New Face of a Well-Known Hazard
Arsenic Alters H1N1 Response in Mice

One of the puzzles of the 2009 pandemic of novel H1N1 influenza virus is why some populations are being hit harder than others—a reminder that known susceptibility factors cannot always explain why otherwise healthy people succumb to diseases that others survive. A team of researchers from Dartmouth Medical School may have uncovered a potential previously unrecognized susceptibility factor, demonstrating that exposure to arsenic significantly weakened mice’s immune response to a mouse-adapted subtype of H1N1 flu [EHP 117:1441–1447; Kozul et al.].

The team believes their study is the first to link flu morbidity to arsenic, which occurs naturally in the drinking water of hundreds of millions of people worldwide. In the United States public drinking water must meet the U.S. Environmental Protection Agency (EPA) arsenic limit of 10 ppb, but private well water is unregulated. Up to 25 million Americans with private wells may be exposed to arsenic levels above the EPA limit. In many regions of the United States and in Mexico, where the novel H1N1 outbreak began, arsenic levels in well water commonly exceed the EPA limit by tenfold or more.

The current study was inspired by recent epidemiologic research indicating that chronic exposure to arsenic increased the risk for a variety of pulmonary diseases including impaired lung function, cancer, and bronchiectasis. Other studies, including recent work by members of this research team [EHP 117:1108–1115 (2009)], have indicated that arsenic exposure can suppress the innate immune system. Impairment of the immune cells in the lungs as a result of arsenic exposure could also alter the ability to fight other infectious challenges.

The researchers tested their hypothesis that arsenic could suppress the innate immune response and thereby intensify H1N1 flu infection by giving mice drinking water containing 100 ppb arsenic for 5 weeks. After 5 weeks, the researchers inoculated the arsenic-exposed mice and a group of control mice with the H1N1 virus, and flu morbidity was measured as weight loss.

Control mice experienced moderate weight loss but returned to their original weight by day 16 postinfection. The arsenic-exposed mice had a more dramatic weight loss of up to 20% of their body weight by day 8 postinfection, at which point the researchers euthanized them to prevent suffering, in compliance with institutional animal care standards. In subsequent analyses at day 7 postinfection, examination of the exposed mice’s lungs revealed hemorrhaging, edema, and 10 times more virus than was seen in the lungs of control mice.

Millions of people worldwide are infected with seasonal flu each year, and hundreds of thousands die. Understanding the risk factors that may increase flu cases and deaths could have a potentially significant impact on preventing and treating this common disease.

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Polycarbonate Plastics and Human BPA Exposure
Urinary Levels Rise with Use of Drinking Bottles

Public and scientific concerns about exposure to bisphenol A (BPA) have risen in the last few years, with Canada and some U.S. states and cities banning BPA from polycarbonate baby bottles and other products sold for use by infants and children. Despite these concerns, little is known about whether the use of polycarbonate food or beverage containers actually contributes to BPA body burden in people. A new study of human exposure to BPA from drinking containers now shows that study participants’ urinary concentrations of the molecule increased by two-thirds after they used polycarbonate drinking bottles for 1 week [EHP 117:1368–1372; Carwile et al.].

Rodent studies have associated prenatal and neonatal exposure to BPA with early onset of sexual maturation, reproductive tract lesions, and altered development of the mammary gland, among other reproductive abnormalities. However, limited information is available on human health effects. Nevertheless, human exposure to BPA is widespread: the chemical was detected in the urine of more than 92% of the participants aged 6 years and older in the 2003–2004 National Health and Nutrition Examination Study (NHANES).

Not all polycarbonate plastics contain BPA, but nearly three-quarters of the BPA used in the United States in 2003 went into the manufacture of this one material. The hard, nearly shatterproof plastic is widely used in drinking bottles, baby bottles, and nonfood uses ranging from eyeglasses to labware. Earlier studies of polycarbonate drinking containers containing BPA have shown that under normal use—washing, rinsing, and exposure to high temperatures or to alkali or acid solutions—the plastic can degrade and release small amounts of the constituent chemical.

BPA is believed to be rapidly metabolized and eliminated. Therefore, in the current study, 77 college students aged 18–22 underwent a weeklong “washout” to minimize any preexisting BPA load that could have arisen from the use of polycarbonate drinking bottles. During the washout, participants were instructed to drink any cold beverages only from stainless steel bottles and to avoid drinking water from the polycarbonate dispensers in the college dining halls. After the washout, the group switched to drinking cold drinks only from new researcher-provided polycarbonate bottles for 1 week. Exposure to other BPA sources was not controlled; thus, the study yielded a conservative estimate of the potential for BPA exposure via polycarbonate drinking bottles.

