Hit or Miss?

Benefits and Risks of Using Nanoparticles for *in Situ* Remediation

Nanotechnology holds the promise of vastly expanding our ability to clean up hazardous waste sites and decontaminate polluted groundwater *in situ*. Polychlorinated biphenyls, organic solvents, petroleum products, arsenic, and many more contaminants are on the list that specifically engineered nanoparticles could rapidly remove from contaminated soil and water, saving billions of dollars that would have been spent on more expensive conventional remediation methods. These possibilitites are discussed in a review of field tests using nanomaterials [***EHP* 117:1823–1831; Karn et al.**]. However, the authors caution, our knowledge of the potential environmental and health hazards posed by these nanomaterials is in its infancy.

A comparison of remediation using nano- versus microscale zero-valent iron particles at Hunters Point Naval Shipyard (shown here in 1971) showed that each size had unique advantages.

In the United States alone there are hundreds of thousands of sites contaminated with hazardous wastes, with more than 1,200 requiring priority attention. Using traditional remediation technologies, such as pumping out and treating contaminated groundwater and removing contaminated soil, the job of cleaning up U.S. hazardous waste sites could take 35 years and $250 billion. The authors of this review, however, report on results showing that nanoscale zero-valent iron (nZVI) nanoparticles could dramatically reduce the time required to remediate soil and water as well as, according to one report, save 80–90% compared with conventional methods.

Although tiny in diameter, the surface area of these iron-based nanoparticles reaches 20–40 m^2/g. This relatively large surface area can greatly increase the particles' reactivity. Flowing with the groundwater they can spread out to react with pollutants, transforming them into safer compounds, including compounds that bacteria can break down. The authors provide many examples of how nZVIs, currently the nanomaterials most widely used for *in situ* remediation, produced measurable effects within days or, in some cases, hours.

However, the authors also emphasize that we do not know much about the potential adverse effects of nanoparticles deployed into the environment—agents that could, for example, end up in our drinking water. Some nanomaterials have already been found to enter organisms. Might some be toxic or transport bound pollutants to places they might not otherwise have gone? Can they be biomagnified? How do they affect living organisms?

The authors point out that, although the environment is full of naturally occurring nanoparticles, manufactured nanoparticles may behave in unpredictable ways. They recommend that while we improve engineering applications using nanoparticles for *in situ* remediation, we also develop the analytical tools to enable the study of manufactured nanoparticles in the environment and increase research on the ecosystem effects of these materials.

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Cadmium and Breast Cancer

Exposure Associated with Basal-Like Phenotype

Cadmium has been linked with several human diseases including chronic kidney disease and cancer. As a carcinogen, cadmium targets several sites that are considered endocrine-sensitive, and some data suggest the breast may be among them. Although cadmium has been hypothesized to act as a metalloestrogen—a metal that triggers an estrogen-like reaction—research to date has not confirmed this as a mechanism linking cadmium and breast cancer. Additionally, although many breast cancers are estrogen-dependent, some of the most difficult-to-treat cases are not. A new study finds that cadmium can induce malignant transformation in breast cells *in vitro* regardless of the absence of estrogen receptors, strengthening evidence that cadmium exposure may be a factor in breast cancer, a leading cause of cancer deaths among women [***EHP* 117:1847–1852; Benbrahim-Tallaa et al.**].

MCF-10A cells, which are derived from normal human breast epithelium, were grown with either no cadmium exposure or continuous cadmium exposure (2.5 µM) for up to 40 weeks. Positive controls included MCF-7 human breast cancer cells (which express the estrogen receptors ER-α and ER-β) and SKBR3 breast cancer cells (which express HER2, a receptor that can be overexpressed in certain malignant breast cancer cells). In contrast, MCF-10A cells do not express ER-α, ER-β, or HER2 proteins, although expression can be acquired in carcinogenesis.

Chronic cadmium exposure of the MCF-10A cells yielded increased expression of matrix metalloproteinase-9, an enzyme that facilitates tumor cell invasion. These cells also formed cell mounds, indicating a loss of contact inhibition (the natural process of cell growth stopping once a certain density of cells is reached). When these transformed cells were implanted in mice, they formed highly aggressive tumors that demonstrated metastatic potential.

Transformed MCF-10A cells remained negative for ER-α and ER-β and also lacked HER2 protein. However, metallothionein, typically overexpressed in ER-negative breast cancers, was elevated as were several other breast cancer markers. These characteristics collectively suggest that cadmium could be a risk factor for a basal-like breast cancer phenotype, which is clinically associated with a higher risk of relapse after treatment and lower survival rates.

The precise mechanism by which cadmium may transform breast cells is unknown, but the results of this study suggest it is unlikely to be a metalloestrogenic effect via estrogen receptors. Although additional research is needed to define the mechanism, the current study provides strong evidence that cadmium may play a role in human breast cancer.

