Biochemistry

Cat allergy? Try tea and sympathy

Cats are notoriously fastidious. But each cleansing lick of their rough tongues brushes a fine coating of feline proteins across the fur, and the proteins can accumulate for years in carpeting and upholstery. Produced in the animal's salivary and sebaceous glands, these proteins trigger the runny eyes, stuffy nose and wheezing that characterize cat allergy in millions of Americans, including an estimated 30 percent of asthmatics. Physicians usually advise cat owners who develop the allergy to find their animals a new home and stock up on antihistamines. But allergy sufferers may soon receive an additional recommendation: Treat carpets and furniture with a spray containing tannic acid, a compound found in oak bark, coffee, cocoa and tea.

Over the past six years, several research teams have shown that tannic acid can chemically alter house dust, pollen and dust-mite antigens so that they no longer elicit allergic reactions. In homes with up to three cats, a commercially available tannic acid spray also dramatically reduced carpet levels of the primary cat allergen, according to new research led by allergist Jeffrey D. Miller of Danbury, Conn., and Thomas A.E. Platts-Mills of the University of Virginia in Charlottesville.

The team gauged antigen levels in seven homes by vacuuming a square meter of carpet for two minutes. They took samples before, 24 hours after and one week after spraying the carpet with a 3 percent solution of tannic acid in water. Homeowners kept cats off the carpets the day following treatment.

On average, the spray initially reduced antigen levels to about 6 percent of pretreatment levels. Six days after the cats returned, concentrations returned to pretreatment levels in the four carpets that had harbored the lowest antigen levels before spraying (less than 1,450 micrograms per gram of dust). But antigen levels remained at less than half the pretreatment levels in the other three carpets, which initially held 41,000 to 80,000 micrograms per gram of dust.

The researchers conclude that the pale-tea-colored sprays "could be of clinical benefit" for allergy sufferers once their cat has departed, or when residents prepare for allergic guests.

Aromatic alternative to oat bran

Add yet another soluble fiber to the dietary arsenal against diabetes and elevated serum cholesterol: a gel derived from the aromatic seeds of fenugreek, an Asian legume. Two years ago, Zecharia Madar at the Hebrew University of Jerusalem in Rehovot, Israel, showed that dietary supplements of ground fenugreek seeds could lower blood glucose levels in both healthy people and diabetics. Now he and Ilan Shomer of the Agricultural Research Organization in Bet Dagan, Israel, have identified a viscous substance called galactomannan as the ingredient apparently responsible for fenugreek's glucose- and cholesterol-moderating roles.

In experiments with living rats and isolated rat intestines, they found that the galactomannan-based gel fraction in fenugreek seeds inhibits the gut's digestion and absorption of starch — probably by surrounding the starch in a sticky goo. This may have been what hindered the release of glucose into the rat's blood, preventing the type of blood sugar increase that can characterize a diabetic's response to a meal. Glucose inhibition was highest when the rats ate the gel and the starch at the same time, the researchers report in the August JOURNAL OF AGRICULTURAL AND FOOD CHEMISTRY.

The experiments also showed that fenugreek gel tied up bile acids — cholesterol-derived compounds that aid in fat digestion. This suggests an explanation for the herb's reported ability to lower cholesterol, Madar and Shomer say. Trapped bile acids may get excreted rather than recycled, signaling the body to convert more cholesterol into new bile acids.

Biomedicine

Bad eggs indicted in *Salmonella* probe

When Edward E. Telzak and his crew of sleuths arrived at a New York City public hospital in 1987 to investigate an outbreak of food poisoning, they knew the illnesses stemmed from *Salmonella enteritidis* bacteria but they didn't know where the microbes originated. After an extensive probe, Telzak's team identified the culprit. The diarrhea afflicting 404 hospital patients, and the deaths of nine severely ill patients already suffering from lowered immunity, apparently resulted from a batch of bad eggs.

Between 1976 and 1989, the incidence of *S. enteritidis* infections rose more than ninefold in the northeastern United States, according to the Centers for Disease Control in Atlanta. Scientists cannot entirely explain the surge, although past investigations have implicated Grade A raw eggs as one source of the bacteria. Telzak's team probed the largest U.S. hospital outbreak of *Salmonella* food poisoning to date, which they describe in the Aug. 9 NEW ENGLAND JOURNAL OF MEDICINE.

The first clue to the cause of this outbreak came when Telzak's crew discovered that food poisoning symptoms appeared in 41 percent of the hospital patients eating low-salt meals but in only 28 percent of those eating the hospital's regular-salt meals. Suspecting a lunch served on July 28, the scientists soon focused on a tuna-macaroni salad made with either of two types of mayonnaise — a commercially prepared mayonnaise or a low-salt version prepared in the hospital kitchen with raw eggs.

Laboratory tests revealed *S. enteritidis* in both the regular tuna salad and the low-salt version, but the researchers saw no trace of the microbe in the canned tuna or the commercial mayonnaise — a finding that implicated the hospital's own mayo and perhaps the eggs used in its preparation. Sure enough, the team found *S. enteritidis* in the remaining eggs left in the hospital's pantry. The researchers believe kitchen staff inadvertently contaminated the regular-salt tuna salad by stirring it with the same paddle used to mix up the low-salt version, says Telzak, who conducted the investigation while working for the city health department. He has since moved to Memorial Sloan-Kettering Cancer Center, also in New York City.

Family ties reflect lung cancer risk

Researchers have yet to unravel the complicated tangle of factors leading to lung cancer. But two reports in the Aug. 1 JOURNAL OF THE NATIONAL CANCER INSTITUTE suggest that a still-unidentified gene influences the risk of lung cancer.

Thomas A. Sellers at the University of Minnesota in Minneapolis and his colleagues tailored a mathematical model to study 337 lung cancer patients and their families. After controlling for lung cancer risk factors such as smoking, the researchers discovered a pattern of lung cancer incidence suggesting that certain family members inherited a gene that raised their chances of developing the disease. In their model, nonsmokers who inherit the mysterious gene from both parents have a lung cancer risk more than 2,200 times greater than that of nonsmokers lacking the gene, the team reports.

In separate work, a team led by Neil E. Caporaso of the National Cancer Institute found a heightened risk of lung cancer in people who inherit from one or both parents a gene that helps the body metabolize certain chemicals in cigarette smoke. Caporaso's study of 96 lung cancer patients and 92 controls, including some with other respiratory diseases or cancers, links lung cancer to the gene coding for an enzyme called CYP2D6. Caporaso says it remains unclear whether this is the same gene implicated by Sellers' model, and he notes that a variety of genes may influence the risk of lung cancer. But he emphasizes that cigarette smoking remains the most important factor contributing to the development of the disease.