Slime Molds on the Wing

It is no small irony that although the biochemistry, genetics, and developmental biology of the cellular slime molds are relatively well explored, knowledge of the natural history of these popular experimental organisms remains patchy at best. However, owing to the fortuitous combination of two diverse interests—those of slime mold biology and the habits of migratory songbirds—in a Princeton researcher, Hannah Bonsey Suthers, one curious aspect of this natural history appears to have been solved: to wit, the very broad geographical distribution of cellular slime molds. Suthers reports that ground-feeding, migratory songbirds play a major role in carrying these microscopic organisms great latitudinal distances in the Americas and presumably elsewhere (*1*). Although birds are known to be important in the dispersal of higher plants, mosses, and fungi, this is the first demonstration that they also influence the natural history of slime molds.

The life cycle of slime molds involves a stage at which free-living, dispersed amoebas forage independently, followed by an aggregation stage in which the amoebas come together to form a mobile slug, which behaves much like a tiny multicellular organism. After migrating, the slug forms a fruiting body, which can contain several hundred spores. Now, although the slug is mobile, its range is limited: 1 inch in 24 hours is typical. As far as is known, more widespread dispersal of spores occurs by attachment to passing insects (by electrostatic attraction) and by water; both routes are relatively restricted in geographical terms. The discovery that birds may passively carry propagules of slime molds immediately extends the range of potential dispersal. What is of interest here, however, is the distribution of single species over thousands, not merely hundreds, of miles.

Cellular slime molds are remarkable both in the very small number of species in the group—50 as compared with 500 in the myxomycetes, for example—some of which have worldwide distribution. These organisms are very ancient in evolutionary terms, and a global distribution could therefore simply be a consequence of having been passive passengers on landmasses that periodically coalesce and fragment. But the fact that single species are recognizable across several continents implies a continual interchange between populations, because geographically isolated populations would be expected to diverge over relatively modest tracts of geological time.

The initial steps of Suthers's study involved culturing the droppings of ground-feeding birds, simply to see if slime molds could be found there. After showing that they could, she began a systematic survey of slime mold species that could be found in ground-feeding, migratory birds in New Jersey and Central America, which variously represent summer or winter homes for many songbirds. Comparison of slime mold species from droppings with those cultured from the soil surface throughout the seasons shows clear pulses of immigration of slime molds as a result of the various northerly and southerly avian migrations.

Unlike the seeds of higher plants, which pass through birds relatively quickly, slime mold propagules, particularly the resilient spores, can persist as long as 10 days. As the journey from the northeast coast of North America to the Caribbean and South America is accomplished in just 72 hours by millions of songbirds every fall, the potential for long-distance dispersal is obvious. The return journey in the spring is a more leisurely affair of some 20 days, which effects a different pattern of dispersal.

Although this study explains the great latitudinal dispersals in the Americas, it leaves unsolved the distribution of a recently discovered cellular slime mold species, *Polysphondylium filamentosum* (*2*), which occurs in the Swiss Alps, Central America, and, albeit rarely, in North America, apparently transported there by the ovenbird. Suthers notes that no ground-feeding bird routinely makes a trans-Atlantic migration that could be responsible for this distribution.—**ROGER LEWIN**

References

1. H.B. Suthers, *Oecologica* **65**, 526 (1985).
2. F. Traub, H.R. Hohl, J.C. Cavender, *Amer. J. Bot.* **68**, 162 (1981).

Many people think that is the first step in the development of atherosclerotic plaques," Ginsburg explains. A strain of pigs with von Willebrand's disease is protected against heart attacks, even if the pigs are fed a high fat diet which causes normal pigs to get atherosclerosis and heart attacks.

Ginsburg speculates that certain variants of von Willebrand factor could conceivably be an atherosclerosis risk factor. People who have a slightly overactive protein, for example, might be at increased risk. A few years ago, the National Heart, Lung and Blood Institute decided to investigate the proposed link between von Willebrand factor and heart disease by compiling a registry of von Willebrand's disease families and following them to see if they had an unusually low incidence of heart disease, but the project proved too expensive and difficult to do and so it has not been completed. A European and Israeli group, the European Thrombosis Research Organization, also is looking at the incidence of heart disease in people with severe von Willebrand's disease. But Ginsburg and his associates hope to get the answer by reversing the strategy.

Using a molecular probe for von Willebrand factor, they plan to look at families with a high incidence of atherosclerosis to see if heart disease is linked with the inheritance of particular von Willebrand factors. "These studies are doable," Ginsburg remarks. "We can take a DNA probe and look for polymorphisms that are linked to von Willebrand factor. That gives us a way to look at DNA in families and see if a von Willebrand gene is a risk factor. We have already found polymorphisms for von Willebrand factor and now we are starting to look at families." Nonetheless, Orkin cautions, this research is very preliminary and, he remarks, the chances of a payoff are still slim. "My own view is that it's a long shot," he says.

All three groups that cloned the von Willebrand factor gene say they are primarily interested in using the gene to study how the protein is synthesized and how its synthesis is regulated. Dennis Lynch of the group at Dana Farber that cloned the von Willebrand factor gene and, independently, Evan Sandler of the Washington University group point to a number of unanswered questions. For example, the factor is made only in megakaryocytes, which are bone marrow cells that are precursors of platelets, and in endothelial cells. "If we could get the appropriate regions of the gene, we could look at why the gene is only expressed in those two cell types," says Sandler.