More evidence ties smoke to artery disease

Two new reports provide additional evidence of the cardiovascular risks faced by cigarette smokers and by their nonsmoking families, friends and colleagues exposed to secondhand smoke.

Regardless of the number of cigarettes smoked per day, lifelong smokers face a greater risk of clogged carotid arteries — vessels in the neck that carry blood to the brain — than people who quit smoking earlier in life and thus limit the duration of their smoke exposure, report researchers at the Mayo Clinic in Rochester, Minn. They say their study is the first to suggest that the number of years a person smokes cigarettes provides the most important predictor of carotid artery disease. If a blood clot blocks a carotid vessel already narrowed with fatty plaque, a debilitating or life-threatening stroke can result.

"The information about smoking that is most critical is how long a person has smoked," says study leader Jack P. Whisnant. Previous research by others focused on the number of cigarettes smoked but did not isolate duration of smoking as a separate risk factor for atherosclerosis, he says.

The Mayo team studied 752 men and women who underwent arteriography, in which physicians inject dye into the bloodstream and then take X-ray pictures of the arteries. They asked the volunteers about their smoking history, including the duration of the habit and the number of cigarettes smoked per day.

In the May STROKE, they report that 60-year-olds who had smoked for 40 years were 3.5 times more likely to show severe carotid artery disease — in which 90 percent of the vessel is clogged — than people of the same age who had never smoked. The Mayo researchers also found that 60-year-olds who reported smoking for 20 years were nearly twice as likely as lifelong nonsmokers to develop carotid artery disease.

A number of studies have linked cigarette smoking in men to the buildup of plaque in heart arteries, which can lead to heart attacks. Several years ago, researchers at Harvard University provided evidence that smoking posed a similar threat to women (SN: 11/28/87, p.341). Their study indicated that smokers who quit reap immediate benefits to their coronary arteries.

In carotid arteries, however, damage lingers long after smoking stops, the Mayo results suggest. People who stop puffing on cigarettes can slow the rate of new plaque buildup in these arteries but cannot reverse preexisting damage, Whisnant says.

Underscoring the dangers of inhaling others' fumes, an as-yet-unpublished review of the scientific literature shows that passive smoke exposure can cause nonsmokers a number of the short- and long-term cardiovascular problems linked to smoking. Researchers described the review in Boston this week at the World Conference on Lung Health, sponsored by the American Lung Association and several other medical groups.

Stanton A. Glantz of the University of California, San Francisco, told conference participants that when nonsmokers breathe air laced with cigarette smoke, even for short periods of time, their blood platelets get sticky which can help form clots. For nonsmokers with coronary artery disease, that scenario may lead to a heart attack if a clot disrupts the heart's blood supply, Glantz notes.

In addition, Glantz says chemicals in cigarette smoke can injure the endothelium lining the coronary arteries — the first step in the atherosclerotic process — both in smokers and in nonsmokers who inhale others' smoke. "Passive smoking causes heart disease in otherwise healthy nonsmokers," he says. While the scientific literature doesn't reveal whether secondhand smoke exposure raises the risk of carotid artery disease and subsequent stroke, Glantz predicts that further research will establish that link. — *K.A. Fackelmann*

Herpes may disarm immune system

Recurrent genital sores and fever blisters on the mouth provide two of the more painful reminders that infection with herpes simplex virus (HSV) persists for life. Although researchers know HSV's two strains can lie dormant in several tissues, the question remains: Why doesn't the immune system eliminate them?

New *in vitro* findings suggest an answer. Researchers at the University of Minnesota in Minneapolis report that prolonged contact with HSV-infected cells disarms key components of the immune system, preventing them from attacking the virus or several other infectious agents they would normally destroy. The laboratory work not only offers an explanation for HSV's persistence, but also suggests the virus may help other pathogens escape immune obliteration.

Dennis L. Confer, Harry S. Jacob and their colleagues began their study after noting a puzzling phenomenon among kidney transplant patients experimentally given a protective dose of the anti-HSV drug acyclovir. (Organ transplants carry a risk of transmitting the virus.) As expected, these patients showed a lower incidence of HSV infection than did transplant patients not receiving acyclovir. But the drug had another payoff: Treated patients also had lower rates of cytomegalovirus (CMV) infection, even though laboratory tests suggest that acyclovir cannot block CMV replication at the low doses used in the study.

The observation prompted Confer's group to investigate how the two HSV strains — HSV-I, which causes oral herpes, and HSV-II, which causes genital herpes — might promote other infections. They covered HSV-infected cells removed from human foreskin and blood vessel walls with a layer of one of two types of immune-system cells, natural killer (NK) cells or interleukin-2-activated killer (LAK) cells. After eight hours of direct contact with the infected cells in culture plates, the immune cells lost their ability to attack the virus. Moreover, they could no longer destroy two laboratory lines of human leukemia cells, the researchers say.

"Rather than a 'kiss of death,' contact with HSV-infected cells gives NK and LAK cells a 'kiss of paralysis,'" says Jacob. He suggests the finding may explain how acyclovir might reduce the success rate of other infectious agents. In the May PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (Vol.87, No.9), the Minnesota researchers report that several of their tests strongly indicate the immune cells themselves were not infected. Instead, they propose that an HSV glycoprotein on the surface of the infected cells eventually disarms LAK and NK cells. The team found that when they chemically prevented HSV glycoproteins from forming, infected cells no longer inhibited immune-cell function. Other researchers have reported that an AIDS virus glycoprotein similarly disables NK cells.

Immunologist Ronald B. Herberman, who directs the University of Pittsburgh Cancer Institute, cautions that the laboratory work will remain inconclusive until researchers can establish a firm link between HSV and immunologic impairment in humans. Confer suggests looking for impaired LAK and NK cells in blood from HSV patients who harbor the virus in the cells lining their vessel walls.

HSV may promote cancerous changes independent of its effect on the immune system. For two decades, researchers have compiled strong evidence linking cervical cancer to both HSV-II and human papillomavirus, another sexually transmitted agent. Joseph A. DiPaolo of the National Cancer Institute told SCIENCE NEWS he and his colleagues recently found that a laboratory cell line of papilloma-infected genital epithelial cells changed into carcinoma cells when exposed to HSV-II. DiPaolo says the transformation, which his team will detail in a forthcoming VIROLOGY, occurred in the absence of immune cells, thus adding another dimension to the virus' potential for assisting other pathogens. — *R. Cowen*