

Temperatures Rising Sprawling Cities Have the Most Very Hot Days

The urban heat island effect, the phenomenon in which a city has higher temperatures than surrounding countryside, is known to contribute to higher rates of heat-related mortality in summer months when temperatures soar. Although extreme heat events have become more common in large U.S. cities, a new study indicates that sprawling cities experience more than double the rate of extreme heat events in the summer compared with more compact urban areas [*EHP* 118(10):1425–1428; Stone et al.].

The authors considered 53 U.S. metropolitan areas for which data were available on sprawl and extreme heat event. Sprawl had been assessed in a 2003 study using an index based on land use data from the 2000 census along with measures of population density, average street block size, proximity of homes to businesses, and land use mix. Extreme heat events were defined as days on which the minimum, maximum, or average temperature exceeded the 85th percentile of a base period of 1961–1990.

The authors compared the extent of sprawl for each metropolitan area with that area's average annual rate of extreme heat events from 1956 through 2005. Over the 50 years examined,

the number of extreme heat events increased by an average of 14.8 days in cities with the most sprawl (e.g., Atlanta, Tampa, and Grand Rapids), whereas more compact cities (e.g., Chicago, Boston, and Baltimore) saw a lower average increase of 5.6 days of very high temperatures. The observed connection between extreme heat and sprawl was independent of climate zone and variations in size and growth of metropolitan populations. However, data analysis indicated that between 1992 and 2001 the deforestation rate in the most sprawling areas was more than double that of the most compact cities.

The study did not allow for an examination of the differences in rates of heat-related morbidity and mortality between sprawling and compact cities. However, the authors write that the numerous adverse effects associated with urban sprawl (e.g., high levels of ozone, poor water quality, and decreased physical activity) signal a need for public health officials to adopt more risk-reduction strategies such as preserving regional green space, installing green roofs, and replacing vehicular traffic with more public-transit options. They note that many of these strategies can also increase urban resilience to other climate-related risks including increased severe precipitation.

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A Compendium of Challenges Assessing the State of the Science on Occupational Carcinogens

Uncertainties abound about the adverse health effects of exposure to carcinogens found in today's workplaces. Even with substantial toxicologic evidence of carcinogenicity, cancer risks for humans often remain inconclusive, thus delaying regulatory action and the search for safer alternatives. A new systematic review by the International Agency for Research on Cancer (IARC) identifies research gaps and needs for 20 agents prioritized for review on the basis of evidence of

widespread human exposures and potential carcinogenicity in animals or humans [*EHP* 118(10):1355–1362; Ward et al.].

Drawing from an international collaboration by 25 health and research agencies and institutions, the report summarizes recommendations and broaches key topics pertaining to several chemicals, metals, dusts, and physical agents for which there is widespread human exposure, predominantly in occupational settings. The authors emphasize that carcinogenic agents can act through multiple pathways and mechanisms, including oxidative stress, epigenetic mechanisms, and immuno- and hormonal modulation. They then discuss overarching issues pertinent to the study of these mechanisms. For example, regarding the validation of oxidative stress biomarker assays, they write, "Research is needed to examine the relationship between exposure to toxic agents and oxidative stress biomarkers, and between these biomarkers and risk of cancer, while controlling for the many individual factors that contribute to oxidative stress."

Concerning genetic susceptibility to carcinogenic exposures, the authors caution that stable and reproducible associations are few. Examining genetic polymorphisms related to carcinogen metabolism and/or DNA repair may aid the identification of higher cancer risks in susceptible subgroups and clarify the role of specific agents in mixed exposures. Nonetheless, the magnitude of such associations may be modest and could entail multiple genes or metabolic pathways—thus making them hard to detect.

The report deals with only a fraction of the potentially carcinogenic agents found in today's workplaces, most of which have sufficient evidence of carcinogenicity in animals but limited evidence for carcinogenicity in humans. Because of a paucity of well-designed animal bioassays and human studies, insufficient evidence exists to evaluate animal or human carcinogenicity for most other agents. The report ends on a somber note, noting that substantial challenges for the study of environmental carcinogens remain, including a recent decline in funding for occupational cancer research, and that fewer scientists are entering the fields of epidemiology, toxicology, and exposure assessment.

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Diminished Protection? Early Childhood PCB Exposure and Reduced Immune Response to Vaccinations

Polychlorinated biphenyls (PCBs) constitute a class of persistent organic pollutants suspected or known to cause adverse health effects. Among these effects are immune system dysfunctions that may hinge on both the magnitude and the timing of PCB exposure. A new study uses the backdrop of routine childhood immunizations to explore the developmental immunotoxicity of PCBs and finds that higher PCB exposure in toddlerhood is associated with reduced antibodies against diphtheria and tetanus later in childhood [*EHP* 118(10):1434–1438; Heilmann et al.].

