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it to see how dangerous it is. This "predator inspection" is risky for the scouts, but the information can benefit them as well as the rest of the school-if the interloper is not a predator or if it's not hungry, the smaller fish don't need to scatter. A group of scouts approaching a predator, Milinski noted, is playing out a Prisoner's Dilemma: Each has a strong incentive to defect and let the others take all the chances, but if all defect, they learn nothing about the predator. Full cooperation, on the other hand, minimizes the risks because the predator becomes confused if it can't focus on a single target. Because potential predators approach the school again and again, Milinski thought that a tit-for-tat strategy might have evolved among the fish.

Milinski and Dugatkin have independently tested the idea—Milinski in sticklebacks and Dugatkin in guppies—and both find that the fish do indeed use a tit-for-tat strategy in predator inspection. Guppies that are paired up in a tank with a predator confined at one end will approach the predator in a sequence of moves, Dugatkin says. "If one of them is trailing, the lead fish will turn around and head back. It will wait for the other to head out, and then it will go by its side." In other words, if one fish defects (holds back), the other will, too, and it then waits for the first one to cooperate (swim forward) before cooperating itself. The guppies even remember from day to day what other guppies did, Dugatkin found. If one of a pair defects in one trial, the other will defect in turn on a second trial the next day.

The verification of the tit-for-tat strategy has led to new and more detailed models of the guppies' behavior, Dugatkin says. "After doing that experiment, watching the fish, and thinking about the model, I realized that guppies should prefer to associate with cooperators because it would be in their interest to be near cooperators if a predator appeared." He later found that, given a choice, guppies did indeed spend more time with fish that had cooperated than with defectors. "These models make some new and very interesting predictions about the evolution of cooperation," Dugatkin says, "and we hope they will spur even more empirical work."

Researchers from other fields will be watching this work unfold, says Hammerstein. Take economists, who have a hard time explaining how markets end up in Nash equilibrium, in which no competitor can gain an advantage by unilaterally changing strategy. Studies of markets suggest that Nash equilibria—the equivalent of ESSs in animals—do arise, but the theory predicting them assumes that the players act in a perfectly rational fashion, which is impossible.

As a result, says Hammerstein, a number of economists are "looking to evolutionary game theory for processes other than rational decision making that could lead to a Nash equilibrium." Perhaps, he says, these stable strategies arise in much the same way as cooperation arises among guppies: People base their behavior not on rational calculation but on experience.

Robert Axelrod, a political scientist at the University of Michigan, raises the possibility that evolutionary game theory might even offer insights into the election-year strategies deployed by candidates for the U.S. Congress. Perhaps politicians imitate the strategies of others, or perhaps some other process from evolutionary game theory is at work. One can only hope that negative campaigning does not prove to be an evolutionarily stable strategy.

-Robert Pool

PHYSICS.

Making Light Work of Brownian Motion

 ${f T}$ he bacteria Listeria monocytogenes are notorious for causing dangerous illnesses such as meningitis. Among a small group of biophysicists and physicists, however, they are famous for a different talent: their ability to swim by harnessing the random jitter called Brownian motion, generated by millions of water molecules constantly striking the bacteria. The trick is in their tails-bushy appendages that ordinarily hold the bacteria steady. When Brownian motion jostles a bacterium forward, explains biophysicist George Oster of the University of California, Berkeley, the microbe briefly sheds its tail. Then it quickly fills in the gap, fixing itself in place until it gets another push forward.

Listeria's scheme for turning random thermal motion into net movement has fascinated researchers because it extracts work out of something long regarded as useless "noise." Over the last 2 years, inspired by Listeria and a few other examples from biology, scientists have conceived simple laboratory schemes that could harness Brownian motion and even turned a few of them into working systems. The 27 February issue of Physical Review Letters reports the latest and, say some researchers, the most elegant: an "optical thermal ratchet," invented by Albert Libchaber and his colleagues at Princeton University and the NEC Research Laboratory in Princeton, New Jersey, that uses light itself to convert Brownian motion into

directed movement of a plastic bead.

