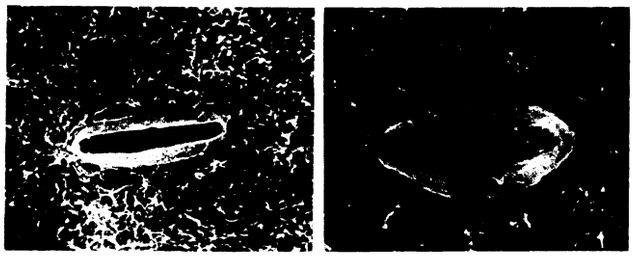
Biology

A rose is a rose . . . no longer

By any other name, a patented hybrid rose just isn't the same. Worth $44 million a year, the breeding and selling of thousands of rose plant varieties can lead to concerns about patent rights. An identification process approved by the U.S. Department of Agriculture (USDA) uses scanning electron microscopy, with results that should solve any questions regarding a particular rose's ancestry.

Developed by Charles R. Krause, a plant pathologist at the USDA laboratory in Delaware, Ohio, the technique thus far has characterized 20 rose hybrids on the basis of such leaf traits as shape of openings, hairs and wax arrangement. For example, the photo on the right shows the Antigua rose, with its recessed leaf pore; the photo on the left shows the Promise rose's own characteristic pore. Krause says the technique could be used on other plants.



Krause

When growing down isn't good enough

There are "renegade" tree roots in the Amazonian rain forest that apparently listen to the beat of their own drummer. Rather than grow downward into the soil as good roots should, they grow vertically up the trunks of neighboring trees—most likely in search of a better meal.

A study of these so-called apogeotropic roots by Robert L. Sanford Jr. of North Carolina State University in Raleigh is reported in the Feb. 27 SCIENCE. During field experiments in Venezuela, Sanford found that the roots of at least 12 species of tropical trees send out climbing roots that originate in the soil and then decide to take the high road — often creeping more than 13 meters up the trunks of other trees, as fast as 5.6 centimeters in three days.

Sanford proposes that this "nutrient cycling pathway" develops in response to the nutrient-poor soil found in tropical forests. Previous studies have found that the canopy, or thick-foliage top, of such forests scavenges nutrients from rainfall. A certain amount of that precipitation flows down tree trunks.

To test his hypothesis that this "stem flow" is related to upward-growing roots, Sanford used artificial tree stems of plastic pipes, and near their tops attached cylinders containing manure, forest soil or nothing. He found that those with nutrient-rich manure being leached down by rain supported climbing roots from nearby trees sooner and more abundantly.

National animal health system

In an effort to monitor infectious diseases and other health problems of livestock, as well as their economic effects, the USDA is developing the National Animal Health Monitoring System. Pilot programs already completed in seven states have been used to refine the system, which is expected to be functional by the end of fiscal year 1988. Individual states will be responsible for collecting information for the federal government's computer system; the USDA's Animal and Plant Health Inspection Service (APHIS) in Hyattsville, Md., will distribute summary reports based on the compiled data. According to APHIS officials, livestock health problems cost producers an estimated $14 billion annually.

Biomedicine

Alzheimer/Down syndrome bond tightens

The latest in a series of reports suggesting a common genetic defect for Alzheimer's disease and Down syndrome comes from an international corps of scientists in Paris and the National Institutes of Health (NIH). According to a report in the March 13 SCIENCE, there is an extra copy of a gene — responsible for the production of the protein amyloid — in cells from both Alzheimer's and Down patients. Abnormal deposition of the protein in brain tissue is characteristic of the two diseases, and the extra gene could lead to excessive amyloid production.

Earlier studies had localized the amyloid gene on chromosome 21, known for years to be abnormal in people with Down syndrome. Also, the gene was found near a genetic defect responsible for an inherited form of Alzheimer's. After observing that similar abnormalities are found in brain tissue from both Alzheimer's patients and older Down syndrome patients, researchers had begun searching for the link between the two disorders (SN: 1/25/86, p.60).

Last fall, scientists from NIH announced their characterization of a gene on chromosome 21 that codes for the protein amyloid (SN: 11/22/86, p.327). That announcement was followed several months later by a flurry of published reports dealing with the amyloid-coding gene and its possible role in both diseases. Although the growing amount of data implicating the amyloid gene does not prove that the genetic aberration is the sole or even primary cause of either condition, it strongly suggests a genetic defect in Alzheimer's disease.

Probable eye cancer gene scrutinized

At the University of California at San Diego in La Jolla, researchers have cloned and characterized the gene thought responsible for susceptibility to hereditary retinoblastoma, an eye cancer found in children (SN: 1/5/85, p.10). Altered genetic material — apparently due to deletions in the gene located on chromosome 13 — was detected in cells taken from retinoblastomas and two other types of tumors, according to an article in the March 13 SCIENCE. The authors say their results offer "a framework for further study of . . . genetic mechanisms in human cancers."

Mutation causing cleft palate found

Another study that could be a research springboard into the broad arena of genetic disorders has been completed by research groups at the University of London, the Whitehead Institute for Biomedical Research in Cambridge, Mass., and the National University Hospital in Reykjavik, Iceland. In the March 5 NATURE, the scientists report that they have localized the gene mutation causing cleft palate.

The genetic defect, assayed in a large family in Iceland, appears to be linked to the X chromosome, say the scientists. Because cleft palate develops during the stage of embryo development associated with disorders like spina bifida, closer analysis of the newly identified gene mutation may elucidate the mechanism of other developmental abnormalities.

Shivering mice 'warmed' by gene therapy

Injection of a normal gene that codes for a nerve-cell-sheathing protein can cure a type of mouse suffering from tremors and convulsions, say the authors of two reports in the Feb. 27 CELL. Mice carrying the "shiverer" gene mutation, which can lead to early death, do not produce myelin basic protein (MBP), essential for the conduction of nerve messages. The therapeutic genes were injected into fertilized shiverer eggs, which developed into individuals free of the abnormal behavior caused by lack of MBP. The research was done at Pasadena's California Institute of Technology and at Harvard Medical School in Boston.