Biology

Bovine leukemia's hidden toll

Bovine leukemia virus kills cattle, saddling the U.S. dairy industry with annual losses of about $44 million from the full-blown disease. However, this total may greatly understate the damage, according to the first study of the infection in its advanced inapparent stages, says immunogeneticist Harris A. Lewin of the University of Illinois in Urbana-Champaign.

The new findings indicate the virus is most prevalent among cows bred to produce large amounts of milk, and that it thins the milk of infected cows that have developed abnormally proliferating white blood cells. Thus, Lewin says, it squeezes profits on two fronts in addition to its lethality. Dairy farmers, he notes, are paid on the basis of milk fat and volume.

Lewin and co-workers Ming-Che Wu and Roger D. Shanks sampled blood from 219 outwardly healthy cows over a two-year period, looking for antibodies to the virus as well as for an abnormally high level of B lymphocytes, which indicates a stage of the ailment known as persistent lymphocytosis. Lymphocytosis occurs in about 30 percent of virus-infected animals and increases a cow's chances of developing terminal disease, but it does not make them visibly sick, Lewin says.

The scientists matched the blood data with estimates of each cow's genetic potential for milk and fat production, actual milk and fat production and milk-fat percentage. They found that estimates of genetic potential for milk production were significantly greater for both groups of infected cows than for noninfected cows and that the genetic potential for fat production was greater in cows with lymphocytosis.

"These results show that cows with superior genetic potential for milk and fat yield were more susceptible to subclinical progression of [bovine leukemia virus] infection," the researchers report in the February PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (Vol. 86, No. 3).

Lewin believes stress accounts for the greater infection rate among high milk producers, some of which churn out as much as 65 pounds of milk in a day. "Producing a lot of milk is stressful for them, and stress influences the progression of the disease," he suggests. Cows with persistent lymphocytosis produced milk with about 9.2 percent less fat than that of uninfected cows, probably because they "are using fat to make more lymphocytes," Lewin says.

To confirm and refine his results, Lewin is conducting a five-year study of how milk production and fat content change in individual cattle as the disease progresses.

Ticks follow fellows to tastiest cows

The southern African bont tick, which produces a deadly malady in cattle known as heartwater disease, prefers animals tested out by its peers, according to a study conducted in Zimbabwe and reported in the Jan. 20 SCIENCE.

The tick's discriminatory practice may aid the spread of heartwater disease, allowing ticks to steer clear of cattle treated with tick-killing agents in places where other animal hosts abound. It may also cause some unexposed cows to lose their immunity to the disease, report Conrad E. Yunker and Howard R. Andrew of the University of Florida in Gainesville and Zimbabwe government scientist Andy Norval.

Bont ticks apparently are drawn to their hosts by a chemical signal of approval left by their male tick predecessors. To test whether this results in choosy behavior, the researchers released 200 fluorescently tagged, male-female tick pairs downwind from two pairs of steers. One pair had already been infested with signal-emitting male ticks. Ticks downwind from the clean cows dispersed, but the tick-harboring cows served as a magnet for both marked and wild ticks. Another experiment confirmed that the tick-emitted chemical alone would do the trick, the scientists report.

Chemistry

Splicing on-off switches into proteins

The virus T4 breaks into bacterial cell walls and replicates once inside. Using a cutting enzyme called a lysozyme, the virus snips through the wall – a fence of interconnected sugar and protein molecules. To work, the lysozyme's "mouth," or active site, must have access to the wall's sugar units. Scientists interested in engineering enzymes for new uses report controlling the enzyme's cutting activity by building a reversible barrier across its mouth.

Using a genetic engineering technique called oligonucleotide directed mutagenesis, Brian W. Matthews and Masazumi Matsumura of the University of Oregon in Eugene replaced each of two amino acids near the enzyme's active site with cysteine, a sulfur-containing amino acid. Since the substitute cysteines were close to each other in the mutant enzyme's structure, their sulfur atoms could join into a disulfide bond. "Our results demonstrate the novel use of an engineered disulfide bond for the control of enzyme function," the researchers write in the Feb. 10 SCIENCE. The mutant enzyme molecules cannot perform their usual job because the disulfide bond blocks their active sites, the researchers found. But by breaking the bond with reducing agents such as dithiothreitol, the scientists can reopen the active site and restore most of the enzyme's bactericidal activity.

Light ways of making a future fuel

Despite current comfortable supplies of fossil fuels, future-oriented scientists continue to develop alternative fuel sources. Chemist John M. White and his colleagues at the University of Texas at Austin are focusing on clean-burning hydrogen. In the January/February LANGMUIR, a journal of surface and colloid research, the scientists report generating small amounts of hydrogen using water, light and polymer-coated particles of the semiconductor titanium dioxide.

The scientists pepper the semiconductor particles with water-splitting platinum catalysts and coat this combination with the electrically conducting polymer polypyrrole. Light, predominantly of ultraviolet wavelengths, bumps electrons out of the semiconductor, and the conductive polymer coating then ferries them to tiny catalyst islands, which use the electrons to chop water molecules into hydrogen and oxygen. By adding the light-harvesting dye fluorescein to the polymeric covering, the chemists sensitize the particles to work with visible wavelengths.

White envisions placing panels of the hydrogen-generating particles in sunny places, then collecting and using the fuel in machinery modified to run on hydrogen. But before that happens, White says, the group needs to make its fledgling technology a good 1,000 times more efficient.

Car exhaust gets another indictment

Among the pollutants belched into the air by gasoline-powered vehicles are multi-ring chemicals known as polycyclic aromatic hydrocarbons (PAHs). In recent years, scientists have been finding nitrogen-containing versions of these on organic particles collected from the atmosphere. In the Feb. 3 JOURNAL OF ORGANIC CHEMISTRY, chemist William A. Pryor and his colleagues at Louisiana State University in Baton Rouge report a possible connection between these two observations.

The researchers discovered that naphthalene – a two-ring PAH found in exhaust – can react with nitrogen dioxide, another important air pollutant, via a new mechanism involving unstable molecular fragments, or free radicals. The result is a variety of mutation-producing nitro- and dinitronaphthalenes, identical to those pollutants found on the organic particles. The conditions of the laboratory reactions also occur in the atmosphere, the researchers say.