Systems like Libchaber's ratchet could provide a novel way to separate various-sized particles and molecules. And, coming full circle to biology, they provide a model of a process that some researchers think might be integral to the work of so-called motor proteins such as myosin, which drives muscle contraction as it moves along filaments of the protein actin. "Can these mechanisms

possibly explain how biological motors work? That's the \$64,000 question," says Steven Block of Princeton University, a physicist who studies motor proteins.

Although these recent laboratory systems were inspired by biology, they also take a cue from a centuriesold mechanism consisting of a toothed ratchet wheel and a pawl, which engages the teeth of the ratchet and allows it to spin in one direction only. In the Princeton system, the wheel is replaced by a microscopic plastic sphere in water, illuminated by an infrared laser beam that rotates rapidly, tracing a circle 7 micrometers in diameter. The beam



induces a fluctuating electric charge in the

team sends the laser light through a "chopper," a filter that modulates the intensity of the beam so as to create a series of sawtoothshaped "hills" around the circle. Each hill has a gentle slope of increasing intensity on

Diffusion

Laser ratchet. A bead caught in

(top) drifts randomly when the in-

an optical trap's intensity peak

tensity is evened out (middle).

Recreating the peaks either re-

turns the bead to the same peak

or advances it one step (bottom).

one side and a steep dropoff in intensity on the other. Because the sphere's fluctuating charge makes it want to reach the brightest spot, says Libchaber, the bead rolls up the nearest hill and then stops at the peak—just as if a pawl had been engaged.

By periodically removing the chopper, Libchaber and his colleagues can force the bead to travel in one direction around the circle. When the chopper is turned off, the sphere diffuses away from the peak where it had been trapped. If it diffuses down the gentle side of the same peak, the sphere will slowly return to its original posiBecause Libchaber's device uses only a single bead and a beam of light, other researchers praise it as a clean, simple demonstration of directed Brownian motion. "It's a very clever device," says Oster. The first Brownian ratchet, reported last year by Juliette Rousselet and Laurence Salome of the Centre de Recherche Paul Pascal in Bordeaux along with Armand Ajdari and Jacques Prost of the Centre National de la Recherche Scientifique in Paris, relied not on light but on an electric field to create the ratchet's hills and valleys. But because the French team was working with a large set of colloidal particles, they couldn't say just how much of a particle's movement was actually due to directed Brownian motion and how much to local interactions among the many particles.

The use of multiple particles did suggest one way to put artificial ratchets to work, however: as laboratory separation devices. The French researchers observed that the speed at which objects move through a ratchet depends on their size. As a result, they speculated, artificial ratchets could sort collections of cells, viruses, or chromosomes. At this stage, however, Libchaber cautions that

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it's too early to say whether this technique would offer any improvement over existing separation methods like electrophoresis.

Even as Brownian ratchets have emerged in the laboratory, they may be looking scarcer in nature. Some researchers have long speculated that the motor protein kinesin, which ferries vesicles within a cell, might be propelled by Brownian motion. But recent studies have suggested that the "steps" it takes as it moves along cellular filaments are too big to explain by thermal motion alone. There's still hope, however, that myosin might rely on a Brownian ratchet, says Oster. It would be a shame, after all, if nature has reserved this elegant scheme for a few deadly bacteria. -John Travis

Playing Chicken With an Epidemic

As if Mexico's current economic and political troubles weren't enough, now its chickens have the flu. The virus, avian influenza A, poses no threat to human health, but it can be lethal to poultry, and some 26 million chickens in three Mexican states are already infected with a deadly strain, says Eduardo Rivera, coordinator of Mexico's National Avian Influenza Campaign. Although Rivera and other authorities are determined to contain the epidemic, some veterinarians say their efforts may be too little, too late to prevent Mexico's flocks from being devastated.

