Chemistry

Stefi Weisburd reports from New Orleans at the 194th national meeting of the American Chemical Society

Hunting for Legionnaire's bacteria

In July 1976 a little-known bacterium became infamous when it killed 34 people attending an American Legion convention in Philadelphia. Since then, at least 700 nonfatal cases of Legionnaire's disease have been reported each year. The discovery of *Legionella pneumophila* in the plumbing system of hospitals and cooling towers in a number of cities has caused concern among officials who worry that the bacterium may be ubiquitous throughout city water supplies.

In this regard, a recent study of the Pittsburgh water system offers mostly good news. Stanley J. States at the Pittsburgh Department of Water and his colleagues report that they "were unable to detect [the bacterium] anywhere in the system, including a number of sites in the treatment plant, the reservoir and the main system." The results, he says, are consistent with findings in several other cities. However, the researchers did isolate *Legionella* in very low numbers in the Allegheny River and in some hospital tapwater samples. They also pegged a few sites – such as the reservoir bottoms – where *Legionella* could easily multiply since the water was stagnant, nutrient-rich or inadequately chlorinated.

According to States, *Legionella* is less susceptible to chlorine than are the coliform bacteria that are usually used as a guage of water quality, but he notes that chlorine levels of well-maintained water systems should discourage the bacteria from homesteading. The group also found that zinc and iron, which can be leached from plumbing pipes, and potassium in the water supply enhance the organism's growth.

Finally, States stresses that the presence of the bacterium is by itself not necessarily dangerous. In general, it has attacked only people whose immune systems have already been compromised in some way. More work is needed, he says, to understand all the factors that must conspire to make people ill.

Radical dangers up in smoke

Where there's smoke, there's fire – and free radicals. These electron-hungry compounds are highly reactive and can cause severe damage to biological tissues. For example, cigarette smoke is loaded with free radicals that scientists think are involved in emphysema and possibly lung cancer. Now researchers at the Biodynamics Institute at Louisiana State University in Baton Rouge report that free radicals are also present in the smoke of burning buildings. They suggest these radicals may account for at least some of the 500 or so unexplained fire-related deaths that occur in the United States each year, and they suggest possible ways to protect against such radicals. The group also has some new findings on how free radicals in cigarette smoke and tar may damage the lungs.

Graduate student Thomas M. Lachocki and chemists Daniel F. Church and William A. Pryor detected several kinds of free radicals in the smoke of burning cellulose materials, paint, polyethylene and rubber. They did not find the radicals in nylon, polytetrafluorethylene or polyvinyl chloride.

The researchers also discovered that while tobacco-smoke free radicals are formed within seconds, the free radicals that they detected could appear in cellulose smoke more than 20 minutes after combustion. Since the radicals themselves have extremely short lifetimes, the researchers propose that some as-yet-unidentified, long-lived "metastable intermediate" is created in building fires and that this compound later decomposes into a free radical.

This would mean, according to Pryor, that metastable intermediates could travel a distance from a fire, make their way deep into the lungs and then later produce free radicals, which might themselves be lethal or make a person more susceptible to low levels of fire-produced toxins. This mechanism, he says, may explain the deaths that can't be attributed to heat, flames or inhalation of deadly levels of known toxins such as hydrogen cyanide or carbon monoxide.

"If all this is true," he adds, "it suggests a strategy for treating fire victims. . . . We're envisioning that a person brought out of a fire could inhale a vapor that would protect against free radicals." Possible protective substances include vitamin E, vitamin C and superoxide dismutase.

In another study led by Church, researchers set out to test a long-held assumption that the black color in smokers' lungs is due to tarry deposits from cigarettes. Instead, they found that the color is due to an inorganic iron compound.

"That's a striking observation because iron is known to play a very critical role in propagating free radical damage in tissue," observes Pryor.

Because this compound is spectroscopically similar to hemosiderin, a protein used by the body to store iron, Church speculates that the action of iron may be similar in the two compounds. Based on what is known about hemosiderin, he proposes that the iron deposits trigger a runaway cascade of oxidative damage in smokers' lungs. In particular, free radicals in cigarette smoke may cause small hemorrhages, which would lead to the release of iron from the blood and its subsequent collection at the damage sites, where it would catalyze the production of more free radicals, and so forth.

In yet another study, Pryor's group homed in on a possible mechanism by which free radicals may abet mutagenesis of cells, leading to lung cancer. Previously the researchers had shown that a water-soluble component of tar reacts with oxygen to form free radicals. They found that these free radicals break one of the strands of DNA and that the DNA nick is such that it takes several enzymatic steps to repair the damage. These breaks, notes Pryor, are similar to those caused by radiation, which has also been linked to cancer.

"DNA damage is an ongoing fact of life," Pryor says. "All cells suffer damage. . . . But the more [repair] processes you have to go through, the more likelihood there is for a mistake. Some types of DNA damage, such as that observed in this study, may be a better source [than others] of mutagenic events."

Needed: A cure for curing snuff

Even when there's no smoke, tobacco can blacken human health. Smokeless tobacco, chiefly chewing plugs and snuff, has been linked to oral cancer, gum disease and nicotine addiction. In the United States, 12 million people were using it as of 1985.

Chemist William J. Chamberlain of the U.S. Department of Agriculture in Athens, Ga., and his colleagues report that the amount of potent carcinogens called nitrosamines in smokeless tobacco hinges on a crucial step in the plant's growing and production process, namely curing. While the researchers detected, at most, trace amounts of nitrosamines in green tobacco plants, the compounds abound after curing. Moreover, they found that nitrosamine levels in five types of fire-cured black tobacco were nearly double those in air-cured samples. Nitrosamines, essentially the only known cancer-causing agents in smokeless tobacco, are formed when nitrites from nitrogen fertilizers react with the plants' nicotine alkaloids.

Chamberlain suggests that more air-cured tobaccos be used in smokeless tobacco blends, but he says he doubts that the industry will give up on fire-curing entirely since it is largely responsible for giving tobaccos their flavor. In a survey of products on the market, his group also found that snuff products have what he calls a high level of nitrosamines – 17 micrograms per gram of tobacco – but he notes that this is lower than nitrosamine levels of four or five years ago. "So obviously, the tobacco companies are already doing something to lower these levels," he says.