Chemistry

From our reporters at the meeting in Washington of the American Chemical Society

Pockets of flavor production in cheese

In the olden days, lucky actions of microorganisms turned milk into delicious cheeses. But these high-flavored cheeses were difficult to distribute because of their short shelf lives. Modern food processing techniques allow contemporary cheese makers high quality and long shelf life for processed cheese, but they do not consistantly produce the flavors that so please the palate. Now scientists at the University of Wisconsin are working to cage the enzymes responsible for the complex array of gourmet flavors, in order to add the missing taste distinction to processed cheese.

Cell-free extracts of each of several microorganisms used in cheese production have been encapsulated in butterfat shells about 10 microns in diameter, reports Norman F. Olson. Each capsule contains, in addition to the microbial enzymes, the raw materials for flavor-producing reactions. The capsules are added to milk and then trapped in the curd when the milk is clotted. The flavors produced in the capsules diffuse out into the cheese.

Olson reports that four encapsulated extracts have produced a variety of dairy-related (buttery or cheesey) flavors. The final levels of these products could be altered by changing the concentrations of raw material, enzyme, coenzyme or acidity within the capsules. The final product concentrations were relatively stable within the cheese during storage.

"These data indicate that flavors can be produced at controlled rates to specified, stable levels in foods with the microencapsulation systems," Olson says. In contrast, enzymes added directly to foods can result in uncontrollable side reactions with undesirable products, flavor imbalances and overproduction of flavors during the food's distribution and sale. And adding flavors directly can be expensive or technologically impossible.

The microencapsulation approach to flavor development should be applicable to any food in which intact capsules can be entrapped and dispersed, Olson says. Therefore it may become possible inexpensively to intensify flavor or speed flavor development in many foods, especially those requiring fermentation.

Super drug for super rodents

In 1945, scientists got the jump on rats with the discovery of warfarin, a safe-to-use and effective rodenticide that supresses the blood clotting factor Vitamin K. But by 1960, rodents had evened the score; Norwegian rats and then mice became resistant to anticoagulant poisons, and soon populations of so-called "super rats" appeared in other parts of Europe and in the United States. Now, researchers at Lilly Research Laboratories in Greenfield, Ind., have chalked another one up for science by developing bromethalin, a potent rodenticide that kills mild-mannered and super rats alike.

Barry A. Dreikorn and George O. P. O'Doherty were investigating chemicals to control fungus, using a routine rat toxicity test. One compound proved so lethal — too toxic to be used as a fungicide — that the researchers decided the drug might make a good rat poison. The compound did not appeal to the rodents' palate, however, so the scientists changed the chemical subtly, making it appetizing without reducing its toxicity. The result is bromethalin, a drug that slows the transmission of nerve impulses, causing paralysis, and finally death.

In field trials, the new poison averaged more than 90 percent effective in killing rats and mice — including resistant species — after a single feeding. Because rodents don't die until two to three days after ingesting bromethalin, scientists doubt the animals will learn to avoid this poison, as they have learned to avoid arsenic and strychnine. The researchers also doubt that bromethalin-treated rodents will pose a threat to animals that eat the poisoned carcasses.

Behavior

The chemistry of doubt

We all experience uncertainty and the need to double-check: Was the stove left on? the door locked? But for some people such checking can be a debilitating obsession: doubt about one thing or another is constantly intruding on their thoughts, and checking two, ten or even thirty times does nothing to relieve the uncertainty. Two National Institute of Mental Health psychiatrists, Thomas R. Insel and David Pickar, reasoned that such a disorder — officially known as obsessive-compulsive disorder — might derive from a defect in the brain's neurochemical system for reward and satisfaction. Since the brain's natural opiate system has been implicated in reducing animal "drive," they hypothesized that obsessive checkers might suffer a deficit in this reward system — a deficit that is manifested intellectually as an inability to reach certainty.

The researchers studied two obsessive-compulsives, giving them either a placebo or a dose of naloxone — a drug that inhibits the opiate system. Both patients became agitated and absorbed in checking rituals within an hour of taking the drug (the placebo had no effect). Furthermore, the obsessional symptoms were identical in quality and intensity to those accompanying normal exacerbation of the illness, suggesting that, while spontaneous doubt itself may be natural, the irrational persistence of uncertainty may result from a deficit in the biochemical system underlying self-gratification.

Malnutrition by choice

Dieting is a central fact of American culture; indeed, diet book publishing, a successful industry, is founded upon what has been called "the tyranny of slenderness." As scientists have debated the pros and cons of a little body fat, millions of dieters have opted for a svelte figure, whatever the risks. Now, it appears, these values are being passed on to a highly vulnerable group: Children, some quite young, are voluntarily restricting their diets because they fear being fat, with consequences that include stunted growth and delayed puberty. Michael T. Pugliese and his co-workers at the North Shore University Hospital in Manhasset, N.Y., evaluated 201 children referred to the hospital because of short stature or delayed puberty, and found that 14 of them (nine boys, five girls) had failed to grow normally because of self-imposed malnutrition. As they report in the Sept. 1 New England Journal of Medicine, this behavior was rooted in the children's fear of growing fat.

All 14 of the children (who ranged in age from 9 to 17) were below the fifth percentile in body weight, and 11 were also below the fifth percentile in height; 7 of the older children suffered from delayed puberty. The researchers found through interviews that the children were severely restricting caloric intake (to as little as 32 percent of normal) and that they were habitually skipping meals. Interviews with the patients and their parents revealed that all 14 were intentionally dieting to stay thin.

None of the patients or family members had a severe psychiatric problem, the researchers report, and although they say that such precocious dieting (if untreated) could be a precursor of anorexia nervosa, it seems to be a different condition: Unlike anorectics, this group was dominated by males; they experienced no distortion in their perception of body image; they did not use vomitting, laxatives, or compulsive exercise to control weight. However, the researchers add, eight of the families did report a preoccupation with slenderness, and sometimes this preoccupation included a concern about the child's figure. Indeed, the researchers report an additional case in which an infant failed to grow because of the parents' fear of obesity. These findings, they emphasize, may represent only the tip of the iceberg: The children who are seen are those who are at the low extreme in growth, but children who would be tall, and by dieting end up average height, might not be recognized as abnormal.