Natural Sciences

Birth of a swamp

The Great Dismal Swamp, a 210,000-acre wet wilderness that straddles the Virginia-North Carolina border (SN: 3/3/73, p. 132), began forming 9,000 years ago when subterranean water under artesian pressure pushed toward the surface, conclude U.S. Geological Survey hydrologists William F. Lichtler and Patrick N. Walker of Richmond, Va. Previous theories stated that beaver dams or rises and falls in sea level caused the swamp to form.

According to the USGS report, the development of the swamp was largely controlled by what is called the Norfolk Formation, sandy, water-permeable beds that lie beneath most of the Dismal Swamp. Formed about 100,000 years ago, the formation was sandwiched between clayey silt above and marine clay below. The marine clay restricted vertical inflow and outflow of water.

As time passed, the authors explain, surface erosion began removing the overlying silt and exposing the Norfolk Formation. This allowed subterranean water under artesian pressure to seep to the surface triggering a chain of reactions. The upward flow of water plus abundant rainfall and poor drainage led to the formation of peat (particularly decaying plant matter) on the floor of the swamp. The peat further inhibited drainage, which in turn speeded the accumulation of peat in a progressive cycle that is still going on today.

Early conifer fossils found

Charcoal fragments and fossils of small leaves and leafy shoots recently discovered near Wakefield, England, are said to represent the oldest remains of conifers ever found. Discovered by British botanist Andrew Scott of Birkbeck College, the cone-bearing evergreens have been dated in the Upper Carboniferous (270 to 350 million years ago).

Scott reports in the Oct. 25 NATURE that unlike other plant fossils found in the same location the small, narrow and occasionally forked leaves reveal both epidermal and internal structure. Electron micrographs of the fossils reveal collapsed epidermal cells and occasional absence of cuticles, suggesting to Scott that the leaves may have been charcoalified by a forest fire. Though the plant material has not been assigned to a genus as yet, it is believed that it belongs to the family Lebachiaceae.

In autumn plants . . . commit suicide?

Why do most annual plants die during the autumn? Why do biennials live happily through the winter, then die after two growing seasons? What stops turning them on and starts turning them off? A team of plant physiologists from the University of Michigan at Ann Arbor think they "commit suicide."

Larry D. Nooden and Susan L. Schreyer are studying a chemical "death signal," probably a hormone, which they have traced to plant seeds. They hypothesize that seeds inside mature fruits, such as soybean pods, send out hormones which cause plants to yellow and die even before the autumn nights are cold enough to freeze them.

"Gardeners have noticed for years that if faded flowers are picked before they go to seed, the plant will continue to grow and produce more flowers," Nooden says. He and Schreyer tested this on soybeans by plucking growing pods from one side of a plant and allowing pods to remain and mature on the other side. Predictably, the side with the mature pods and seeds turned yellow and died, while the other side remained healthy. Nooden hopes to isolate the hormone factor and learn more about the suicide physiology.

In evolutionary terms, he hasn't yet determined the adaptive significance of the mechanism. One idea, that the established parent plant is "making way" for the young seedlings, does not always make sense, Nooden says. "In the desert for example, yuccas 'commit suicide,' but in that environment, it is of advantage for the plant to be established in one spot. So why would the established parent die and make room for vulnerable seeds?" Although perhaps hidden, there probably is some evolutionary advantage, Nooden says, "because, obviously, the characteristic has been selected for and persists."

Two toxin-producing bacteria in one

It has been known for several years that many species of toxin-producing bacteria cannot make the toxins unless infected by phages (bacterial viruses). Such is the case with two insidious species of the genus *Clostridium*. One, when infected, produces botulism toxin and the other produces lethal toxins in gangrenous wounds. Many of these toxin-producing species, including the two mentioned, are so similar in appearance and physiological characteristics that they can be distinguished only by the nature of their toxins.

A team at the Pacific Utilization Research Center in Seattle, headed by Melvin W. Eklund, now reports in the Nov. 1 SCIENCE that a botulism-producing species and a gas gangrene species can be "interconverted." It appears that they aren't two species at all, but a common bacterial strain that produces botulism toxin when infected with one phage, and gas gangrene toxin when infected with another. This finding probably will affect the future classification of such bacterial species, and may lead to a common method of controlling both dreaded afflictions.

How an insect hardens its armor

Swarms or trails of insects often have been compared to marching armies. This is an appropriate comparison when it is remembered that insects, and arthropods in general, wear a kind of armor. Instead of an internal support system of bones, insects have exoskeletons that support and protect the soft internal organs. This stiff exoskeleton consists of several layers of cuticle, but the cuticle is not always hard. Immediately after an insect sheds its hard "skin" (the only way it can increase its size), the thin cuticle layer forming below is soft and almost clear.

The exact physiological changes that take place within the cuticle layers as they harden and in some cases darken are as yet unknown, but a recent report clarifies the process somewhat. Danish zoologist S. O. Andersen from the University of Copenhagen reports evidence in the Oct. 11 NATURE for two mechanisms of cuticle hardening (called sclerotization).

He reports that an amino acid substrate called N-acetyldopamine is acted upon by one or more enzymes to cause the cross-linking of cuticle proteins and thus the hardening of the layers. The substrate molecule has a ring portion and a chain portion, and Andersen presents evidence that the ring is acted upon to form clear cuticle and the chain is acted upon to form brown cuticle. The two enzymes might both act at the same time, he says, with their relative concentrations determining the darkness of the cuticle. Probably a third enzyme, which he says remains to be studied, controls the deposition of pigment molecules.