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Is China's Pollution Poisoning Its Children?

Epidemiologists find molecular clues to air pollution's impact on youngsters

By Dan Fagin

ILL WIND: A young boy wears a mask against pollution in Linfen, China. Preliminary results from a study conducted in Tongliang, China, reveal that children exposed to highly polluted air while in the womb had more changes in their DNA—and a higher risk of developmental problems—than did those whose mothers breathed cleaner air during pregnancy.
Peter Parks AFP/Getty Images

KEY CONCEPTS

A central goal of molecular epidemiology is to tie environmental factors to genetic changes that contribute to disease.

Some biologists have questioned the approach, because few candidate molecular markers of susceptibility, exposure or early disease have yet been proved to foretell future illness.

Now researchers may have found the best test case yet for environmental molecular epidemiology: a city in China whose coal-fired power plant was shut down in 2004.

Preliminary analysis shows that children born in 2002, when the plant was still operating, have smaller heads and lower scores on developmental tests than those born a year after the plant closed. They also have correspondingly higher levels of pollution-related genetic abnormalities.

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A few heaping piles of scrap metal and a rusty coal shed are all that is left of the power plant that until recently squatted like an immense, smoke-belching dragon in the middle of Tongliang, a gray city of 100,000 in south-central China. As we walk toward the shed, a Belgian Shepherd begins barking furiously, jerking its iron chain and baring sharp teeth. A brown-eyed face peeks out from the open doorway—it belongs to a girl in a stained shirt, holding a tabby cat that jumps away to hide under a slab of concrete as we approach. The girl is no more than six or seven years old and appears to be living in the shed with her father, who watches us warily from within.

The delegation of local officials who are taking us on a tour of the site are embarrassed; they want to hustle us along to a nearby office to show us an elaborate scale model of an extravagant (by Tongliang standards) 900-unit housing development planned for the property. But Frederica Perera is intrigued. She strides toward the girl and gives a friendly "*ni hao*" and a smile. The girl smiles back before retreating back into the shadows with her father.

Children, after all, are why Perera is here. She is looking for connections between air pollution and disease, especially in children who were exposed to pollutants in the womb. The director of Columbia University's Center for Children's Environmental Health, Perera helped to pioneer the field of molecular epidemiology, which applies the tools of molecular analysis to identify genetic and environmental factors that contribute to disease. She and other molecular epidemiologists who focus on environmental links to illness increasingly do much of their work in the developing world, where pollution is so ubiquitous that its complex connections to health can be calibrated even in small study populations. But their conclusions should also apply in places such as the U.S., Europe and Japan, where environmental exposures are subtler and their effects more difficult to measure in small-scale studies.

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Wherever they work, what distinguishes the approach of molecular epidemiologists is their search for biological indicators that closely correlate with toxic exposures and illness. Often these markers take the form of chemicals bound to DNA or of changes in gene structure or activity that match up with particular types of contaminants and disease. Now that DNA microarrays and other screening technologies are making it much easier to measure many of those biomarkers, routine use of such tools could, at least theoretically, save lives by identifying populations at risk from specific pollutants.

The science is still controversial, however, because relatively few candidate molecular biomarkers of susceptibility, exposure or early disease have been fully validated—that is, proved to herald future illness—and because it is very difficult to factor out confounding variables such as diet and genetic predisposition that may be at least as important as exposure to pollutants in causing various ailments. What has proved even more difficult is getting a handle on how those disparate risks may be interacting to affect health.

As a result, more than 25 years after Perera's first paper on the topic, molecular epidemiology has progressed more slowly than its architects had initially hoped, and the consensus among researchers is that no matter how enticing it sounds in theory, real-world complexities have limited its usefulness. Initial enthusiasm for the idea that changes in a few specific biomarkers—the tumor-suppressing protein p53 is a prominent example—could be reliable indicators of early illness has faded as researchers have identified much more complicated etiologies, involving cascades of biochemical changes, for many diseases. "You could say that the