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Lt. A. Legare/U.S. Navy

Pesticides and Parkinson's Disease

The Legacy of Contaminated Well Water

Epidemiologic studies since the early 1990s have suggested that exposure to various classes of pesticides increases the risk of developing Parkinson's disease (PD). Animal studies have backed up that link, revealing how pesticides may target the dopaminergic system, which is damaged in PD. New data from the Parkinson's Environment and Genes Study show that residents of California's Central Valley who over many years drank well water that was probably highly contaminated with certain pesticides were more likely to have PD or PD symptoms than residents who didn't drink contaminated well water [***EHP* 117:1912–1918; Gatto et al.**].

Private wells are at risk of pesticide contamination because pesticides can drift several hundred meters from application sites and travel through the soil. Moreover, many of the private wells studied were less than 15–20 yd deep, lessening the likelihood that pesticides will have degraded by the time they reach the water supply. Unlike municipal water supplies, private well water is not required to be monitored for contamination.

The researchers analyzed long-term data on pesticide application rates near the homes of the study participants, who included 368 people clinically confirmed to have possible or probable PD and 341 controls. The authors had access to 26 years' worth of data collected under California's mandated pesticide use reports program on the commercial application of pesticides, including where the pesticides were applied, on what date, and in what quantities. They studied 26 pesticides that were potential groundwater contaminants or that had been previously linked to PD.

The researchers combined those data with land-use maps, which the California Department of Water Resources updates every 7–10 years, to pinpoint more precisely where pesticides had been applied. Using geographic information system software, they were able to merge historical data on home addresses, land use, and pesticide applications. The result was a prediction of amounts of pesticides applied per acre per year within 500 m of the study participants' homes.

People with PD were more likely to get their water from private wells and to have drunk well water longer than controls. Whereas people with exposure to ambient pesticides—essentially, proximity to sites where pesticides were applied—were 15–57% more likely to be classified as having PD than people without ambient exposure, those with combined ambient exposure and exposure via well water potentially contaminated with methomyl, chlorpyrifos, or propargite were 67%, 87%, or 92% more likely to be cases. The odds of PD also increased as the number of different pesticides that potentially contaminated a subject's drinking water increased.

Unlike previous research on the link between PD and pesticide exposures, this study used a semiquantitative approach to estimating pesticide exposure and did not rely on study subjects' recall. Also, all PD cases were clinically confirmed by a movement disorder specialist. The results therefore considerably strengthen the evidence that exposure to pesticides in well water may contribute to PD.

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Chronicle of a Health Crisis

Global Implications of the 2008 Melamine Event

Melamine is commonly used to manufacture strong and durable laminates, plastics, adhesives, and flame-resistant textiles. It also has been deliberately added to food and animal feed, sometimes in high amounts, to boost the appearance of protein content based on nitrogen analysis. The result can be serious health threats, including renal failure and death. In 2007–2008, for instance, the deliberate addition of melamine to raw milk used in powdered infant formula and other milk and dairy products caused an outbreak of kidney stones and renal failure in Chinese infants and raised significant implications for global food and feed safety [***EHP* 117:1803–1809; Gossner et al.**].

Because melamine is used in such a wide range of products, its trace presence in many foods is inevitable—action is not usually taken if levels are below 1.0 mg/kg for infant foods or 2.5 mg/kg for other food products. In comparison, contaminated powdered infant formula produced by the Sanlu Group and distributed in China contained up to 2,563 mg/kg.

Using information originally reported by the Chinese Ministry of Health to the World Health Organization and shared through the International Food Safety Authorities Network (INFOSAN), the authors describe the unfolding of events from the first reported cases of sick babies in China to the export of contaminated dairy and nondairy products (including ammonium bicarbonate, fresh and dried eggs, nondairy creamer, and animal feed) that eventually reached 47 known countries. Although parents who used Sanlu formula first began filing complaints in December 2007, the global community did not become aware of the crisis until September 2008.

Countries responded in a variety of ways ranging from taking no action at all to banning all imports of milk and dairy products from China. Meanwhile, China reported a total of 6 child deaths and 294,000 cases of children affected by consumption of contaminated formula and milk products. Health effects ranged from discolored urine to kidney stones to acute renal failure and subsequent death. Because milder cases were often asymptomatic, many more children may have been affected both in China and abroad.

Given the potential global impact of the 2008 event, the authors state that well-structured national food safety systems—combined with coordination among food safety authorities and rapid communication through INFOSAN—are key components in controlling such outbreaks. There also should be one harmonized set of international standards for acceptable levels of potential contaminants in food and feed products as well as universal methods of detection, prevention, and containment.

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Chinese parents wait to have their babies examined for melamine effects at a Suzhou hospital, 22 September 2008.

Wang Jiankang/Imaginechina