celebration to Honor Professor Dr. Her Royal Highness Princess Chulabhorn on Receiving Two Prestigious Scientific Awards

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The Ramazzini Award
from the
Collegium Ramazzini

The Windaus Award
from the Georg-August Göttingen
University and the German
Chemical Society

contributions in occupational and environmental medicine, and the Windaus Award from the University of Georg-August, Göttingen, and the German Chemical Society for her important role in science and organic chemistry.

Representatives from both the Collegium Ramazzini and the University of Georg-August Göttingen were present at the banquet at which a special program featuring a performance of the classical Thai Blessing Dance as well as video presentations to honor Her Royal Highness was organized for the distinguished guests.

A New Approach to Identifying Ages When Children Are Most Susceptible to Lead Effects

Although the weight of evidence from a number of studies conducted during the last 15 years indicates that adverse effects of lead exposure on cognitive development occur at childhood blood lead levels (BPb) < 10 µg/dL, the age of greatest susceptibility has not been well defined. Several recent analyses have suggested that lead exposures at school age may be more strongly related to decrements in IQ scores or neuroanatomical deficits. These studies encountered problems in separating the effects at various ages due to the close tracking of BPb concentrations during childhood. Current guidelines of the US Center for Disease Control and Prevention for screening children focus on 1- and 2-year olds, making a better determination of the age of greatest susceptibility very important to clinical practice. The analyses presented in a new study suggest that screening school-age children who are undergoing evaluation for cognitive deficits or behavioral problems may help to identify underlying reasons for their learning or behavioral problems.

Researchers applied a new method that avoids the problem of serial correlation in BPb concentrations during childhood. The method instead focused on the pattern of BPb concentrations over time by comparing the usual pattern of BPb peaking at around 2 years of age with that of children whose highest childhood exposure occurred at school age, while holding average childhood exposure constant. Researchers found that the greatest susceptibility to both cognitive and behavioral development was at 5-6 years of age. These results suggest that several cross-sectional analyses showing stronger associations between BPb in older children and reductions in IQ scores are not simply residual effects from higher exposures at 2 years of age.

Although the relatively large size of the combined cohorts, availability of serial BPb measurements, and numerous covariates are strengths of these analyses, there are also a few limitations. Relatively few children in these analyses had higher BPb levels at 6 years than at 2 years of age. The model estimates, therefore, depend somewhat on the upper end of the distribution of 6-year : 2-year ratios. Although relatively few children were in this upper range of the ratio, regression diagnostics did not indicate that these results were disproportionately dependent on these data.

Another potential limitation is that the method focuses on temporal patterns in BPb levels relative to the 2-year BPb concentration. Researchers chose the 2-year measurement as the reference because it is typically the highest level during childhood and because it has been the focus of clinical screening efforts. Serial BPb level patterns relative to BPb measurements at ages other than 2 years may produce different results. In support of the

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A New Approach to Identifying Ages When Children Are Most Susceptible to Lead Effects

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method, however, the researchers investigated ratios relative to ages 1 and 3 years and found similar results, although ratios using 2-year levels showed the strongest relationship to IQ and criminal arrest rates.

The results of this new statistical approach strengthen the emerging evidence that lead exposure in school-age children may be more strongly related to cognitive and behavioral development than exposures during earlier childhood. This finding has important implications regarding testing of children for high lead exposure. Although it is still very important to reduce lead contamination in early childhood, these analyses indicate that efforts to reduce lead exposure should continue as children progress to school age. Perhaps more directly relevant, this analysis suggests that BPb testing might be an important part of a diagnostic workup for school-age children who have cognitive delay or behavioral problems, such as attention-deficit disorder and related behavioral and learning problems. The researchers believe that this statistical approach may be useful for disentangling the age of greatest vulnerability to other serially measured environmental toxicants such as pre- and postnatal tobacco smoke.

Source: Environmental Health Perspectives, Vol. 117, No. 8, August 2009.