By deciding not to act when a mild form of the virus surfaced last spring, "Mexico made the same mistake we made in 1983," says Charles Beard, a veterinary virologist with the Southeastern Poultry and Egg Association in Tucker, Georgia. In that year, poultry producers allowed a similar mild strain to spread freely; after 6 months it mutated into a virulent form that claimed 17 million birds and cost \$63 million to eradicate. Now Mexico has experienced the same grim progression. With the mutation-prone mild strain present in at least half the country, the epidemic is likely to spread beyond the three states that have already been hit hard by the pathogenic mutant, perhaps even reaching the United States. Says Robert Webster, a virologist at the St. Jude Children's Research Hospital in Memphis, Tennessee, "The virus scares the hell out of the whole poultry industry in the U.S."

Last June, U.S. and Mexican veterinarians and agriculture officials held one of several meetings in Mexico City to discuss the situation. At that point, infected chickens were suffering a mild infection confined to the lungs and gut, and Mexican officials were reluctant to take the steps needed to forestall an outbreak of more serious disease: quarantining infected farms and increasing hygiene measures elsewhere. "I don't think they were convinced that the mild virus could turn into a real problem," says Beard, who spoke at the meeting.

Like the United States before it, Mexico allowed the virus to circulate-and as before, a few critical mutations in the virus's hemagglutinin (HA) protein transformed the mild virus into a serious pathogen. For the virus to infect a bird, host enzymes have to cleave the HA protein, converting it into a form that can infect cells. In mild strains, the protein is cleaved by enzymes in the lungs and gut. But in a process unraveled after the 1983 outbreak, a series of mutations can turn the protein into a more readily activated form, converting a relatively benign virus into a killer. "Once the HA protein acquires a series of basic amino acids, it can be cleaved by an enzyme that occurs in every tissue in the body, including the brain," says Webster. Chickens infected with the mutated form suffer a mortality of 20% to 100% from a devastating systemic infection with internal hemorrhaging and central nervous system collapse.

By the start of this year, this lethal form had surfaced 150 miles southeast of Mexico City in Puebla, on a farm of 1.25 million chickens. Since then it has turned up in another 35 flocks. By this point in the 1983 U.S. epidemic, agriculture officials and farmers had taken aggressive steps. Soon after detecting the virulent virus, the U.S. Department of Agriculture (USDA) killed all chickens infected with either the mild or the virulent strains. The federal government established a quarantine zone, compensated owners, and disinfected farms—and the outbreak was stopped dead.

Mexico, unable to afford the cost of destroying tens of millions of birds, is instead trying to contain the epidemic by vaccinating flocks and enforcing quarantines. Because Mexico has a limited supply of the vaccine, it will be available at first only in states that

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harbor the pathogenic virus, reports Beard. Birds will be vaccinated based on a pecking order, with grandparent genetic stock getting the first doses, followed by broiler breeders, erg havers, and besiders

Flu's in the coop. Avian flu already infects flocks in half of Mexico.

Pathogenic virus

by broiler breeders, egg layers, and broilers. By setting up checkpoints and requiring permits for moving poultry into and out of infected areas, Mexico hopes to maintain disease-free havens, especially in the remote states of Yucatán and Sonora. "The government wants disease-free areas for the breeders so there will be a source of chicks to repopulate the industry," says Beard.

In this country, meanwhile, the USDA and the poultry industry are urging producers to watch for sick birds, send samples to diagnostic laboratories, and step up hygiene practices, such as disinfecting vehicles, equipment, and clothes. For producers with affiliates in Mexico, the authorities recommend limiting travel to the Mexican facilities.

The USDA is also considering a plan to monitor the wild waterfowl that will soon begin their migration northward from South and Central America. Shore birds such as the ruddy turnstone and the red knot are known reservoirs of the mild avian flu, says Webster. And that is raising fears that their arrival could herald not only the coming of spring but also the reappearance of a deadly visitor. –Bernice Wuethrich

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