BIOLOGY

Julie Ann Miller reports from the meeting in Los Angeles of the Society for Neuroscience

Nerve growth factor's double influence

It's difficult to study something essential. Nerve cells need exposure to the material known as nerve growth factor if they are to survive. And no nerve cell is naive—all have experienced the factor before any experiment can begin. To sidestep these problems Lloyd A. Greene of New York University Medical Center uses a nerve cell imposter. Chromaffin cells of the adrenal medulla arise in development from the same embryonic tissue as nerve cells. Greene employs a line of laboratory-grown cells derived from a chromaffin cell tumor (pheochromocytoma). "This is the most popular nerve cell mimic," he says.

The cells, called PC12, are round when growing as tumor cells in culture. When they are exposed to nerve growth factor, they flatten and develop spikes. They grow long processes, called neurites, which resemble the dendrites of a nerve cell. If exposure to nerve growth factor is ended, the neurites fall off and the PC12 cells start reproducing as tumor cells again. The nerve growth factor effect does not seem to involve calcium or cyclic AMP, and it is not mimicked by other hormones.

Nerve growth factor appears to have a two-pronged influence on the PC12 cells. Greene finds that it acts both in the cell nucleus to moderate gene expression and more rapidly at the neurite endings. In the nucleus, nerve growth factor initiates the build-up of a pool of material required to generate neurites. After analyzing a thousand proteins of the cells before and after nerve growth factor exposure, Greene reports no qualitative change, but only increased production of a few minor proteins. One is a surface glycoprotein that is found on all nerve cells. Another is a phosphoprotein associated with microtubules in neurons. Greene speculates that nerve growth factor treatment makes the microtubules more stable, and thus aids neurite growth.

In addition to these rather slow changes dependent on gene expression, nerve growth factor triggers very rapid effects. Within minutes, projections extend from the cell, probing the environment and elongating. If the process is sliced from the cell body, nerve growth factor still starts process movement and growth. Greene believes this local effect is necessary both for its speed and for its asymmetry. If one process comes upon nerve growth factor, the growth of the cell's other processes is not necessarily increased. "We hope this model will eventually tell how nerve growth factor works and also answer questions of development, such as how nerve cells achieve such incredible asymmetry," Greene says.

Do-it-yourself giant cells

For some experiments it is not enough to have manipulable cells that mimic neurons. Paul H. O'Lague of the University of California at Los Angeles wanted cells large enough to penetrate with microelectrodes and to inject with solutions of macromolecules. So he used a chemical treatment to fuse groups of PC12 cells (see above). The groups form single cells, with up to 25 nuclei, and the diameter is proportional to the number of nuclei. O'Lague finds the large cells retain the desirable properties of PC12. They contain the same neurotransmitter, and they grow neurites in response to nerve growth factor. While normal PC12 cells extend only about three neurites per cell, a fused cell of 150 micron diameter can extend ten neurites. In both normal PC12 and fused cells, the membrane channels responsible for a nerve cell's characteristic action potential only appear after exposure to nerve growth factor, O'Lague says. In both cases the nerve growth factor has to work on the outside of the cell to elicit its responses. O'Lague expects the large fused cells to be useful to further characterize the nerve growth factor effects and to detect mechanisms underlying the growing neurite tips.

ENVIRONMENT

Janet Raloff reports from Amherst, Mass., at the International Indoor Air Pollution, Health and Energy Conservation symposium

Basement parking and high-rise CO

Field surveys of carbon monoxide (CO) levels in commercial California settings turned up one anomalous site. Persistently high indoor CO readings—equal to or greater than 9 parts per million in air—were recorded on most of the first 10 floors of a relatively new Palo Alto office building on four of the seven dates it was visited. The building's architecture allowed exhaust gases from the basement parking garage to infiltrate offices above it. "If one assumes these high CO levels persisted for as long as 8 hours on the four dates such levels were recorded, then it is very probable that the 8-hour [National Ambient Air Quality Standard] was violated on most floors of this building," the survey's investigators report.

Since there are no federal indoor air-pollution standards, however, the point is academic. But the finding worries the study's principal investigators—Wayne Ott of Stanford University and Peter Flachsbart of the University of Hawaii at Manoa—because workers received their occupational exposures unwittingly. And if the problem here is repeated in many of the nation's other high rises sitting atop parking garages—particularly residential apartment buildings—architects and builders could find themselves in court one day defending themselves against claims for chronic CO poisoning.

Other buildings in this survey were designed so that offices were largely shielded from any basement exhausts. But Ott told SCIENCE NEWS he would like to see a follow-up survey investigate whether the worrisome site in his survey represents a national anomaly or a sleeping urban hazard.

Building illness

A sharp rise in absenteeism plagued workers of a Vancouver, British Columbia, firm following its move to new quarters. Also accompanying the move was a rise in nonspecific health complaints, including eye irritation, headaches, nausea and drowsiness. Questioning what may lie behind it all, the tenants, mostly lawyers and secretaries, called in a Canadian father-and-son research team to investigate—Theodor Sterling, a biostatistician at Simon Fraser University in Burnaby, B.C., and Elia Sterling, an architect with T.D.S. Ltd. in Vancouver. Results of their surveys suggested "a slow buildup of a common eye irritant—photochemical smog—in the study building."

The tenants had moved from an older building with operable windows into a recently renovated one that was mechanically ventilated; its windows were permanently sealed. Occupants of an adjacent building of the same vintage, but "still in the original condition, with operable windows for ventilation and hot-water radiant heat," were selected for a control group. According to the Sterlings, "The study group reported a much higher rate of complaints of Building Illness symptoms than did the control. For example, the study group reported 60 percent more eye irritation and 20 percent more headaches."

Questionnaires were given to the study group twice weekly for several months as the building's ventilation was increased and its sunlight-simulating fluorescent lights were replaced with standard "cool white" bulbs. Changing only the lighting reduced complaints of too-bright lights by 19 percent and of glare by 23 percent. More interesting, when the lighting change was coupled with increased ventilation, an additional 8 percent found lighting more comfortable (no longer too bright) and an additional 5 percent reported reductions in glare. These changes also reduced many of the health complaints. The Sterlings conclude that without sufficient ventilation, photochemical alteration of indoor-air pollutants from ultraviolet light—such as that emitted in "significant" amounts by sunlight-simulating fluorescent bulbs—may generate irritating indoor smog.