HUMAN HEALTH IMPLICATIONS OF DDT USED IN MALARIA CONTROL

DDT has been used annually in the Limpopo Province in northern South Africa, and in particular the Vhembe District Municipality, since 1945 to control malaria transmission by the *Anopheles funestus* and *Anopheles arabiensis* vectors. DDT is applied to the indoor walls and roofs of traditional unpainted mud-walled, thatched huts using indoor residual spraying (IRS). Once in the environment, DDT can persist as long as 15 years or is broken down to the main metabolites dichlorodiphenyl-chloroethane (DDE) and dichlorodi-phenyldichloroethane (DDD). DDE is the main and most persistent metabolite in the environment.

The various DDT metabolites and isomers are also known endocrine disruptor chemicals (EDCs) as they mimic or antagonize the action of hormones. These EDCs can therefore interact with physiological systems and cause alterations in development, growth, and reproduction in wildlife and particularly in exposed fish. Human exposure to environmental compounds with estrogenic activity and the potential effects on human health is the subject of an ongoing scientific debate. There is evidence that environmental estrogens can influence the human endocrine system, and in consequence both reproduction and tumour development. *p,p'*-DDE, the persistent metabolite of *p,p'*-DDT, acts both as an androgen receptor antagonist and inhibitor of testosterone. An earlier study indicated the estrogenic effect of *o,p'*-DDT on rats, when treatment with *o,p'*-DDT resulted in the same effects as estrogen on juvenile rat uteruses after 24 h. Adverse reproductive system effects associated with *in utero* DDT or DDE exposure in male animals include, amongst others, abnormal development of ovarian tissue, reduced penis size, hypospadias and cryptorchidism.

DDT was banned for agricultural purposes in South Africa in 1976. Therefore, most environmental DDT residues are certainly derived from malaria control activities. Malaria areas are characterised by numerous small and large settlements and low economic development status. Since annually-applied DDT in IRS is likely to reach the outdoor environment through dust and air, as well as from possible spillages during application, the levels in exposed biota, water, sediment, and soil in malaria areas need to be understood to determine exposure pathways and sinks, possible risks to humans and the environment, and to identify possible remedial interventions. For this reason a research study was recently undertaken to establish, interpret and report on DDT levels in domestic and wild birds, water, sediment, and fish, and to identify any human health implications these may pose in DDT sprayed areas.

The study concluded that the presence of *p,p'*-DDT residues and particularly *p,p'*-DDE in chicken that share the immediate environment with humans is a reason for concern. Their contamination is likely related to the use of DDT for malaria control and chickens should further be evaluated as a possible animal biomarker for human IRS exposures. Rural chickens probably have an average life span of 2 years and both the eggs and the meat might be dietary sources of DDT intake by humans.

The study also found that in an aquatic system flowing into a DDT-sprayed area, changes in DDT levels and profiles in fish occur that had not previously been identified, and that might impact on human health and other biota. Although the levels of DDT in water are low, bio-concentration, as well as bio-magnification seems to be in effect, but further trophic-level research, and especially human consumption patterns of fish, is needed to determine possible risk. Moreover, since wild terrestrial birds also showed detectable levels, further investigation as to routes of uptake is needed. Finally, chickens had very high levels, and may be an important route of uptake by inhabitants of sprayed dwellings. Both wild and domestic bird parameters indicate the importance of having to consider and investigate how IRS-applied DDT can find its way to the outdoor environment and river systems.

Source: Chemosphere, Vol. 77, Issue 3, November 2009.

Health Effects of Human Exposure to Halogenated Hydrocarbons: A Study of Their Concentration Distributions in Blood and Breast Milk of Primiparous Mothers

Polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are halogenated hydrocarbons and highly persistent environmental pollutants.

