Cell-phone muffler squelches street noise

A technique that quiets noise by producing counteracting sounds may soon mute the background din that often plagues cellular-phone conversations.

Researchers at Lucent Technologies in Arlington, Va., reported last week that they have developed a modified cell-phone prototype that combats low-frequency, ambient noise, such as wind and traffic sounds. The antinoise circuitry can squelch sound at frequencies below roughly 1,200 hertz, reducing the noise to as little as one-eighth of its untreated loudness, says Michael A. Zuniga, leader of the development team.

Zuniga described the nascent technology last week at the 136th biannual meeting of the Acoustical Society of America in Norfolk, Va.

Cell-phone makers have expressed interest in conducting more extensive testing of the innovation, he says. The producers then might license the technology from Lucent, which no longer manufactures or sells telephones. The noise-fighting phones "could appear in Japan next year, depending on how things go this fall," Zuniga says.

"It is absolutely a very exciting application," says Irene Lebovics, president of NCT (Noise Cancellation Technologies) Hearing Products in Stamford, Conn. "Walking on the street or riding in a car, you're always subject to low-frequency noise. [It] has a masking effect on speech," she adds.

Zuniga says his team experimented with two approaches to the ambient-noise problem, both of which involve detecting noise with an extra microphone and generating sound waves that cancel it. One method, a feedback approach, reads noise signals directly at the ear. Since feedback can cause squeals, the researchers had to use less-than-optimal circuit settings to avoid them, yielding marginal noise reduction.

The other method, called feed-forward, positions the microphone about 2 centimeters from the ear, so that it detects ambient sounds before the ear does. The microphone converts the sound to electric signals, which are then sent to a programmable, digital microchip custom-made by Fujitsu Microelectronics in Dreieich-Buchschlag, Germany.

After analyzing the signals, the chip races noise-canceling waveforms to the phone's speaker, which converts them to sound just in time to counteract arriving noise. Because sound exists as compression waves in the air, opposite peaks and dips of compression from the speaker nullify target noise in the cavity between the phone and the ear, Zuniga explains.

The modifications also include internal changes to the earpiece so that the speaker produces effective compression patterns despite differences in ear size and shape, he says.

The performance of the feed-forward system was, "across the whole range of users, pleasantly, pleasantly surprising," Zuniga says. He and his colleagues both measured the noise reduction electronically and heard positive reviews from five test subjects who tried the prototype phone.

Using sound to cancel sound is not a new idea. For decades, acoustics designers have been developing products with what they call active noise reduction, such as headphones that protect the ears against loud noises or make it easier to hear an in-flight movie on an airplane.

Cellular phones, however, are a new frontier, says Graham Eatwell, a consultant and president of Adaptive Audio in Annapolis, Md. "As an active-control person, I think it's very interesting," he says of the Lucent design, which has several patents granted or pending.

In particular, Lucent's use of digital circuits might signify that such circuits are ready for use in other battery-operated, portable equipment, he adds. Designers of such products previously shunned digital circuits as too slow and power hungry. "One very big market is hearing aids," says Eatwell. "That's the one everyone has their eye on." —*P. Weiss*

Clues hint how particulates harm lungs

For more than a decade, epidemiologists have been homing in on the health risks posed by dustlike, inhalable pollutants known as airborne particulates. Though ample evidence suggests that particulates can aggravate existing respiratory disease or heart problems—and perhaps even cause cancer—scientists have been at a loss to explain how.

By eavesdropping on the chemical chatter within lung cells growing in test tubes, researchers have now identified a cascade of intracellular commands provoked by these pollutants. In the Oct. 15 CANCER RESEARCH, they report that the particulates trigger the cells to synthesize new DNA.

This might constitute a first step in preparing the cells to repair any damage, notes study leader Cynthia Timblin, a molecular biologist at the University of Vermont in Burlington. For example, they might need to reverse changes caused by oxidants created in reactions with metals on the particulates' surfaces. However, she adds, "it is interesting to note that the particular cascades initiated by these particulates have, in other cell types, led to certain types of proliferation," including cancer.

For 2 days, the scientists incubated epithelial cells from the interior surface of the lungs with a solution containing tiny particles, having an average diameter just under 40 nanometers. Some were particulates collected by state pollution-sampling stations, others were similarly fine spheres of titanium dioxide. The latter is a nontoxic industrial whitening agent.

Some researchers had speculated that ultrafine particulates might induce lung damage through some interaction fostered by their size, irrespective of their chemical makeup. If so, the titanium dioxide and particulates should induce similar changes in lung cells.

Indeed, both activated a gene called c-*jun*. Though it normally helps cells mature, c-*jun* can also act as a proto-oncogene, Timblin explains. If expressed at inappropriate times, proto-oncogenes can foster the development of tumor cells and cancer.

To trigger such abnormal cell proliferation, c-*jun* has to make a protein that serves as a building block of a larger cell-signaling protein, AP-1. By binding to specific portions of DNA, AP-1 turns genes on or off. Though cells incubated with the pollutant particulates showed AP-1 activity, those exposed to the titanium dioxide did not.

Probing further downstream in the cell-signaling cascade launched by c-*jun*, Timblin's team found that only the pollutant-exposed cells had synthesized new DNA, a necessary step if they were going to proliferate.

"As someone who's worked on c-*jun* and AP-1 for a long time, I'd be delighted if there were some rational data to link [them] to human cancer," says Michael J. Birrer of the National Cancer Institute in Rockville, Md. However, he told SCIENCE NEWS, "I don't think we have the data anywhere, including in this paper."

The problem, he explains, is that cells exposed to almost any stress can increase their c-*jun*–AP-1 activity. So while the new paper is interesting and suggestive, he says, it fails to demonstrate that the pollutants' activation of c-*jun*–AP-1 is what caused the DNA synthesis.

Timblin agrees, noting that her team plans to look for such a link. For now, the group is examining whether the increased DNA production triggered by the particulates leads to abnormal cell proliferation. It's also looking to see whether particulates activate cascades of genes leading to other toxic responses, such as inflammation. —*J. Raloff*