Biomedicine

Here comes the sun . . . and wrinkles

Ever wonder why wrinkles slowly line your face while less exposed skin stays as smooth as a baby's bottom? Blame it on the sun, say dermatologists. Exposure to the sun's ultraviolet-B rays—the rays that give you a tan—breaks down collagen and elastin, structural proteins that hold skin cells together.

Exactly how this photoaging occurs has been a mystery until now. Investigators at the University of Michigan Medical School in Ann Arbor report in the Jan. 25 NATURE that just a few minutes' exposure to the sun's ultraviolet-B rays prompts skin cells to make metalloproteinases, enzymes that can chew up collagen and elastin.

John J. Voorhees and his colleagues made this observation by exposing skin samples taken from human buttocks. But dermatologist Barbara A. Gilchrest of the Boston University School of Medicine cautions that this finding may not hold true for more frequently exposed skin. "It's a very important and elegant study, but they only show the effects of a single exposure and in skin not previously exposed," she says.

Voorhees' group also reports that *trans*-retinoic acid, the main component of some commercially available acne and wrinkle treatments, helps prevent production of these degrading enzymes if it is slathered on the skin before exposure. Consequently, *trans*-retinoic acid may prevent photoaging, and perhaps even some forms of skin cancer, says Gilchrest.

Death trap lands an Alzheimer's gene

When you set a trap, you may snare a surprise. That's what Luciano D'Adamio and his colleagues at the National Institute of Allergy and Infectious Diseases in Bethesda, Md., recently discovered. The investigators devised a system to find genes involved in apoptosis, a process by which cells commit suicide.

This "death trap," as the researchers call it, caught two gene fragments out of a library of mouse genes. One fragment belongs to a gene whose protein binds calcium, an important signal molecule. The other belongs to a gene resembling a human gene that, when it goes awry, causes Alzheimer's disease, D'Adamio's group reports in the Jan. 26 SCIENCE.

The unusual catch hints that Alzheimer's results when brain cells undergo apoptosis because of a defective gene. The case is far from closed, however. "We want proof the full gene has something to do with cell death," says D'Adamio.

New weapons for tuberculosis war

Investigators are scrambling to defeat drug-resistant forms of *Mycobacterium tuberculosis*, the microbe that causes tuberculosis. Now, two groups report potential new strategies in the Jan. 23 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Investigators from the Whitehead Institute for Biomedical Research in Cambridge, Mass., say they have added new genes to the bacterium used in the BCG vaccine against tuberculosis. The genes produce cytokines, compounds that may boost the vaccine-generated immune response.

Researchers at the Worcester Foundation for Biomedical Research in Shrewsbury, Mass., announce they have learned how to prepare short DNA strands called antisense molecules so that an *M. tuberculosis*-like organism absorbs them. The strands can bind to and block the function of specific genes, thus killing the bacteria, even those resistant to traditional antibiotics.