When PCDDs, PCDFs, and PCBs are released into the environment, these toxicants accumulate in the human body through ingested food. In fact, over 90% of human exposure to PCDDs, PCDFs, and PCBs has been attributed to food intake, particularly from food of animal origins. PCDDs, PCDFs, and PCBs accumulated in the maternal body have been reported to be transferred from the mother to her fetus via the placenta during pregnancy and from mothers to infants via breast milk. Moreover, because fetuses and infants are considered significantly more sensitive to a variety of environmental toxicants compared with adults, the possibility of adverse effects by these toxicants on these fetuses and infants is anticipated.

Studies in humans and other vertebrates have demonstrated that pre- and/or postnatal exposure to PCDDs, PCDFs, and dioxin-like PCBs may contribute to numerous adverse health effects, including growth retardation of the fetus and infants, thyroid deficiency, immune deficiency, reproductive, carcinogenic effects, and endocrine disruption effects, and that exposure to non-dioxin-like PCBs may also elicit many adverse health effects, such as behavioral abnormalities, thyroxin, and reproductive effects. Moreover, the effect of PCBs on neuropsychological function was shown in several birth cohort studies. Therefore, to elucidate the influence of PCDDs, PCDFs, dioxin-like PCBs, and non-dioxin-like PCBs on the health of fetuses and infants, researchers have conducted exposure surveys of these compounds in maternal blood and breast milk in various countries, and they have reported an association between the concentration levels of PCDDs, PCDFs, dioxin-like PCBs, and non-dioxin-like PCBs in maternal blood and those in breast milk. However, specific data comparing the concentrations of PCDDs, PCDFs, dioxin-like PCBs, and non-dioxin-like PCBs in paired samples of blood and breast milk collected from the same mothers are limited, and the concentration pattern and the relationship of these compounds between blood and breast milk remain unexplained.

Therefore, to elucidate this relationship, researchers have measured the concentrations of PCDDs, PCDFs, dioxin-like PCBs, and non-dioxin-like PCBs in paired samples of blood and breast milk collected from 89 primiparous mothers in Sapporo City, Japan plus 30 primiparous mothers in whom these data had been previously reported.

The present study is one of the few studies in which the concentration distributions of PCDDs, PCDFs, dioxin-like PCBs, and nondioxin-like PCBs has been investigated in blood and breast milk collected from the same mothers. The arithmetic mean toxic equivalent (TEQ) concentrations of PCDDs, PCDFs, non-*ortho* PCBs, and mono-*ortho* PCBs in blood and breast milk of these 119 subjects were 8.2, 2.9, 5.1, and 0.4 pg TEQ g^{-1} lipid, respectively, and 4.8, 2.0, 4.0, and 0.4 pg TEQ g^{-1} lipid, respectively, with the total TEQ concentrations of these dioxin-like compounds being 7.0-36 (mean: 17, median: 14) and 5.7-41 (mean: 11, median: 10) pg TEQ g^{-1} lipid, respectively. The sums of the concentrations of 56 non-dioxin-like PCB congeners that were measured in the subjects' blood and breast milk were 43-445 (mean: 120, median: 106) and 34-366 (mean: 90, median: 81) ng g^{-1} lipid, respectively, indicating that the total TEQ concentration and the total concentration of 56 non-dioxin-like PCB congeners in the maternal blood were notably higher than those in the breast milk. Statistically significant correlations were observed between maternal age and the total TEQ concentration of PCDDs, PCDFs, and dioxin-like PCBs or the total concentration of 56 non-dioxin-like PCB congeners in maternal blood, and significant correlations were also observed between maternal age and the total TEQ concentration of these dioxin-like compounds or the total concentration of 56 PCB congeners in breast milk. The total TEQ concentration of PCDDs, PCDFs, and dioxin-like PCBs in maternal blood showed a close correlation to that in subjects' breast milk, and there was also good correlation between the total concentration of 56 non-dioxin-like PCB congeners in maternal blood and that in subjects' breast milk. Pearson and Spearman correlation analyses showed a relationship between the total TEQ concentration of PCDDs, PCDFs, and dioxin-like PCBs and the total concentration of 56 non-dioxin-like PCB congeners in maternal blood, and also showed an association between the total TEQ concentration of these dioxin-like compounds and the total concentration of 56 PCB congeners in breast milk. The concentration of hexaCB-153 in maternal blood showed significant correlations to the total TEQ concentration of PCDDs, PCDFs, and dioxin-like PCBs or the total concentration of 56 non-dioxin-like PCBs in that sample. Moreover, the concentration of hexaCB-153 in breast milk also showed significant correlations to the total TEQ concentration of these dioxin-like compounds or the total concentration of 56 non-dioxin-like PCB congeners in that sample. These findings suggested that hexaCB-153 may be an indicator of total TEQ concentrations of PCDDs, PCDFs, and dioxin-like PCBs and total concentrations of 56 non-dioxin-like PCB congeners in blood and breast milk of primiparous mothers.

