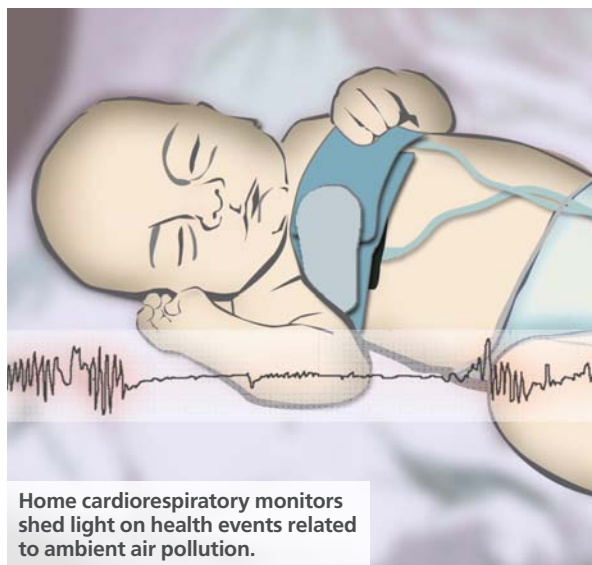


Ambient Air Pollution and Infant Health

Home Monitors Make Cardiorespiratory Connections

Children are particularly susceptible to the health effects of air pollution because they spend more time outdoors, have higher respiratory rates, and breathe in a greater volume of air relative to their body weights. Babies may be especially sensitive to the effects of air pollution because their immune, respiratory, and central nervous systems are not fully developed. To date, infants' respiratory responses to air pollution have been studied much less extensively than those of older children. A new study now links ambient air pollution to an increased risk for apnea (prolonged pauses in breathing) and bradycardia (decreases in heart rate) in babies at high risk for these conditions [*EHP* 119(9):1321–1327; Peel et al.].

The study involved 4,277 infants living in the Atlanta area (about 80 square miles) between 1998 and 2002 whose heart rates and respiration were recorded on home cardiorespiratory monitors. Most of the infants were being monitored because of previous apnea events related to premature birth; others, including some full-term infants, were being monitored for reasons such as gastroesophageal reflux disease. Concentrations of ground-level ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, oxygenated



Home cardiorespiratory monitors shed light on health events related to ambient air pollution.

hydrocarbons, and particulate matter were measured at a centrally located monitoring site.

The team of researchers documented 8,960 apnea events (in which the child stopped breathing for at least 20 seconds) and 29,450 bradycardia events (in which the child's heart rate fell below a value determined by his or her age and prematurity status) recorded on the monitors. They examined associations between these events and the 2-day average levels of air pollution, recorded the same day as and the day before each event.

The researchers found significant associations between bradycardia and increases in 8-hour levels of ozone and 1-hour levels of nitrogen dioxide. The relationship between ozone and apnea was similar but not statistically significant. In general, stronger associations were observed for full-term infants with normal birth weights than for premature infants with low birth weights—a surprising but not unprecedented finding.

There also was an association between apnea events and increased concentrations of organic carbon in fine particulate matter for full-term, normal-birth-weight infants.

These findings are consistent with previous studies linking air pollution with respiratory symptoms, related hospital admissions, and increased mortality in infants. Although the underlying causes of apnea and bradycardia are unclear, some evidence suggests that immaturity in the autonomic control of the nervous and/or respiratory systems may be involved, which makes a link with increased vulnerability to the effects of air pollution plausible.

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Warm Reception?

Halogenated BPA Flame Retardants and PPAR γ Activation

Adverse effects highlighted in experimental studies of the industrial chemical bisphenol A (BPA) have centered on reproductive targets, but recent research suggests metabolism also may be affected. BPA is widely present in the environment, as are its halogenated derivatives. A new study investigates the structure and function of these halogenated compounds and shows they could potentially disrupt energy balance (i.e., lipid and carbohydrate homeostasis) in humans and wildlife [*EHP* 119(9):1227–1232; Riu et al.].

Halogenated BPA derivatives are used as flame retardants, with tetrabromobisphenol A (TBBPA) and tetrachlorobisphenol A (TCBPA) as parent compounds. Previous studies have shown that BPA, TBBPA, and TCBPA have estrogenic activity and that BPA can alter adipogenesis (fat cell formation) in rats. Peroxisome proliferator-activated receptor γ (PPAR γ) plays a key role in adipogenesis, and interference with this receptor can contribute to type 2 diabetes and obesity.

Cell lines were used to test the ability of BPA, TBBPA, TCBPA, and other halogenated BPA derivatives to activate human estrogen receptors (ER α and ER β) and human PPARs (PPAR α , PPAR δ , and PPAR γ). The compounds also were applied to undifferentiated fat cells to test their effects on adipogenesis and the genes involved in this process. Competitive binding assays were conducted to further

characterize PPAR γ activation in both reporter cell lines and undifferentiated fat cells, and crystals of PPAR γ bound to TBBPA and TCBPA were purified to study how the chemicals bound and activated the receptor. Another set of experiments used *Xenopus* and zebrafish PPAR γ to test the response to BPA compounds in other species.

TCBPA and lower brominated BPAs such as monoBBPA and diBBPA partially activated both ER subtypes, whereas TBBPA had little effect on either. Both TBBPA and TCBPA were able to partially activate PPAR γ and promote fat cell differentiation as well as disrupt PPAR γ activity in all species studied. Structural examination of the area where compounds are bound to the PPAR γ receptor yielded evidence that TBBPA, TCBPA, and other halogenated BPA derivatives partially activate this receptor.

What does all this mean? Essentially, the findings support the hypothesis that environmental contaminants may contribute to the disruption of energy balance in humans and wildlife. It also suggests that halogenated derivatives of BPA used as flame retardants, which have not been extensively studied to date, may contribute to obesity. The main active form of PPAR γ is the retinoid X receptor (RXR)/PPAR γ heterodimer. According to the authors, environmental exposure to BPA compounds alongside other endocrine disruptors with similar RXR and/or PPAR γ activity sets the stage for additive and synergistic effects, potentially leading to increased risk of metabolic disease.

Julia R. Barrett, MS, ELS, a Madison, WI–based science writer and editor, has written for *EHP* since 1996. She is a member of the National Association of Science Writers and the Board of Editors in the Life Sciences.