

Mosquito-Killing Fungi May Join the Battle Against Malaria

They already kill insects in fields, greenhouses, and gardens around the world. Now, a duo of fungi may also become a new weapon in the fight against malaria. In this issue of *Science*, two research groups report the results of lab experiments and field tests in Tanzania indicating that fungal spores can infect and kill adult *Anopheles* mosquitoes, the vectors of malaria parasites. Applied just like chemical pesticides, sprays containing the spores could be a new, environmentally friendly weapon against malaria, the researchers say.

“They have a pretty strong case,” says Christiaan Kooyman, who studies locust control using fungi at the International Institute of Tropical Agriculture in Cotonou, Benin. New control tools are necessary, Kooyman adds, because mosquitoes are increasingly becoming resistant to chemical pesticides.



Getting fuzzy. Fungi that infect and slowly kill mosquitoes create a fuzzy covering on the parasite-carrying insects.

But whether the fungal strategy is technically or economically feasible remains to be seen, others caution. “I have seen plenty of false technological dawns” in vector control, says Jo Lines of the London School of Hygiene and Tropical Medicine.

That strains of the two fungi—called *Beauveria bassiana* and *Metarhizium anisopliae*—can kill mosquitoes didn’t come as a surprise. Both species are used in agricultural biopesticide products, and so many different strains of each fungus exist that there’s probably one to kill almost any insect species, Kooyman says. But no one had set such fungi loose on malaria mosquitoes until recently.

In 2003, one group, led by Bart Knols of Wageningen University and Research Centre in the Netherlands and the International Atomic Energy Agency in Vienna, published a lab study showing that spores of several fungi infected *Anopheles gambiae* when applied directly to the insects’ bodies. Whereas pesticides kill overnight, these fungi grow slowly, often taking 10 or 12 days to kill.

As they report on page 1641, Knols’s team has now tested this idea in the field. They suspended 3-m² cloths impregnated with the fungus *M. anisopliae* from the ceilings of five traditional houses in a rural Tanzanian village, collected mosquitoes in the homes for 3 weeks, and kept the insects alive on glucose. Some 23% of female *Anopheles gambiae* mosquitoes became infected, shortening average life span by 4 to 6 days compared to controls from five untreated homes.

The study was much too small to detect an effect on malaria transmission and not designed to do so. But when the team modeled how such results would alter malaria transmission in a village if the cloths were applied year-round, they found that the number of infective bites for the average villager would fall from 262 to 64 annually. In order to make a dent in malaria cases and deaths, that number has to come down much more, to close to one bite per year. But that is feasible by upping the dose and spraying entire walls in many more houses, says Knols’s collaborator Kija Ng’habi of the Ifakara Health Research and Development Centre in Tanzania.

On page 1638, a team led by Andrew Read of the University of Edinburgh and Matt Thomas of Imperial College London reports that the true effect of a fungus—in their case, *B. bassiana*—on malaria transmission may be even more pronounced than Knols’s data suggest. In lab studies using *Plasmodium chabaudi*, a rodent malaria ▶

French Science Policy Shakeup

PARIS—Junior research minister François d’Aubert has been ousted as part of the new government formed by Prime Minister Dominique de Villepin following France’s overwhelming rejection of the European constitutional treaty. Politician François Goulard, 51, who served as junior transport and sea minister in the last government, now assumes France’s top science policy position and will be responsible for higher education, which d’Aubert was not. Bringing the two portfolios together is “good news,” says Alain Trautmann, spokesperson for France’s researcher protest movement. A long-awaited science reform bill is due to be published next week.

—BARBARA CASASSUS

Final Biodefense Centers Announced

The final pieces of a 10-site national network of biodefense research centers have been put into place. Last week, the National Institute of Allergy and Infectious Diseases announced grants totaling \$80 million over 4 years for two new Regional Centers of Excellence for Biodefense and Emerging Infectious Diseases Research.

One center, a consortium led by Colorado State University, will focus on diseases transmitted by animals. Another team, based at the University of California, Irvine, will host clinical trials of vaccines as well as basic research on bioterrorism agents and infectious diseases. Director Alan Barbour says the center will provide “immediate research capability” in case of an outbreak. —JOCELYN KAISER

Stem Cell Institute Faces Possible Vote

California legislators were expected to vote this month on a measure that would tighten conflict-of-interest rules for advisory bodies to the state’s new institute for regenerative medicine (CIRM). A committee of overseers at the nascent stem-cell institute moved last week to consider toughening its policies in hopes of heading off the legislation, seen as potentially limiting the participation of experts. If passed by two-thirds of both houses, the proposed constitutional amendment will go before voters in November.

Meanwhile, amid pending lawsuits and financial uncertainty, CIRM this week received \$5 million from San Francisco sound pioneer Ray Dolby to help it get started. The institute is also pursuing a \$100 million loan.

—CONSTANCE HOLDEN

parasite, and a mosquito species called *Anopheles stephensi*, the group found that even in surviving mosquitoes, the fungus severely hampered the parasites' ability to develop and mature. "That looks like an important extra benefit," says Wendy Gelernter, a biopesticide consultant at PACE, a company in San Diego, California. In addition, both teams have data suggesting that a fungal infection dampens mosquitoes' appetite for blood meals, making them less

likely to pick up parasites in the first place.

Ken Neethling, production director for BCP, a South African company specializing in biopesticides, says his firm may explore the malaria biocontrol strategy commercially; others are interested as well, Thomas says. For now, both teams plan to tinker with the sprays' formulations to see if they can improve infection rates. One key problem: The spores start losing their infectiousness in a matter of weeks. If that can't be solved, the

spray would have to be applied over and over. (Pesticides, in contrast, can last a year or longer.) That could be "a near-fatal flaw," says Lines.