Source: Chemosphere, Vol. 78, Issue 2, January 2010.

POLYCARBONATE PLASTICS AND HUMAN BISPHENOL A EXPOSURE

The endocrine-disrupting chemical bisphenol A (BPA) has recently received heightened attention because of widespread human exposure and disruption of normal reproductive development in laboratory animals. BPA is thought to disrupt normal cell function by acting as an estrogen agonist as well as an androgen antagonist. In animal studies, prenatal and neonatal exposure to BPA has been linked to early onset of sexual maturation, altered development and tissue organization of the mammary gland, induction of preneoplastic mammary gland and reproductive tract lesions, increased prostate size, and decreased sperm production in offspring. Most recently, exposure to BPA has also been associated with chronic disease in humans, including cardiovascular disease, diabetes, and serum markers of liver disease.

Orally administered BPA is rapidly metabolized by glucuronidation during first-pass metabolism, with a biological half-life of approximately 6 hr and nearly complete elimination within 24 hr. However, because of continuous and widespread exposure, > 92% of the 2,517 participants ≥ 6 years of age in the U.S. 2003-2004 National Health and Nutrition Examination Survey (NHANES) had detectable concentrations of BPA in their urine. The geometric mean (GM) urinary BPA concentration in that study was 2.6 µg/L (2.6 µg/g creatinine), and the 95th percentile was 15.9 µg/L (11.2 µg/g creatinine).

An important source of human exposure is thought to be the ingestion of food and drink that has been in contact with epoxy resins or polycarbonate plastics. Polycarbonate is a durable, lightweight, and heat-resistant plastic, making it popular for use in plastic food and beverage containers. Indeed, nearly three-fourths of the 1.9 billion pounds of BPA used in the United States in 2003 was used for the manufacture of polycarbonate resin. Other common uses of BPA include the manufacture of epoxy resins used as composites and sealants in dentistry and in the lacquer lining of aluminum food and beverage cans.

Laboratory studies have demonstrated that biologically active BPA is released from polycarbonate bottles after simulated normal use. High temperatures as well as acidic and alkali solutions cause polymer degradation via hydrolysis, resulting in increased BPA migration. After incubation for 8, 72, and 240 hr in food-simulating solvents (10% ethanol at 70°C and corn oil at 100°C), mean BPA migration increased with incubation time. After a sequence of washing and rinsing, it was found that new polycarbonate bottles leached 1.0 ± 0.3 µg/mL BPA (mean ± SD) into the bottle content after incubation at room temperature for 7 days. Although exposure to boiling water increased the rate of BPA migration up to 55-fold, used bottles did not leach significantly more BPA than new ones. However, other studies have found that higher concentrations of BPA leach from used polycarbonate plastic than from new. BPA has been observed to leach from polycarbonate animal cages after 1 week of incubation at room temperature, with higher levels of migration from used versus new cages. Similarly, after incubation in 100°C water for 1 hr, the amount of BPA leached from baby bottles subjected to simulated use (including dishwashing, boiling, and brushing into the bottle) exceeded the amount that leached from new baby bottles.

