Schizophrenia caregivers take health hit

Although people suffering from long-term, incapacitating psychiatric disorders were once consigned to mental institutions and hospital back wards, as many as 2 out of 3 now live with family members. People caring for a mentally ill relative not only face a draining and seemingly endless task but may in certain cases catch far more than their fair share of colds and other infectious illnesses, a new study suggests.

The frequent appearance of hallucinations, delusions, and other so-called positive symptoms in people diagnosed with schizophrenia accompanies high numbers of infectious ailments in their caregivers, asserts a team led by psychologist Dennis G. Dyck of Washington State University in Spokane.

Symptoms of schizophrenia that are classified as negative, such as persistent apathy and social withdrawal, exhibit no link to caregivers' infectious ills, the scientists report in the July/August PSYCHOSOMATIC MEDICINE.

These long-lasting, negative symptoms did, however, create a greater burden for caregivers than intermittent positive symptoms, the researchers contend. In the study, the team measured burden by evaluating care-related money woes, worry about one's afflicted relative, self-blame for the situation, and stigma attached to having a mentally ill family member.

The new evidence "contributes to a growing body of literature suggesting that . . . caregiving may be not only burdensome but actually hazardous to the caregiver's health," comments psychiatrist Igor Grant of the University of California, San Diego in the same journal issue.

Other researchers have found that people who care for spouses with Alzheimer's disease report high levels of burden and depression and also exhibit signs of weakened immune function (SN: 4/6/91, p. 217). Unlike Alzheimer's disease, however, schizophrenia typically strikes young adults who then require decades of care from relatively young parents or siblings.

The mental and physiological consequences of this type of "very-long-term stress" for caregivers have attracted little scientific attention, Grant says.

In the new work, a nurse interviewed 70 people who cared for relatives with schizophrenia—mainly mothers tending adult children—about their own physical symptoms and physician visits in the past 6 months. Caregivers averaged 52 years old, and their charges, 33 years.

A second interviewer then assessed each caregiver's perceived burden, depression, anger, support from friends and family, and style of coping with stress. Coping styles included wishful thinking (such as hoping for a miracle), focusing on problems (making and following a plan of action), avoidance (trying to forget about the problem), relying on religious faith (such as frequent prayer), and self-blame for the relative's illness.

Several independent raters also estimated the extent of the burden that each patient placed on his or her caregiver.

Caregivers who engaged in wishful thinking, avoidance, and self-blame tended to find their situations extremely burdensome. Surprisingly, caregivers who formulated specific plans also reported a comparably high level of burden.

Although not citing especially high or low burden, those who relied on religious faith or were satisfied with the support received from friends and family cited the lowest rates of infectious illness, the researchers hold.

Caregivers with excessive burdens didn't necessarily report an elevated rate of illness. Burdens imposed on caregivers apparently do not lay the groundwork for infectious illness, Dyck's group theorizes. Instead, specific aspects of the condition under care—such as positive symptoms in schizophrenia—and a caregiver's coping style influence the immune system's ability to fend off infections, in their view.

This health-undermining process may hinge on caregivers' behavioral responses, such as eating poorly and exercising little, or on emotional distress that sparks a cascade of hormonal and immune changes, Grant says. *—B. Bower*

Synthetic drug slows glaucoma in rats

In glaucoma, pressure buildup in the eyeball can lead to blindness by disabling the nerves in the back of the eye that send images to the brain. The pressure causes excessive production of nitric oxide, which can assume a toxic form that kills the retinal cells that carry information through the optic nerve.

By neutralizing an enzyme known to spur nitric oxide production, researchers studying rats at Washington University in St. Louis are now able to hold such nerve damage at bay—even if pressure in the eye remains high.

Eye pressure rises when normal fluid discharge from the eye becomes blocked. Pressure-reducing medication such as beta-blockers can ease this condition in many glaucoma patients, but some forms of the disease resist these drugs and continue to destroy nerve cells, which don't regenerate.

The scientists cauterized blood vessels to induce high pressure in one eye of each of 16 rats. Half the rats then received aminoguanidine in their drinking water. This synthetic drug inactivates nitric oxide synthase-2, or NOS-2, an enzyme that responds to the tissue damage by launching—for reasons that are still unknown—a flurry of nitric oxide production.

Over 6 months, nerve cells called ganglion cells remained constant in each rat's healthy eye. The unmedicated rats, however, lost an average of 36 percent of the ganglion cells in the retinas of the eyes with glaucoma. Those getting aminoguanidine did not lose significant numbers of these cells, says study coauthor Arthur H. Neufeld, an ophthalmic pharmacologist.

Abnormally high pressure persisted in the cauterized eyes of both groups of rats. "We showed that despite the pressure, we were still able to protect the ocular ganglion cells," says Neufeld. The findings appear in the Aug. 17 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Monthly examinations of the rats revealed cupping of the optic disk at the back of the untreated eyes with glaucoma. Such malformation serves as a sign of glaucoma in people.

Since a rat's life span is only a few years, the 6 months needed for significant nerve damage to accumulate parallels the period of years over which the common chronic form of glaucoma develops in people, Neufeld says.

In this study, the scientists were able "to find a way to really protect the optic nerve, not just lower eye pressure," says Paul L. Kaufman, an ophthalmologist at the University of Wisconsin-Madison. Because this study opens the door to treatments aimed at inhibiting the NOS-2 enzyme, "it will likely be considered a classic in years to come," he says.

Excess nitric oxide begets a toxic substance called peroxynitrite. Neufeld hypothesizes that curbing nitric oxide production limits peroxynitrite concentrations. The compound is thought to degrade nerve cells and possibly induce apoptosis, or programmed cell death.

Whatever the precise mechanism behind the nerve damage, the study shows that aminoguanidine protects against it in the rat model, Neufeld says. Aminoguanidine was originally developed and tested, without much success, as a treatment for diabetes. Its prospects as a glaucoma drug seem brighter.

While lowering eye pressure eases glaucoma in many cases, too much pressure reduction can do damage. As a result, the enzyme-blocking approach for glaucoma "is very much welcomed," says Carl Kupfer, director of the National Eye Institute in Bethesda, Md. *—N. Seppa*