

Swimmer's Lung? Indoor Pools and Respiratory Effects

Bronchiolar epithelium cells known as Clara cells are thought to help defend airways against damage; Clara cell protein (CC16) is a lung-specific protein thought to protect the respiratory tract from inflammation. Studies have shown a positive association between ozone exposure and increased concentrations of CC16 in blood serum, leading researchers to suggest that ozone exposure damages



Bathers beware? Repeated exposure to disinfection by-products around indoor swimming pools may damage cells that produce protective lung-specific proteins.

the lung epithelium, causing it to “leak” proteins such as CC16. Now Birgitta Jön Lagerkvist of Umeå University in Sweden and colleagues present findings that complicate that idea [*EHP* 112:1768–1771].

Lagerkvist and colleagues report that children who regularly visited indoor swimming pools actually showed decreased concentrations of CC16 both before and after sessions of exercise outdoors in ambient levels of ozone. The researchers theorize that repeated exposure to disinfection by-products around indoor swimming pools may damage Clara cells, decreasing production of CC16 and possibly masking any effect that ozone exerts on CC16 levels.

This report is part of a set of studies examining possible changes in CC16 serum levels in relation to ambient ozone exposure as well as exposure to other environmental agents such as the chlorine around swimming pools and its by-products. In addition to the current study, which examined children in the summer, three other studies have examined children in winter, adults in summer, and adults in winter.

Subjects were screened using blood samples, lung function tests, and questionnaires. The final 57 subjects were 33 boys and 24 girls aged 10–11 years who had no asthma, pollen allergy, or initial decreased lung function. One subset of the 34 children had

visited an indoor pool for at least one hour per month for six months or longer; the remaining 23 children had not.

The children exercised lightly outdoors for two hours on the Umeå University campus, where the ambient ozone was a moderate 77–116 micrograms per cubic meter. Lung function testing and blood sampling were conducted both before and after exposure.

The researchers saw no impairment of lung function in any of the children, and in looking at the entire study group, they did not find any statistically significant relationships between ozone exposure and CC16 concentrations. Among the children who did not visit swimming pools, there was a marginally significant tendency toward such a correlation. However, the children who regularly visited chlorinated indoor swimming pools showed significantly lower CC16 serum concentrations both before and after ozone exposure, compared to those who didn't visit pools.

The authors say this may indicate that repeated exposure to disinfection by-products around indoor swimming pools damages Clara cell function. This theory is supported by previous studies, including one by report coauthor Alfred Bernard that showed an association between regular pool attendance among people taking swimming lessons and reduced CC16 concentrations. The researchers speculate that if regular indoor pool attendance does decrease CC16 production, then this effect may mask any increased leakage of CC16 caused by ozone. They further suggest that such masking may have happened in this study, indicated by the slight tendency to show a correlation between ozone exposure and increased CC16 concentrations only in the children who did not visit swimming pools.

They conclude that Clara cell damage associated with indoor pool use may diminish the anti-inflammatory effect of CC16 in the lung. Another previous study by Bernard showed increased incidence of asthma among children who regularly visited indoor pools, and the authors of the current report call for further

investigation of the possibility that exposure to disinfection by-products around indoor pools can play a role in inducing asthma through impaired Clara cell function. —**Angela Spivey**

Roe, Interrupted Estrogen Exposure Impairs Fish Fertility

Major research efforts have shown that endocrine disruptors—environmental chemicals that can interfere with the endocrine system—may affect reproduction of wildlife and even humans. Studies in fish, for example, have shown that endocrine disruptors can reduce sperm count, induce both male and female gonadal tissue or intermediate sexual characteristics in the same individual, and induce female-specific proteins in males. But little evidence to date has elucidated the effect of such changes on fertility. This month, Jon Nash of the Katholieke Universiteit Leuven in Belgium and colleagues report that long-term exposure to low concentrations of a synthetic estrogen may severely undermine the breeding success of wildlife populations, chiefly by producing sexually compromised males who disrupt breeding dynamics [*EHP* 112:1725–1733].

Using zebrafish because of their short generation time, the researchers measured effects of exposure over three generations. They

began with 720 fish divided into 60 groups of 12. The team recreated natural conditions in the aquaria to optimize fish breeding, and eggs were collected each day.