Recently, some polycarbonate bottle manufacturers voluntarily eliminated BPA from their products, and several retailers withdrew polycarbonate bottles from their stores altogether. Canada has imposed a ban on the use of BPA in polycarbonate baby bottles in order to reduce exposure of infants to BPA, and similar legislation is being considered by several U.S. states. However, such actions have been largely preemptive, as no epidemiologic study has evaluated the physiologic consequences of polycarbonate bottle use. In a recent study, the impact of cold beverage consumption from polycarbonate bottles on measurable urinary BPA concentrations was monitored in a Harvard College population. Researchers also measured exposure to the phenols triclosan (TCS), methyl paraben (MePB), propyl paraben (PrPB), and benzophenone-3 (BP-3), which occurs mainly through the use of personal care products. Therefore, because exposure of these chemicals is considered unrelated to polycarbonate bottle use, the study assessed their association with polycarbonate bottle use as a negative control.

The study population included 77 subjects who ranged in age from 18 to 23 years, with a median of 19 years. On the basis of self-reported data, the study categorized race/ethnicity into four groups: Caucasian, Asian, African American, and Hispanic. Thirty participants (39.0%) were Caucasian, 38 were of Asian descent (49.4%), 5 were African American (6.5%), and 4 were Hispanic (5.2%). Forty-one subjects were male (53.3%). Protocol compliance for the week in which participants drank from polycarbonate bottles ranged from 50% to 100% but was generally high, with a median of 90%.

Nine samples (11.7%) from the washout week and three samples (3.9%) from the intervention week (period in which participants drank from polycarbonate bottles) had BPA concentrations < limits of detection. BP-3 and MePB were detected in all participants, and PrPB was detected in all but one participant each week. TCS was detected in 75.3% of the samples taken at the end of the washout week and in 74.0% of the samples collected after the intervention week. The GM concentration of BPA was 1.3 µg/L (1.2 µg/g creatinine) during the washout period and 2.1 µg/L (2.0 µg/g creatinine) during the intervention week. GM concentrations for the washout phase and intervention week were 46.1 and 66.8 µg/g creatinine for BP-3; 51.3 and 48.4 µg/g creatinine for MePB; 8.4 and 8.8 µg/g creatinine for PrPB; and 15.5 and 17.3 µg/g creatinine for TCS, respectively.

Urinary BPA concentrations in weeks 1 and 2 were compared. Urinary BPA concentrations increased by 69% after polycarbonate bottle use. A larger difference was observed between the intervention and washout weeks in the stratum with intervention compliance ≥ 90% (77% increase) relative to the stratum with compliance < 90% (55%); however, the strata were not significantly different from

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Well-Water Consumption and Parkinson's Disease: A Study Conducted in Rural California

Multiple lines of evidence link pesticides as possible contributors to the pathogenesis of Parkinson's disease (PD). Many epidemiologic studies previously reported associations between pesticide exposure, rural living, farming and the development of PD. A number of animal studies have also supported a potential etiologic role of pesticides in PD. The ingestion of contaminated drinking water is a potentially important vehicle for pesticide exposure in human populations. Epidemiologic studies previously examined links between well-water consumption and PD, and most provided support for positive associations. All existing studies have relied on self-reports of well-water consumption and used broad ever/never exposure categories. Such studies may have suffered from recall bias and exposure misclassification. Most importantly, no study to date has attempted to specify pesticide exposure levels by assessing or estimating the contamination of well-water with specific pesticides.

