Biomedicine

Fly control yields fewer trachoma cases

A concerted effort to kill off houseflies and their cousins, bazaar flies, in two West African villages has reduced the incidence of trachoma, a bacterial disease that causes blindness. The experiment indicates that these common flies carry trachoma and that controlling them can stifle the spread of the disease.

The scientists sprayed the insecticide deltamethrin in and around one village in Gambia every other day in the rainy season for 2 weeks, while leaving another village unsprayed. For the next 10 weeks, the researchers treated the test village twice a week. In the dry season, the scientists carried out a similar experiment on two other villages. The spraying induced no ill effects in the residents.

Researchers used traps to measure fly populations. In both seasons, the average number of flies tallied in the sprayed areas was less than half that in the unsprayed areas.

Cases of trachoma, caused by the bacterium *Chlamydia trachomatis*, declined in both treated villages but not in the untreated sites, the scientists report in the April 24 LANCET. In the wet season, only 11 of 295 villagers had trachoma after 3 months of spraying, compared with 37 of 271 in the untreated village. In the dry season, 19 of 189 people in the treated area had the disease, compared with 32 of 169 in the unsprayed village.

Flies were long suspected to carry trachoma bacteria from person to person, but until this study, "nobody had successfully demonstrated it," says coauthor Robin L. Bailey of the London School of Hygiene and Tropical Medicine. The researchers also demonstrated that captured flies harbor the microbe.

Doctors believe that flies spread the bacteria by alighting on fluid in and around the eyes, mainly of children. Mother-child proximity may therefore explain why more women than men get trachoma, Bailey says.

The disease is an inflammation that causes repeated scarring of the inside of the eyelid. The eyelashes turn inward, scratching the cornea over time. Repeat infections can leave the cornea opaque, causing blindness, usually in late middle age, Bailey says. Antibiotics can readily cure a case of trachoma, but people in areas with little access to clean water face a lifetime of reinfection. A World Health Organization campaign aims at eradicating the disease by promoting sanitation, face washing, antibiotic dosing, and eyelid surgery, when needed. —*N.S.*

Might night-lights blight sight?

For now, shut off the lights in the nursery.

That's the advice of researchers who are puzzling over a study in which they find that babies exposed to light at night grew up to have much more myopia, or nearsightedness, than did babies who slept in the dark.

Of 172 children whose parents reported putting them to bed in the dark as infants, only 10 percent showed nearsightedness when examined between ages 2 and 16 at a Philadelphia clinic. Of 232 children who had slept with a night-light until they were at least 2 years old, the number jumped to 34 percent. Moreover, 55 percent of 75 babies who slept in full room light were nearsighted later.

Earlier studies in chicks also found a link between night-lights and extra growth of the eye, which causes myopia, says study coauthor Maureen G. Maguire, a biostatistician at the University of Pennsylvania School of Medicine in Philadelphia.

She notes that the new data, reported in the May 13 NATURE, suggest an association but not necessarily a causal relationship between lights and nearsightedness. Other studies have linked myopia to heredity and "near work," such as reading.

"There's definitely a genetic component, and there seems to be an environmental component," Maguire says. The researchers are again contacting parents of some of the children to look for hereditary factors that may have biased the study. —*N.S.*

Thwarting killer enzymes of the brain

Caspases are cellular executioners. Activated by damaged cells trying to commit suicide, these enzymes facilitate death by breaking up a variety of proteins. Two recent studies suggest that caspases play a crucial role in the death of brain cells observed in Huntington's disease. One study even shows that inhibiting caspases can slow the development of symptoms and delay death in mice that have an illness that mimics the human neurodegenerative disorder.

In the March NEURON, Junying Yuan of Harvard Medical School in Boston and her colleagues report that certain features of the mutant proteins made in Huntington's disease, so-called polyglutamine repeats, help activate one of the many caspases found in mammalian cells. Rat brain cells containing proteins with such repeats ultimately commit suicide.

The repeats, consisting of multiple copies of the amino acid glutamine, convert caspase-8 into an active form, according to test tube studies performed by the researchers. A caspase inhibitor added to brain cells containing these repeats prevented the cells from dying, says Yuan. Her group has also shown that the affected brain regions of people with Huntington's disease do indeed contain activated caspase-8.

A second study, in the May 20 NATURE, highlights a different caspase. Robert M. Friedlander of Brigham and Women's Hospital in Boston and his colleagues worked with mice harboring a gene for an inhibitor of caspase-1. They bred these animals with mice having part of the mutant human gene that causes Huntington's disease. The latter mice usually develop disease symptoms, such as movement disorders and loss of brain and body weight, within 2 months of birth. The crossbred mice didn't develop such problems until several weeks later.

Friedlander's group also injected a caspase-1 inhibitor into the brains of the Huntington's-disease mice. They developed symptoms more slowly and survived longer than untreated mice did.

"This is the first drug that can slow disease progression in a real mouse model of Huntington's," says Christopher A. Ross, who studies Huntington's disease at the John Hopkins Medical Institutions in Baltimore. If the drug similarly helps people with the disease, they might live 30 years after symptoms emerge rather than the usual 15 to 20, Friedlander calculates.

The caspase-1 inhibitor used by Friedlander's team can act on other caspases, which may link the two groups' efforts. "The two papers together are quite interesting. They don't exactly add up to a clear story, but they do say that caspases are involved in more ways than we appreciated," comments Ross.

Pharmaceutical companies are now racing to develop safer, more potent caspase inhibitors. The caspase-1 inhibitor employed in the mouse study has caused severe heart problems when tested on dogs for another illness.

Caspase inhibitors may also tackle conditions ranging from strokes to Alzheimer's disease. "The lessons we learn in Huntington's disease, of how to slow down the caspase pathway, can also be used for other diseases," says Friedlander. —*J.T.*

Diet of TACOs keeps bacteria alive

Immune cells called macrophages devour microorganisms, storing them temporarily in sacs known as phagosomes. The phagosomes then deliver trapped bacteria to their death by fusing with lysosomes, other sacs containing enzymes that tear apart microbes. Yet mycobacteria, which include the bacteria that cause tuberculosis, happily live in phagosomes.

An explanation for this turn of events lies in a cellular protein called tryptophane aspartate–containing coat protein, or TACO. A research team led by Giorgio Ferrari of the Basel Institute for Immunology in Switzerland reports in the May 14 CELL that mycobacteria recruit this protein to coat the phagosomes that they're inside, which prevents fusion with lysosomes. —*J.T.*