Still, these are problems well worth delving into, says Norbert Becker of the German Mosquito Control Association in Waldsee, Germany. As long as malaria kills more than a million people every year, he says, "every new strategy is appreciated."

—MARTIN ENSERINK

GENETICS

In Voles, a Little Extra DNA Makes for Faithful Mates

Prairie voles are renowned for being faithful mates, but some individuals are more faithful than others. The difference may lie in their so-called junk DNA.

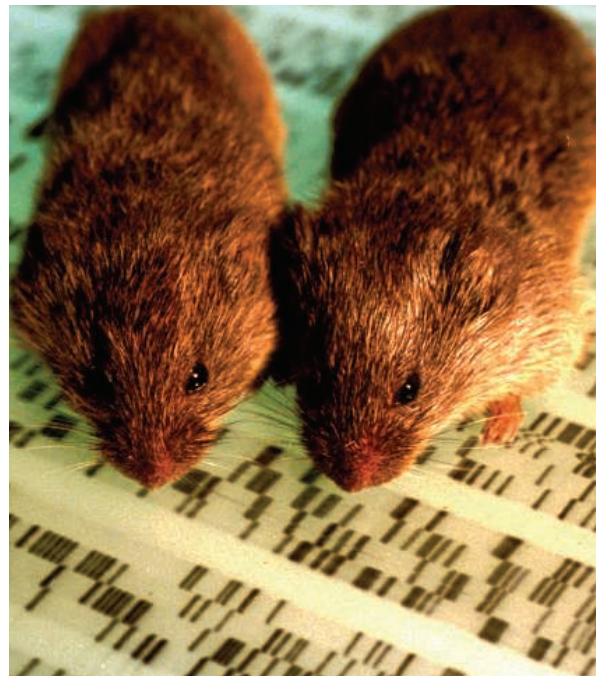
On page 1630, Elizabeth Hammock and Lawrence Young of Emory University in Atlanta, Georgia, report that fidelity and other social behaviors in male prairie voles seem to depend on the length of a particular genetic sequence in a stretch of DNA between their genes. The longer this repetitive sequence, or microsatellite, the more attentive males were to their female partner and their offspring. Those with shorter microsatellites neglected their mates and pups, at least to some degree.

Although there's no evidence that human infidelity or poor parenting stems from similar variations, Hammock and Young, as well as other researchers, have begun to explore whether microsatellites can account for behavioral differences between people and primates such as chimps and bonobos. The new study's results "will force us to think about these variations in so-called junk DNA and how [they] make for changes in behavior," says Scott Young (who is not related to Lawrence Young), a neuroscientist at the National Institute of Mental Health in Rockville, Maryland.

Microsatellites are genetic stutters, usually just two or four bases long. There can be hundreds of these repeats in a row. They can befuddle the cell's DNA replication machinery, so the number of repeats within one may rise or fall from one generation to the next. And when they are in regulatory regions for genes, their changing lengths may affect the activity of those genes. This can have rapid evolutionary implications, Scott Young points out.

In the mid-1990s, researchers discovered a key microsatellite difference between prairie voles and their more promiscuous cousins, such as the meadow voles. Prairie voles have longer microsatellites near the gene encoding a receptor (*V1aR*) for the brain chemical vasopressin, and as a result they make more of the receptor than do meadow voles. This was

the first clue that these sequences may influence social behavior. Last year, Young's team strengthened the connection when they caused meadow voles to emulate the faithful ways of prairie voles by adding extra copies of the *V1aR* gene to a portion of their brains



Honey, I'm home. Sequencing studies revealed that the amount of junk DNA affects how male voles treat their mates.

(*Science*, 7 January, p. 30). "The vasopressin system is likely to be a major player in emotional and cognitive aspects of social bonding," comments Rainer Landgraf, a neuroscientist at the Max Planck Institute of Psychiatry in Munich, Germany.

Now, Young and Hammock, originally one of Young's graduate students and now at Vanderbilt University in Nashville, Tennessee, have found that variations in *V1aR*-associated microsatellites among individual prairie voles influence expression of the gene and overall behavior. They paired and bred voles with long microsatellites and found that the resulting males spend more time licking and grooming

their pups than did males with short microsatellites. They also placed males in cages with a female, allowing 18 hours for them to bond, then added a new female. Males with longer microsatellites spent more time with their partners than did those with shorter microsatellites.

Taken together, the results "help create a picture of some of the building blocks that allow for the evolution of different levels of social behavior," says Catherine Marler of the University of Wisconsin, Madison.

Evan Balaban, a neuroscientist at McGill University in Montreal, Canada, isn't convinced, however. He argues that, instead of simply showing correlations between microsatellite length and a behavior, the researchers should do transgenic experiments to establish that microsatellites were truly responsible for the different behaviors. Furthermore, "the behavioral effects are small," Balaban adds.

Undeterred, Hammock and Young have already noted connections between *V1aR* microsatellites and primate behavior. Other researchers

have associated the length of one of the four microsatellites in the human version of the gene with autism, a disorder of social interactions. In the chimp, this same microsatellite is 360 bases shorter, Hammock and Young note. But in bonobos, which are less aggressive than chimps and form more humanlike social bonds, the microsatellite is nearly identical to the human counterpart.

Even Balaban thinks such intriguing observations deserve follow-up. "Hopefully," he says, "[this will] direct people's attention to studying the role that variation in the control of the regulation of genes plays."

—ELIZABETH PENNISI