Although the Safe Drinking Water Act of 1974 was passed to regulate the public drinking water supply, private wells in the United States are not subject to the same regulations and thus are not similarly monitored or held to the same water-quality standards as are public systems. Furthermore, many private wells are dug or driven at shallow depths (i.e., < 15-20 yards), which place them at risk of being contaminated by land activities such as pesticide applications in the vicinity of a well. Pesticides may move from their initial intended area of application. Investigators have shown that measurable concentrations of pesticides have been detected in air, water, plants, and animals up to several hundred meters from the application sites, emphasizing the need for methods to assess environmental exposures due to drift and contamination of soil, air, and water in agricultural communities. Geographic information system (GIS)-based methods of assessing exposures to pesticides may prove an effective solution when comprehensive pesticide-application data exist. Researchers conducting the present study developed and employed a GIS-based exposure assessment tool to estimate pesticide exposures from applications to agricultural crops using data from California pesticide use reports (PURs), land-use maps, and geocoded residential historical addresses. This information was combined with data on well-water consumption collected in interviews with study participants to estimate exposure to potentially pesticide-contaminated well-water. This study aimed to investigate whether consumption of water from private wells located in areas with documented historical agricultural pesticide use was associated with an increased risk of PD among residents of the Central Valley of California, well known for its intensive agricultural activities.

Study participants were predominantly Caucasian, over 65 years of age, and without a family history of PD. Cases were slightly older than controls, were more likely to be male, and had completed fewer years of education. They were also more likely to have never smoked cigarettes.

In this population, 16.9% of all subjects reported private well-water as their drinking water source some time during the 1974-1999 period. Cases were more likely to have consumed water from private wells than were controls during this period and reported drinking well-water on average 4.3 (of the 26) years longer than controls.

Consuming well-water presumably contaminated at any level by one of the six pesticides that were examined separately was associated with PD, but only for diazinon did the 95% CI exclude the null value of 1. However, high levels of possible contamination resulted in 31-90% increases in risk compared with no well-water contamination, with stronger associations seen for methomyl (OR = 1.67; 95% CI, 1.00-2.78), chlorpyrifos (OR = 1.87; 95% CI, 1.05-3.31), and propargite (OR = 1.92; 95% CI, 1.15-3.20). Only for diazinon in well-water was the dose response reversed; that is, lower rather than higher levels of possible contamination with diazinon resulted in greater increases in risk of PD. Adjusting for ambient pesticide exposures only slightly attenuated all well-water pesticide effect estimates, with the largest change seen for propargite.

For all six pesticides examined individually, PD risk associated with possible contamination of well-water and ambient exposures (19-75% relative increase) was greater than the risk associated with ambient exposures alone (15-57% relative risk increase), also indicating that for most of these agents ambient exposure only still increased the risk.

The combined estimates suggested that a higher number of water-soluble pesticides presumably contaminating well-water increased the risk of PD.

This study claims to be unique among those that have examined PD risk from well-water consumption in that researchers used existing historical California PUR data, which were combined with land-use maps to derive pesticide application rates for the study area over an extended period. Thus, the well-water pesticide measure is an estimate derived from GIS models.

An important strength of the present study is that all of the PD diagnoses were clinically confirmed by a study movement disorder specialist, and thus the results are expected to be only minimally affected by disease misclassification.

The study thus contributes to existing evidence that consumption of well-water potentially contaminated with pesticides may play a role in the etiology of PD.

Source: Environmental Health Perspectives, Vol. 117, No. 12, December 2009.

Contamination of Mineral Water with Xenoestrogens

Food consumption is an important route of human exposure to endocrine-disrupting chemicals (EDCs). To date, this has been demonstrated by exposure modeling or analytical identification of single substances in foodstuff (e.g., phthalates) and human body fluids (e.g., urine and blood). Since the research in this field is focused on few chemicals, the overall contamination of edibles with xenohormones is largely unknown.

Moreover, the causality between the exposure to EDCs and adverse human health effects is still a matter of some controversy due to the multifactoral etiology of hormone related diseases.

However, evidence for causality between exposure to xenohormones and development as well as reproductive disorders is growing.

For instance, *in utero* exposure to phthalate plasticizers has been shown to be associated with a decreased anogenital distance in male infants indicating undervirilization induced by environmental levels of these endocrine disruptors. Vice versa, phthalate exposure to girls is claimed to be correlated with an earlier onset of puberty, an effect that has been experimentally verified in mice in the case of the plastic component bisphenol A as well. Recently, the debate about endocrine disruption has been heated up by findings that some EDCs may exhibit epigenetic transgenerational effects.

