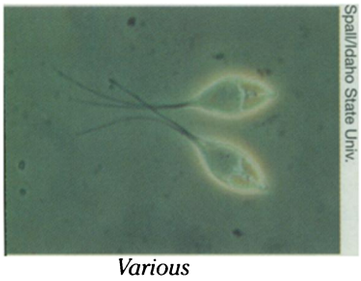
Protist joins metazoan family tree


Spall/Idaho State Univ.

Various myxozoans.

They may look a bit like single-celled organisms such as a euglena or paramecium, but the fish and worm parasites called myxozoans are not protozoans, says Richard D. Spall, a pathologist from Idaho State University in Pocatello.

For years, Spall has spent much of his spare time collecting and dissecting fish and annelids — segmented worms such as earthworms — that often carry myxozoans and studying the microscopic features of these mysterious creatures.

Typically, myxozoan spores consist of just 6 to 12 cells, not enough for taxonomists to promote these organisms from the kingdom Protista to the status of multicellular animals. Yet genetic material taken from the myxozoan's protein production unit, called the ribosome, indicates that this group's closest cousins are actually nematode worms, not paramecia, Spall and his colleagues report in the Sept. 16 SCIENCE.

Spall had suspected as much. The myxozoan spore, 15 to 18 millimeters in diameter, contains highly differentiated cells: two valve cells that cover four end, or polar capsule, cells, which contain ejectable filaments. These polar cells and the cell-to-cell junctions remind him of the stinging cells and junctions seen in jellyfish and hydroids — both radially symmetrical invertebrates. But the myxozoan's ribosomal RNA shows closer ties to bilateral animals, nematodes in particular, he notes.

Little else is known about these organisms. So far, scientists have described 1,100 species of myxozoans, but Spall finds new ones everywhere he looks and suspects many more kinds exist. Though they do not make their natural hosts sick, myxozoans introduced by stocking U.S. waters with nonnative fish have resulted in serious epidemics among native rainbow trout and Pacific salmon. Young native fish develop a whirling swimming behavior and often die, he points out.

Temperature affects mutant protein

Two boys, one from Idaho and another from Minnesota, both with a double dose of rare endocrine abnormalities, have helped researchers pin down the unusual behavior of a defective protein, which in its normal form is one of a group of key signaling molecules in cells.

These molecules, called G proteins, relay messages from the outside to the inside of a cell. They make it possible for hormones to exert their effects, says Henry R. Bourne, a pharmacologist at the University of California, San Francisco.

He and his colleagues became intrigued by both the precocious pubescence in these two toddlers — a result of an overactive hormonal response — and the boys' inability to handle parathyroid and thyroid-stimulating hormones. Both boys need supplements of vitamin D and thyroid hormone.

The researchers reasoned that such rare diseases should occur together only if they involve the same genetic defect. Their hunch proved right. The scientists located the defect and found that it causes one type of G protein to have the amino acid serine where an alanine should be.

Based on what scientists know about the three-dimensional structure of G proteins, Bourne and his colleagues suggest that because serine has an extra side group, its presence alters the G protein's docking site for a nucleotide, GDP. As a result, GDP keeps slipping out, activating the G protein when it leaves and causing the protein to signal incessantly.

In the testicles, this signaling stimulates the production of testosterone, which initiates puberty prematurely, the group reports in the Sept. 8 NATURE.

However, elsewhere in the body, GDP's frequent departures leave the mutant protein vulnerable. This activated form tends to break down at the body's normal temperature, which is about 5°C higher than the testicular temperature, the researchers note. As a result, cells in the body wind up with too little G protein and cannot respond adequately to other hormones, Bourne says.

Colorful coots cash in on care

Although common sense dictates that helpless newborn chicks should bear feathers that blend with their environment, young American coots instead dress up in bright orange and red for their first few weeks of life. Fiery bills and bold skin patches on the head, all flanked by orange feathers on the head and neck, probably make these young rails conspicuous to potential predators. But they also encourage parents to feed their youngsters more often, say Bruce E. Lyon and John M. Eadie of the University of Toronto in Scarborough, Ontario.

When they first observed these flashy chicks, the two biologists wondered whether the red patch and orange plumage were like peacock feathers — eye-catching traits that became exaggerated because they provide their owners with some survival or reproductive advantage (SN: 2/6/93, p.84).

Working with Linda D. Hamilton from the University of Calgary in Alberta, they tested this idea by trimming the orange feathers on half the chicks in 21 broods of these North American marsh birds. They left just a few orange feathers on the throat, so these chicks looked like most other rail chicks and, most likely, this coot's ancestor. In 11 more nests, they removed the orange feathers from all the birds, and in a dozen others they left all feathers intact.

In the group of 21 broods, untrimmed chicks got more food and grew faster than trimmed chicks, the scientists report in the Sept. 15 NATURE. This difference was particularly striking in chicks that hatched late, with orange chicks much more likely to survive than trimmed ones. Yet chicks in broods where all the birds lacked the orange feathers did as well as those in broods where all birds had the orange. Thus, the flashy plumage does not help the parents recognize the chicks but does encourage better care, Eadie says.

Typically, adult birds exercise parental choice because they cannot nurture all their chicks to adulthood. Neglected ones die. Eadie and his colleagues suggest that perhaps at one time, the coot's skin patch turned red to signal a parent that the baby bird was stressed and needed care. Because the flashiness triggers this response, chicks who are always bright have an advantage, one that over time may have caused these birds to evolve ever more elaborate ornamentation. They lose these bright colors after several weeks, the researchers note.


Lyon/NATURE

Bright heads of American coot chicks.