Suppressing suppression

The immune system somehow manages to turn itself off after fighting an infection. Research by David S. Strayer of the University of Texas in Houston indicates that viruses use the same "off" mechanism to dodge the immune system, and that the immune system in turn can eventually counter the ploy.

Strayer's work, presented at the Sixth International Congress of Immunology earlier this month in Toronto, fits into an idea gaining a foothold in immunology – that the immune system actively inhibits its own suppressive response, turning itself back on after it has turned itself off. The process is known as contrasuppression. Proponents of the theory believe overambitious contrasuppression causes the immune system to attack itself in autoimmune diseases.

Strayer infected rabbits with a virus and looked at their immune-cell-harboring spleens. In the test tube, spleen cells collected 7 days after infection were capable of only a low response to immune stimulators, but spleen cells collected 11 days after infection were better able to respond. The liquid portion of the 7-day cell culture, but not from the 11-day cells, contained a substance that suppressed other immune cells.

The virus, Strayer suggests, activates the natural suppression of the immune system, causing the 7-day cells to produce and secrete a substance that suppresses the immune response. By day 11, the rabbits' immune systems were suppressing the virus-induced suppression.

Another target for interferon?

Preliminary experiments with gamma interferon indicate it may be useful in treating lepromatous leprosy, a form of Hansen's disease. While drugs that kill the leprosy bacterium are already available, drug-resistant strains have recently appeared.

There are two major forms of the disease, lepromatous leprosy and tuberculoid leprosy. In tuberculoid leprosy, the bacteria infect the body and the body mounts an immune response. In lepromatous leprosy, the same bacteria are present but the body fails to recognize them and mount a response. The tuberculoid form is generally milder, and only a couple of years of treatment are needed. Treatment for lepromatous leprosy can last a decade or more, or for life.

Carl F. Nathan of Rockefeller University in New York City and researchers from several other institutions injected gamma interferon into leprosy skin sores of six patients. The interferon resulted in a local immune response against the bacteria where there had been none, they report in the July 3 NEW ENGLAND JOURNAL OF MEDICINE. "It looked as if we had converted that limited area from lepromatous to tuberculoid leprosy," Nathan says.

Because tuberculoid leprosy is easier to treat, the researchers suggest that gamma interferon could be useful therapeutically.

Stop that itch

It starts out innocently enough – a walk through the woods, a little contact with nature. But then the chemical reactions occur in people sensitive to poison ivy and poison oak, and the incessant itching begins.

Susan Orchard and her colleagues at the Oregon Health Sciences University in Portland have found that several chemicals added to oils to reduce the incidence of allergic reactions in machinists can also prevent reactions to poison ivy and poison oak. In the July ARCHIVES OF DERMATOLOGY, they report that when the substances were applied to the skin of 48 people before contact with poison ivy extract, 34 of them were protected. If the formulations can be made less "tacky and unpleasant," they say, they may prove useful.

Botulism: New drug buys time

Scientists have found that a little-used drug may prove useful in emergency treatment of the most potent of four human-botulism toxins – type A. The treatment has been identified by researchers with the Army Medical Research Institute of Infectious Diseases at Ft. Detrick in Frederick, Md. This drug, a variant of one used in treating myasthenia gravis, enhances the release of the neurotransmitter acetylcholine. Botulism toxins interfere with acetylcholine release.

In studies involving mice given lethal doses of this bacterial toxin, hourly treatment with 3,4-diaminopyridine (DAP) prolonged survival – sometimes almost doubling or tripling the survival period compared with that of poisoned animals receiving no drug, according to a report in the June 30 TOXICOLOGY AND APPLIED PHARMACOLOGY. However, 3,4-DAP is not a cure, stresses Lynn S. Siegel, one of the researchers. Though it slowed the progressive paralysis that characterizes the disease, treated animals ultimately died of toxin-induced respiratory failure, generally within 15 hours. As a result, she says, 3,4-DAP is best viewed as a way of buying time to get a patient access to preferred therapy – such as treatment with botulism antibodies or respiratory intensive care.

At least as important as finding this potential temporary treatment, says Siegel, is what the research suggests about the toxins. *Clostridium botulina* have been classified into seven types, based on the particular neurotoxin – types A through G – that each makes. "The dogma for people involved in botulism research has been that all seven types of toxin act by the same pharmacological mechanism," Siegel says. However, the finding that 3,4-DAP works against only one of the toxins affecting humans "indicates that the toxins are indeed different and have a different mechanism of action."

A preferred starch for diabetics?

Scientists had long thought that the body digested all starches identically and at a similar pace. Moreover, they had assumed that because starches are chains of many sugars, they would take longer to break down into glucose (blood sugar) than would simple sugars like sucrose (table sugar). But intrigued by a published report indicating that some starches break down and enter the blood as quickly as do simple sugars, research nutritionist Kay Behall and her colleagues decided to see if they could identify which were the quick-digesting food starches. Their finding may one day benefit diabetics attempting to control blood sugar levels through diet.

Food starches come in two forms: amalose and amalopectin. Most foods contain both. Behall, at the Agriculture Department's Human Nutrition Research Center in Beltsville, Md., fed breakfasts of predominantly amalose-based crackers, predominantly amalopectin-based crackers or sucrose patties to 25 healthy, nondiabetic adults who had fasted since the night before During succeeding weeks she offered each subject another of these breakfasts until all had eaten each food once. Blood levels of glucose and insulin were recorded before each meal and at intervals afterward.

Though all diners started their meals with comparable blood levels of insulin and glucose, within 30 minutes of eating, the amalopectin and sucrose eaters had elevated levels of both relative to the amalose eaters: Their blood sugar was an average of 15 percent higher than the amalose diners', and their insulin levels an average of 40 percent higher. Over the next 2.5 hours, blood levels of both glucose and insulin in all three groups gradually returned to about the fasting level.

Amalose, concludes Behall, not only lowered peak blood sugar levels but also slowed sugar delivery into the blood – a potential benefit to diabetics. Moreover, she notes, amalose allowed the body to rid the blood of sugar using less insulin.

Science Service, Inc. is collaborating with JSTOR to digitize, preserve, and extend access to *Science News*. www.jstor.org