After a baseline assessment of egg numbers and egg viability (a cumulative statistic of unfertilized eggs and embryo mortality), the researchers exposed different groups to environmentally relevant concentrations of various estrogens: 5.0 nanograms per liter (ng/L) of the endogenous estrogen estradiol or either 0.5, 5.0, or 50.0 ng/L ethynylestradiol, a potent synthetic estrogen used in oral contraceptives. A control group received no exposure.

Except for the highest concentration of ethynylestradiol, none of the estrogen treatments affected egg numbers or egg viability in the baseline generation. Nor did any of the treatments affect survival of the eggs spawned by this generation.

But after 210 days (a full zebrafish lifetime) of exposure to the middle dose of 5.0 ng/L ethynylestradiol, the second generation of fish showed reduced fertility. None of the male fish in the second generation had normal testes, and they did not produce expressible sperm, although the females were fertile. None of this generation's progeny survived beyond 14 hours postfertilization. In almost 12,000 eggs spawned, none were viable.

When two healthy, nonexposed males were added to the populations that had experienced reproductive failure, embryos began surviving. But the embryos' rate of survival was still significantly less than in the control group. After close observation of the spawning in these tanks, the researchers found that the infertile males showed normal reproductive behavior, chasing the spawning females and competing with the fertile males for access. The researchers suggest that the reduced fertilization was caused at least in part by the compromised males interfering with the fertilization capability of the healthy males.

The researchers say their data show that development of the testes is more sensitive to disruption by ethynylestradiol than is reproductive behavior. Yet the relatively higher threshold of sensitivity of behavioral disruption may in fact produce stronger population-level consequences, as infertile males have a greater ability to interfere with breeding dynamics. They conclude that more information about the effects of endocrine disruptors on the interactions between fish in a spawning group is needed before the population-level effects of endocrine disruption can be understood. —**Angela Spivey**

POPs in Polar Bears

Organochlorines Affect Bone Density

Both organochlorine chemicals—including a series of solvents and pesticides that have been banned in many parts of the world—and their metabolites have been linked to bone loss in a variety of species. Now, Christian Sonne of the Danish National

Environmental Research Institute and colleagues have found a similar association in a new species [*EHP* 112:1711–1716]. Their work shows a link between organochlorine exposure and reduced bone mineral density among polar bears in East Greenland.

Organochlorines are among the chemicals known as persistent organic pollutants (POPs), which resist breakdown, store easily in fat, and bioaccumulate through the food chain. Organochlorines have been accumulating in the Arctic for decades, thanks to northward atmospheric transport. Due to the quirks of atmospheric transport, polar bears from East Greenland, Svalbard, and the Kara Sea have higher body burdens of organochlorines than polar bears from the rest of the Arctic.

In controlled studies of laboratory mammals, organochlorines including the pesticide DDT and the group of industrial chemicals known as polychlorinated biphenyls (PCBs) have caused changes in bone composition including reduced bone mineral density. Organochlorines have also been implicated in other bone diseases including periodontitis, a disorder of the gums and bones around the teeth.

The Danish researchers examined samples from 139 East Greenland polar bear skulls collected between 1892 and 2002. Samples collected in the period 1966–2002 were considered “post-pollution”—that is, they were collected after high concentrations of POPs began appearing in polar bear fat. Those collected in the period 1892–1932 were considered “pre-pollution.”

The researchers measured bone mineral density with dual X-ray absorptiometry—the same test used to detect osteoporosis in humans. They also examined organochlorine body burden in a subset of 58 samples collected between 1999 and 2002 for links with bone mineral density.

Among younger bears and adult males, bone mineral density was significantly reduced in post-pollution samples. The pattern was not seen among

adult females, possibly due to an age-associated decline of estrogen, or because they were pregnant or nursing pups (both conditions mobilize bone calcium for the benefit of the offspring, and reduce bone density). Bone formation and resorption are governed by estrogen and androgen hormones, which help regulate both osteoblasts (cells that form bone cells) and osteoclasts (cells that break down bone cells).

In the 58 skulls collected between 1999 and 2002, exposure to total PCB compounds and to chlordane (a now-banned insecticide) both correlated with low bone mineral density among younger bears. In adult males, concentrations of dieldrin (another banned insecticide) and total DDT residues also correlated with low density. The researchers conclude that disruption of bone mineral composition correlates with the presence of PCBs, DDT residues, and other POPs among polar bears from East Greenland. —**David J. Tenenbaum**



Bear-bones data. New research shows a link between Arctic organochlorine pollution and decreased bone mineral density in East Greenland polar bears.