

## Cold cure, prevention: Nothing to sneeze at

A violent sneeze scatters some 20,000 water droplets through the air. If the droplets carry rhinoviruses causing the common cold, someone nearby may join the millions who "catch cold" each year in the United States, then spend billions of dollars on cold remedies that may or may not work. Two recent studies — on a protein linked to cold symptoms, and on a potential vaccine — show that, while results may lead to new cold therapies, it is far too soon to toss out the chicken soup.

Stymied by the dozens of rhinovirus types responsible for these respiratory infections, scientists have not found the definitive cold cure or a protective vaccine. One promising approach is to find some aspect that all or most of the rhinoviruses have in common, either in their structure or in how they affect humans, the only natural hosts of the cold viruses.

In the recent work, scientists at the University of Virginia in Charlottesville and Johns Hopkins University in Baltimore report that, regardless of the rhinovirus involved, proteins called kinins appear to be the primary cause of painful nasal congestion. Released from plasma proteins activated by enzymes, kinins in the blood cause expansion of blood vessels, which allows fluid to leak into surrounding tissue. This fluid accumulation and its pressure on nerve endings is responsible for characteristic cold symptoms. Although these effects were already known, the researchers were surprised by the close relationship between kinin levels and symptoms.

The study, to be published in the January *JOURNAL OF INFECTIOUS DISEASES*, shows that kinin levels increase as cold symptoms appear and then decrease as symptoms fade. "The striking thing was that the kinins reached a peak on day two or three [after infection], which coincided with the peak of symptoms," J. Owen Hendley of Virginia told *SCIENCE NEWS*.

Using nasal discharges from 40 volunteers infected with rhinoviruses, the researchers found that kinin levels increased 20- to 80-fold in the two-thirds who developed symptoms. What the scientists did not find, Hendley says, is a similar rise in the level of histamines, proteins released from tissue cells during allergic attacks that also dilate blood vessels. "Antihistamines have been used in common cold remedies for years," says Hendley. "But the evidence from studies in the past 10 years saying that they work [for colds] is marginal at best."

"Antikinins," however, may be more promising, says Hendley. Because there is a time gap between infection of nasal passages and the influx of kinins into tissues, he suspects that the viruses may be activating kinin production. Drugs to

stop that process, he says, could be the cold cures of the future. Nova Pharmaceutical Corp. of Baltimore announced this week that it has received the first U.S. patent ever issued for a drug to block the release of kinins from their plasma precursors.

Because prevention is so much more desirable than after-the-infection cures, other scientists are working on vaccines for the common cold. In the Oct. 22 *NATURE*, Joseph McCray and Gudrun Werner at Sandoz Forschungsinstitut in Vienna, Austria, report success with a vaccine preparation against amino acids lining a groove on the surface of rhinoviruses. This "canyon" — which apparently plays a role in attachment of viruses to host cells — contains certain amino acid sequences common among rhinoviruses and their relatives, the polio and foot-and-mouth disease viruses.

After making synthetic peptide structures that included these amino acid sequences, McCray and Werner injected them into rabbits. Antibodies produced

by the rabbits against the peptides inactivated about 60 percent of the 48 types of rhinoviruses tested *in vitro*. These results imply that the vaccine can stop human infection by these viruses, say the scientists. By including more peptides in the injected solution, they say, it may be possible to immunize against all rhinovirus types. Without a nonhuman model for the common cold, however, they agree that testing such a vaccine will be "difficult."

Not only would testing the vaccine be difficult, says Ann Palmenberg of the University of Wisconsin in Madison, but it may be unwarranted. The vaccine approach taken by the Austrian group "is just not feasible," she told *SCIENCE NEWS*. In a commentary accompanying the McCray-Werner report, Palmenberg says the viral groove is too narrow to allow strong binding by antipeptide antibodies. Any neutralization of viruses, therefore, would be too weak for real protection against infection, she says. Palmenberg and colleague Michael G. Rossmann discovered the surface cleft about two years ago while studying the structures of viruses. — *D.D. Edwards*

## Chemical power for visible-light lasers

Energy generated directly by chemical reactions may soon power lasers capable of producing intense visible light. The recent development of the first visible-light chemical amplifiers marks the end of a two-decade search by scientists throughout the world for such systems. It also opens up the possibility of using lasers in remote locations such as space stations or satellites.

Conventional lasers rely on strong bursts of electricity to excite materials into emitting coherent light. Notoriously inefficient, such lasers require large quantities of electrical power to operate. Chemically powered lasers promise a more efficient way to generate intense light.

"It was a long road," says physicist James L. Gole of the Georgia Institute of Technology in Atlanta, who led the research. "What we have done so far is to demonstrate that these lasers amplify radiation." The next step, he says, is to build an oscillator consisting of a mirrored cavity that bounces light back and forth through the reaction zone.

"It's a significant development," says Terry A. Cool of Cornell University in Ithaca, N.Y., who years earlier had developed the first chemically powered infrared laser. Until Gole's persistence paid off, he says, scientists, despite trying many different approaches, had a long record of failure in their search for chemical reactions leading to molecules in excited states that would per-

mit light amplification.

In Gole's pulsed amplifier, thallium atoms pick up energy from collisions with excited molecules formed by a reaction between ozone and silicon or germanium. The collisions pump the thallium atoms up to an excited state. The atoms can then be stimulated to emit their radiation in a coordinated fashion to produce light amplification. The emitted light is green. When developed, says Gole, the system could turn out to be so powerful that it may be difficult to control.

Gole and his team have also developed a less powerful, continuous light amplifier based on the reaction between three-atom sodium clusters and halogen atoms. When a halogen such as chlorine reacts with a three-atom sodium cluster, the result is the formation of sodium chloride and an excited two-atom sodium cluster. The new cluster emits light, dropping to a lower energy level, and immediately reacts with any excess chlorine. Because this reaction is so rapid, the population of excited sodium clusters is always higher than that of low-energy clusters, allowing the laser to operate continuously.

Potential applications of chemically powered lasers depend not only on the possibility of achieving high powers but also on the fact that visible light requires simpler optics and is easier to focus than infrared radiation. Visible-light lasers are also likely to be quite compact.

— *I. Peterson*