Though many endocrine disruptors are ubiquitous in the environment and humans are known to be contaminated with a wide range of compounds, exact routes of human exposure remain largely unknown. Apparently there are various sources and pathways of xenohormone uptake: inhalation (i.e., from indoor air), dermal absorption (i.e., from personal care products), and ingestion of food. The contamination of foodstuff by production-related compounds has been documented analytically. Nonylphenols, as degradation products of commercial and industrial surfactants, for example, are identified ubiquitously in a broad variety of nourishments.

Another source of xenobiotics in foodstuff is rarely taken into account when dealing with endocrine disruption: substances migrating from packaging material into edibles. In order to optimize the properties of packaging materials (i.e., durability, elasticity, color), a variety of additives, such as stabilizers, antioxidants, coupling agents, and pigments, is used in the formulation. Especially additives from plastics (so-called plasticizers) are known to leach out of the packaging and consequently accumulate in the foodstuff. Given the fact that some of these compounds are known EDCs, researchers have hypothesized that the migration of substances from packaging material into foodstuff may contribute to human exposure with xenohormones.

In a recent study, bottled mineral water serves as a model foodstuff because it is a simple matrix and it does not contain endogenous hormones, like for example dairy products. Moreover, consumption of mineral water is increasing worldwide. On the German market, mineral water is available in two major sorts of packaging material: glass and PET (PETE, polyethylene terephthalate, resin identification code 1) bottles. Moreover, some brands of mineral water are sold in a packaging called Tetra Pak (Tetra Brick) although only to a minor extent. These paperboard boxes are coated with an inner plastic film and are more commonly used for packaging milk and fruit juices.

In the study, researchers analyzed commercially available mineral water in an *in vitro* system with the human estrogen receptor alpha and detected estrogenic contamination in 60% of all samples with a maximum activity equivalent to 75.2 ng/l of the natural sex hormone 17β-estradiol. Furthermore, breeding of the molluskan model *Potamopyrgus antipodarum* in water bottles made of glass and plastic resulted in an increased reproductive output of snails cultured in PET bottles. This provides the first evidence that substances leaching from plastic food packaging materials act as functional estrogens *in vivo*.

The results of the research effectively demonstrate a widespread contamination of mineral water with xenoestrogens that partly originates from compounds leaching from the plastic packaging material. These substances possess potent estrogenic activity *in vivo* in a molluskan sentinel. Overall, the results indicate that a broader range of foodstuff may be contaminated with endocrine disruptors when packaged in plastics.

Source: Environmental Science & Pollution Research, Vol. 16, No. 3, May 2009.

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each other. Of the other phenols, only urinary BP-3 concentration was associated with polycarbonate bottle use, with relatively higher concentrations observed after use of polycarbonate bottles (45% increase). A slightly larger change in BP-3 concentration was observed in the less compliant stratum (64% increase) relative to the more compliant stratum (36% increase); however, this difference was not statistically significant.

The researchers claim that this is the first study to assess the impact of polycarbonate drinking bottle use on urinary BPA concentrations. Despite within-person variability resulting from other sources of BPA exposure, a measurable increase in urinary BPA resulted from only 1 week of exposure to beverages contained in polycarbonate bottles. Replication of this study in other populations may help to inform public health policy regarding the use of BPA in polycarbonate food and beverage containers.

Source: Environmental Health Perspectives, Vol. 117, No. 9, September 2009.

