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.

Adrian Burton is a biologist living in Spain who also writes regularly for *The Lancet Oncology*, *The Lancet Neurology*, and *Frontiers in Ecology and the Environment*.

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.

Julia R. Barrett, MS, ELS, a Madison, Wisconsin–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.

Lt. A. Legare/U.S. Navy