Soft contacts: Extended wear poses hazard

People who wear soft contact lenses while they sleep face an increased risk of ulcerative keratitis, an eye infection that can lead to permanent vision loss, according to a pair of scientific reports. The risk appears to mount with each consecutive day of uninterrupted contact wear.

In a study involving 557 people, Oliver D. Schein of the Harvard-affiliated Massachusetts Eye and Ear Infirmary in Boston found that people who used extended-wear contact lenses overnight ran a risk of ulcerative keratitis 10 to 15 times greater than people who used the daily-wear type of lens during the day only.

According to the Food and Drug Administration, 4.1 million people in the United States use extended-wear soft contacts and another 9.1 million use daily-wear soft contacts. Schein and his colleagues found that 11 percent of the daily-wear users disregarded FDA guidelines to remove such contacts nightly. Compared with people who used the same contacts correctly, these users ran nine times the risk of ulcerative keratitis.

People practicing poor lens hygiene showed an increased risk of the eye infection, but frequent cleaning alone did not remove the risk of getting ulcerative keratitis. Even people with good hygiene habits who wore lenses overnight faced a threat of ulcerative keratitis, the researchers found.

Scientists speculate that contacts cause ulcerative keratitis by cutting off the oxygen supply to epithelial cells on the eye surface. When the cells die, they form an ulcer that can become infected if bacteria or other microorganisms gain a foothold. Doctors treat ulcerative keratitis with eyedrop antibiotics, but even successful treatment can leave patients with vision loss if scar tissue forms in front of the pupil, Schein says.

In a second report – a survey of 4,178 New England households and of all practicing ophthalmologists in the study area – Schein, Eugene C. Poggio of Abt Associates in Cambridge, Mass., and their colleagues estimate that one of every 500 extended-wear users each year develops ulcerative keratitis. Their risk estimate for daily-wear users is less: About one of every 2,500 users each year will develop the infection. Both reports appear in the Sept. 21 NEW ENGLAND JOURNAL OF MEDICINE.

Last May, after reviewing prepublication data from both studies, FDA officials asked manufacturers of soft contact lenses to revise their labeling instructions, advising consumers to remove extended-wear lenses for cleaning after seven days of continuous use.

Prior to the May announcement, FDA labeling instructions allowed people to keep such lenses in place for up to 30 days, a practice that "presents too high a risk" of ulcerative keratitis, according to an FDA statement issued in May. "The [Boston] data did not identify a wearing time that will eliminate the risk altogether, but seven days represents a relatively short, easy-to-remember interval which will encourage users to remove their lenses and clean them," FDA says.

Some eye specialists believe FDA should tighten its recommendation further. "It's not at all clear to me why seven days became a magic number," says Alfred Sommer, an ophthalmologist at the Wilmer Institute of Johns Hopkins Hospital in Baltimore. "It's six days too long." Sommer says people should wear contacts overnight only rarely. Ophthalmologist Ronald E. Smith of the University of Southern California in Los Angeles agrees. In an editorial accompanying the two research reports, Smith writes: "Since wearing a soft contact lens continuously for even a week substantially increases the risk of ulcerative keratitis, and since patients are likely to push beyond any suggested guidelines, there is concern that this change does not go far enough in warning the patients of the risk." *– K.A. Fackelmann*

Alzheimer's protein not restricted to brain

A protein that accumulates in the brains of Alzheimer's patients also resides elsewhere in their bodies, new research indicates. Scientists say the unprecedented finding of beta amyloid protein in non-neural tissue should significantly aid their efforts to understand the disease's underlying biology and may improve their ability to develop and assess experimental treatments. Until now, such research has largely been restricted to postmortem examinations of patients' brains.

"This is a development that puts a new perspective on how we think about Alzheimer's disease," says Dennis J. Selkoe, who performed the research with colleagues at the Brigham and Women's Hospital in Boston. "Since 1906, when Alzheimer first described it, the disease was thought to be a brain-only process. Now we're saying there's a clear signature of this process in other parts of the body."

Beta amyloid is the major ingredient of protein deposits common in the brains of elderly people but abnormally abundant in the brains of Alzheimer's patients. Selkoe, Catherine L. Joachim and Hiroshi Mori produced antibodies to beta amyloid, then used the antibodies as bloodhounds to sniff out and bind to similar protein deposits in biopsy specimens taken from various organs in both Alzheimer's patients and normal elderly people. They found evidence of the protein in skin, blood vessels or intestinal tissues in eight of 10 Alzheimer's patients. Only four of 24 normal patients tested positive for the protein; all four were more than 77 years old.

Scientists, not patients, will reap the finding's first benefits. Long hampered by the difficulties inherent in any research restricted to the brain, they now have a chance to track the natural history of amyloid accumulation in the body with relatively noninvasive biopsy techniques. Moreover, the test may allow researchers to identify Alzheimer's patients more accurately. Such an ability would aid genetic linkage studies and might provide more uniform populations in which to test experimental drugs. "If 20 percent of your test subjects don't really have Alzheimer's, your drug study is not going to be very useful," Selkoe says.

In the long run, therapeutic implications may follow, the researchers conclude in the Sept. 21 NATURE. They say their findings suggest beta amyloid may be produced in non-neural tissue somewhere in the body, then get transported via the circulatory system to the brain, where the protein does its damage. Such a mechanism would open the possibility of using a drug either to mop up the circulating protein or to keep it from crossing the blood-brain barrier.

– R. Weiss

Gene tests: So far, so good

The first U.S.-approved infusions of genetically engineered cells into humans have produced no significant ill effects and have begun to provide useful data, researchers told a National Cancer Institute (NCI) advisory board this week. To date, five terminally ill cancer patients have received the one-time doses of about 100 million gene-altered, tumor-fighting lymphocytes.

Scientists had initially obtained lymphocytes from patients' tumors, then inserted a bacterial "marker" gene into each cell in order to track the cells' survival and distribution in the body after reinfusion (SN: 5/27/89, p.324). NCI researcher Steven A. Rosenberg says tests so far indicate the cells begin to concentrate in tumors the fifth day and live and circulate in the body for at least 19 days.

Ultimately, the researchers hope to gain government permission to give patients cells engineered to secrete naturally occurring tumor-fighting compounds. At the meeting, Rosenberg revealed that he and colleagues have recently succeeded in engineering human lymphocytes to produce one such compound, tumor necrosis factor.

– R. Weiss