Comparison of urine samples collected throughout the study showed that after using polycarbonate bottles for 1 week, participants’ mean urinary BPA concentrations increased by more than two-thirds to 2.1 µg/L, compared with the mean of 2.6 µg/L observed in the NHANES 2003–2004 study. The authors anticipate higher urinary BPA concentrations would result from drinking hot beverages stored in the same bottles.

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A Complex Relationship
Psychosocial Stress, Pollution, and Health

In recent years, a growing body of work has shown that psychosocial stress may exacerbate susceptibility to the adverse effects of pollutants such as lead, polychlorinated biphenyls, and combustion emissions. To accurately measure and evaluate the effects of stress on people’s susceptibility to pollutants, researchers need to rely on the tools and findings of both social epidemiology and environmental health science, according to a review of the research to date [EHP 117:1351–1358; Clougherty and Kublansky]. The authors offer specific recommendations for how researchers can combine techniques from these fields to investigate the links between stress, pollution, and health.

In the authors’ own earlier studies, they found that stress seemed to exacerbate effects of pollution, which suggests that stress increases susceptibility to environmental exposures. However, they also noted that the interaction between stress and pollution was no longer evident beyond a certain range of exposure, a phenomenon they refer to as the saturation effect. For example, if air pollution levels are very high, stress may have no additional effect on the likelihood of asthma symptoms occurring, and vice versa.

It’s also important to pay attention to differences in spatial patterns of social and physical exposures. As an example, the authors write, “spatial epidemiologists are challenged to differentiate health effects of traffic-related pollution from those of spatially correlated noise, stress, or poverty.” It can be particularly difficult to separate the effects of different exposures if they affect the same health outcomes. Moreover, not every individual within this sample neighborhood would necessarily experience high levels of stress, nor would every individual receive the same traffic-related pollution exposures, which the authors point out vary dramatically within 50–200 m of major roadways.

To understand the combined effects of stress and pollutant exposures, timing is everything because acute and chronic stress can produce different results. Acute stress can produce “fight-or-flight” responses that might counterbalance the effects of pollution—for example, stress-induced bronchodilation might temporarily reduce or mask bronchial constriction caused by air pollution. Chronic stress is more likely to gradually weaken the immune system, increasing susceptibility to pollution-related illness. Stress is also multidimensional; it includes the stimulus that poses the challenge, the person’s appraisal of the stressor, and finally the psychological and physiological response.

When measuring stress, researchers must consider what stage of the stress experience they are observing. They also must track the relative timing of study participants’ exposures to determine whether the stress occurred before, after, or during the pollutant exposure. In all, the authors write, “These topics are exceedingly complicated, and accurately characterizing both social and physical exposures is a significant challenge, one which must be performed carefully . . . before analyzing and interpreting interactions.”

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Dangerous Delicacy
Contaminated Sea Turtle Eggs Pose a Potential Health Threat

The eggs of the green turtle (Chelonia mydas) and other sea turtle species are a popular food in areas such as Peninsular Malaysia—so popular, in fact, that nesting populations in the region have declined by more than 80% since the 1950s, largely because of their eggs being collected for human consumption. Persistent organic pollutants (POPs) and heavy metals have been reported in the eggs of a number of C. mydas populations. Now a team of Australian and Malaysian scientists reports that the concentrations of POPs found in C. mydas eggs from markets in Peninsular Malaysia could pose a considerable threat to human health [EHP 117:1397–1401; van de Merwe et al.].

In August 2006, the investigators surveyed 33 markets along 730 miles of coastal Peninsular Malaysia. C. mydas eggs were available in 9 of these 33 markets. A random sample of 3–13 eggs was purchased from each market where they were sold. In total, 55 eggs were collected and frozen until they could be analyzed.

The eggs were analyzed for numerous POPs, among them 83 polychlorinated biphenyls (PCBs), 23 organochlorine pesticides, and 19 polybrominated diphenyl ethers. Eggs were also analyzed for zinc, copper, cobalt, selenium, arsenic, cadmium, lead, and mercury. For each metal and category of POP, the authors calculated the percentage of the acceptable daily intake (ADI) found in the eggs, providing an estimate of potential human health risks involved in consuming the eggs. ADIs are set by the World Health Organization.

The concentrations of POPs and metals measured were generally lower than those reported elsewhere for loggerhead sea turtle (C. caretta) eggs. Nevertheless, all the eggs analyzed had at least 3 times the ADI of coplanar PCBs, which are among the most toxic members of their chemical family. One egg had 300 times the ADI for this contaminant.

The authors note that the rate of C. mydas egg consumption in Peninsular Malaysia was not investigated in the present study, nor has it been well quantified. However, there is a cultural perception in this area that sea turtle eggs have medicinal qualities. The authors write that a public education campaign could highlight the health consequences of consuming contaminated eggs. This in turn could reduce egg collection pressure and hence potentially contribute to the recovery of the C. mydas populations in this region.

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