Chemistry

Janet Raloff reports from Toronto at the Third Chemical Congress of North America

Antinutrients may need a new name

Certain chemically active, plant-derived food ingredients — such as enzyme inhibitors, saponins, lectins and phytic acid — have come to be known as antinutrients. At high concentrations, some are toxic. Others bind nutrients, preventing their digestion and use by the body. The value of antinutrients has been so questionable, says University of Toronto nutrition scientist A. Venket Rao, that many nutritionists have recommended either removing them or destroying them by overcooking. However, new evidence is indicating that at relatively low levels, some of these compounds can be beneficial.

Rao says recent studies involving enzyme inhibitors and saponins, for instance, indicate they may be useful in managing a number of diseases, including cancer, diabetes and hypercholesterolemia. And human studies reported last week by his colleague, Lilian U. Thompson, show that by retarding starch absorption, low levels of lectins and phytic acid may benefit those, like diabetics, who can't regulate blood glucose well.

In one study, Thompson rotated 10 healthy and 10 diabetic individuals through 16 different breakfasts—each containing 50 grams of starch. Every morning they ate legumes, potatoes or a cereal-grain food. "We found that the higher the intake of phytic acid and lectins, the lower the blood-glucose levels," she says. Invariably, the potatoes and grains (including breads, oats, rice and bran) caused a rapid, high peak in blood sugar. The seven legumes (including beans and chickpeas) initiated a slower, lower rise in blood sugar. The legumes' sugar-release pattern is desirable, especially for people who have trouble managing blood sugar.

Though some have interpreted low blood-glucose levels after meals as evidence that much of the meal is not being used by the body, Thompson says her data from *in vitro* and other studies suggest otherwise: Food is being used; the pace of its absorption has simply been slowed.

In a follow-up experiment, 10 healthy volunteers ate unleavened bread made from navy-bean flour—with and without the beans' usual phytic acid. Their blood-glucose responses were 52 percent higher after they had eaten the bread *without* phytic acid.

Because high levels of lectins and phytic acid can be toxic, Thompson is quick to caution against adding either to the diet. However, her preliminary studies suggest they should not be removed from the diet either, because the amounts found in a varied, balanced, high-fiber diet may have subtle benefits. She believes that like fiber, which for decades was considered an antinutrient, these compounds deserve serious reevaluation in terms of dietary importance.

Protecting tight bites

Periodontal disease, the leading cause of tooth loss in adults, is an inflammatory process generating enzymes that attack the rope-like collagen fibers anchoring teeth in place. Dentists usually diagnose the disease by the presence of loose teeth and gum inflammation or the formation of pockets between the teeth and gums. By the time such symptoms appear, significant tooth detachment may have occurred, notes University of Toronto biochemist Jaro Sodek. But his preliminary studies indicate that a simple diagnostic assay for the disease may be on the horizon.

Through a process called remodeling, collagen fibers can break and reform, permitting teeth gradually to move and adapt to changing conditions in the mouth. The process begins with single enzyme-initiated breaks in the protein chain of selected collagen fibrils. The initiating enzyme — one of the neutral metalloendoproteinases (NMPs)—not only causes the collagen fibrils "to begin unraveling," Sodek says, but also opens them up to attack and breakdown by other NMPs. These remodeling NMPs are produced by healthy cells in periodontal tissue—the same cells that will later direct the synthesis of new teeth-anchoring collagen.

In periodontal disease, NMPs produced by inflammatory cells—largely white blood cells—trigger a massive breakdown of collagen. And unlike the cells controlling remodeling, inflammatory cells do not replace the collagen they degrade.

Sodek and his co-workers have been studying the NMPs in sulcal fluid exuded from between the teeth and gums as a possible marker of developing periodontal disease. In a study with beagles, they found that levels of such enzymes "could be related to the progression of [periodontal] disease," Sodek reported last week. In another study following teens with localized juvenile periodontitis (characterized by rapid tissue breakdown) for six months or more, treatment reduced NMP activity.

Finally, an ongoing study is analyzing sulcal fluid collected in a mouth rinse. After flushing excess saliva from the mouth, subjects swish distilled water through their teeth and spit it out to be assayed. The researchers have found high levels of active enzymes in people in whom periodontal-tissue breakdown was later confirmed. Inactive enzymes were washed from individuals with inflamed gums but no breakdown of ligaments supporting teeth. And rinses from healthy people showed high levels of proteins that block NMP-enzyme activity. Sodek says data from the several dozen individuals screened thus far suggest the assay is sensitive enough to aid in the early detection of even largely asymptomatic periodontal disease.

Trapping viruses in blood

An estimated 3 to 5 percent of U.S. recipients of blood transfusions will contract infections from contaminating viruses. Though blood is routinely screened to spot such viruses, a few still evade detection. To catch these, chemical engineers at the University of Michigan in Ann Arbor have developed an adsorbent-based virus-immobilizing filter for whole blood.

In tests with the herpes simplex virus, reports project leader Henry Wang, the experimental system reduced massive viral contamination in just one pass through the filter from 100,000 viruses per pint to around 100. But because the system is really aimed at finding and trapping just the trace quantities that elude blood-screening tests today, he and co-developer I-Fu Tsao believe it "can potentially remove all viral contaminants" from donated blood.

Viruses infect healthy cells by attaching to specific receptors on the cell surface. The Michigan researchers bind healthy cells carrying these receptors to sterile, 200-micron dextran beads. Then the bead-bound cells are packed into a 10-milliliter column. As blood passes through, viruses will leave the blood to bind with open receptors on the bound cells, Wang says. And his data suggest free viruses are not the only ones susceptible to such trapping.

"It is well known that the structure of the [normal] cell membrane undergoes certain modifications in the course of virus infection," he notes. These cells usually develop identifying marker antigens on their surface that match those on the surface of the virus infecting them. As long as this antigen is present, Wang says, an infected cell in the blood will be as fatally attracted to the filter as are free viruses.

Depending on which attractant cells are initially bound into the filter, Wang says, his system can be engineered to trap specific viruses or the whole range of those found contaminating blood. For example, upcoming tests will measure the filter's efficacy in immobilizing the AIDS virus using trapped T cells. Wang estimates that the cost for blood filtration using this device should be "much less than $10 per pint of blood."