HEALTH RISKS OF OCCUPATIONAL EXPOSURE TO ORGANIC SOLVENTS

Work-related hearing loss (HL) due to noise exposure remains a prevalent occupational condition. In addition to noise, other agents such as metals, asphyxiants, pesticides, and organic solvents may be hazardous to human hearing. In the occupational health and safety arena, solvents have been associated with dermal effects, neurobehavioral changes, and respiratory effects, among other pathologies. Millions of persons are currently exposed to solvents in their workplaces. One of the first reports on the adverse effects of solvents on human hearing studied a group of workers exposed to trichloroethylene. Animal studies have found cochlear damage due to aromatic solvent exposure. One of the most studied solvents in animals is toluene. Research has shown that toluene can reach cochlea and induce damage in the outer hair cells. Recent studies have also demonstrated that toluene and other solvents may adversely affect the acoustic reflexes due to an anti-cholinergic effect on the auditory efferent motoneurons. Field studies conducted in populations of solvent exposed workers have found an increment in the prevalence of HL among solvent-exposed workers in comparison with nonexposed control subjects. Also, human studies have suggested that due to the neurotoxic effects of solvents, these chemicals may induce dysfunction on the central auditory system.

Most of these clinical studies used a small number of workers exposed to solvents. In 2002, several laboratories started collaborating in a European project called Noisechem. To study the combined effects of noise and solvents on the auditory system, Noisechem proposed the use of an audiological test battery. The approach of a test battery was proposed because of the combined ototoxic and neurotoxic effects of solvents on the auditory system. In summary, solvent-induced hearing loss (SIHL) is a complex pathological entity that may be originated due to a combination of ototoxicity and neurotoxicity. This is different to the deleterious effects of noise on hearing. Noise exposure mainly affects the outer and inner hair cells in the cochlea, whereas solvents can affect both the hair cells and central auditory structures. In experimental animals, the midfrequency region of the cochlea is particularly affected by solvents. Considering that the auditory dysfunction associated with solvents is different to that one associated with noise exposure, the test battery used to assess each condition must therefore not be the same.

Taking into consideration the neurotoxic effects of solvents, the auditory effects induced by these chemicals may be more complex than the sole presence of abnormal hearing thresholds. Indeed, a central auditory dysfunction may likely be related to solvent exposure. Therefore, this aspect should be incorporated in the monitoring of exposed workers. However, there is a lack of scientific evidence on which test should be appropriate to assess the central auditory function in solvent-exposed workers. Only a limited number of studies have examined hearing outcomes using other tests to complement data from pure-tone audiometry. Thus, a research gap still remains on the most suitable tests, which should comprise an audiological test battery for solvent-exposed workers.

From the occupational and safety perspective, an appropriate test is the one not only capable of detecting the dysfunction but also suitable to be carried out in the occupational setting as a screening procedure. This means that it is easy to administer, does not take a long time to be completed, and has high validity and reliability. Taking into account this issue, the present research project aimed at incorporating within the hearing test battery for solvent-exposed workers, a test for central auditory function, which can fulfil these criteria. The selected procedure was the dichotic digits test. Research has demonstrated that this test is sensitive in detecting central auditory dysfunction.

In the present study one hundred and ten workers from a coating factory in New Haven, CT, that manufactures reinforced fabrics were selected. The industrial process in this factory involves application of polyurethane coating on a variety of fabrics to make the fabrics waterproof, weather resistant, and durable depending on the intended use of the fabric.

Jobs in the factory were divided into three different levels of solvent exposure. Hearing status of the workers was assessed with a test battery involving pure-tone hearing thresholds (0.5 – 8 kHz), high-frequency hearing thresholds (12 and 16 kHz), and dichotic listening measured through dichotic digits test. Multiple linear regression models were created to explore possible association between solvent exposure and each of the hearing outcomes.

The study found significant associations between solvent exposure and all three hearing outcomes. Covariates such as age, gender, race, and ethnicity were also significantly associated with the studied hearing outcomes.

The study concluded that occupational exposure to solvents may induce both peripheral and central auditory dysfunction. The dichotic digits test seems to be a reliable and easily applied tool to detect central auditory dysfunction associated with solvent exposure, and hearing loss prevention programs may use this tool to monitor workers' hearing in the occupational setting.

Source: Journal of Occupational and Environmental Medicine, Vol. 51, No. 10, October 2009.

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