The study took place in the Faroe Islands, midway between the Shetland Islands and Iceland in the North Atlantic, and involved 587 children from a 1999–2001 birth cohort. A traditional diet including pilot whale blubber, consumed by some but not all Faroese, creates a wide range of PCB exposures for this population. To assess the transfer of PCB from a mother to her child, maternal blood samples were taken in week 32 of pregnancy. The mothers also provided milk samples at 4–5 days after birth.

Based on the routine vaccination schedule, children were immunized against diphtheria and tetanus at 3, 5, and 12 months, with a booster at 5 years. Approximately one-fifth of the children had blood drawn at 18 months, and blood samples were drawn



before the booster shots at age 5 years for 532 children and 7 years for 464 children. PCB concentrations were assessed in blood and milk samples, and diphtheria and tetanus antibodies were measured in children's blood at ages 5 and 7.

Analysis revealed inverse relationships between PCB concentrations at different time points and antibody concentrations. The associations between concomitant measurements were not significant at either 5 or 7 years. However, higher PCB concentrations in mother's milk samples collected after birth and in children's blood samples at 18 months were clearly associated with lower levels of diphtheria antibodies in the children at age 5. When PCB concentrations at 18 months were estimated for the entire cohort based on known levels at birth and at 5 years paired with breastfeeding duration and PCB concentrations measured in blood samples from a subset of children at 18 months, this relationship became even stronger for diphtheria at both 5 and 7 years, and a similar relationship for tetanus antibody concentration at age 7 became significant.

The authors point out that early-life PCB exposure may increase the risk of incomplete protection against diphtheria and possibly tetanus even if a child receives a full schedule of vaccinations. But the implications of the results extend beyond vaccination because diphtheria and tetanus immune response reflects the efficacy of the immune system against a broad array of infections.

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Thyroid Insult Flame Retardants Linked to Alterations in Pregnant Women's TSH Levels

Polybrominated diphenyl ether (PBDE) flame retardants have been added to a wide variety of U.S. consumer goods including automobiles, airplanes, electronics, home furnishings, and furniture. Although two forms of PBDEs were phased out of use by 2004, data from the Centers for Disease Control and Prevention suggest exposure to these persistent, bioaccumulative, potentially toxic compounds remains widespread among the general U.S. population. New research now links higher exposure to PBDEs with reduced levels of thyroid-stimulating hormone (TSH) and higher rates of subclinical hyperthyroidism in women [*EHP* 118(10):1444–1449; Chevrier et al.]. The findings may have implications for maternal health and fetal development.

Thyroid hormones are known to play an essential role in fetal brain development, and altered maternal thyroid hormone levels may adversely affect child neurodevelopment. The authors cite multiple animal studies showing that PBDEs alter pregnant rodents' thyroid functioning. In the last year, laboratory tests using human cells suggested that PBDEs may affect brain development and linked the alterations to disruptions in thyroid hormone signaling. Recent research also has linked higher PBDE exposure to reduced intelligence in children.

The authors believe their new study to be the largest to analyze associations between PBDEs and thyroid hormone during

pregnancy. The 270 study participants were pregnant in 1999 and 2000 when they were recruited for the Center for Health Assessment of Mothers and Children of Salinas (CHAMACOS) study. The research team measured the women's thyroid hormone levels, then evaluated the data with models that adjusted for potential confounding factors, such as demographic characteristics and exposure to other persistent, bioaccumulative, and toxic compounds.

Women with higher blood PBDE levels had lower levels of TSH. The women with the highest levels of PBDEs were more likely to have lower TSH but normal levels of the thyroid hormone thyroxine (T_4), suggesting subclinical hyperthyroidism. The combination of lower TSH and "normal" T_4 —as defined by the reference range applied to lab results—could mean an individual's T_4 levels actually are elevated above her body's "set point" (or optimal level) of T_4 , which varies from person to person.

Most human studies in this area have associated increased exposure to PBDEs with elevated free T_4 . The only other study known to examine PBDEs and thyroid hormone specifically in pregnant women—which included only nine women—did not find increased T_4 , but the authors did not examine TSH levels. In contrast, other studies of pregnant women's thyroid hormone levels have linked higher exposure to organochlorine pesticides and polychlorinated biphenyls—which have chemical structures and properties similar to those of PBDEs—with increased TSH and decreased T_4 .

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