Butterfly may use flowery stepping-stones

A new study suggests that creating habitat corridors, a commonly proposed strategy for conserving species, would be the wrong approach for the rare Fender's blue butterfly. Rather than cruising down a solid highway of flowers to reach new sites, the butterflies might prefer to hopscotch between flower patches.

The Oregon butterflies live in patches of lupine flowers, but they often stray. Outside their preferred habitat, they pick up speed, moving six to eight times faster than when they meander among lupines, says ecologist Cheryl B. Schultz of the University of Washington in Seattle. Her findings indicate that, to conserve the rare butterfly, "a traditional linear corridor is inappropriate."

"It is unlikely that the butterflies would stay in a narrow corridor," she reports in the April CONSERVATION BIOLOGY. She therefore recommends a series of "habitat stepping-stones."

Some biologists have suggested that habitat corridors are important tools for conservation. Such links would provide a thin green line connecting the remaining fragments of a once-continuous habitat. Much as cars follow interstate highways between cities, animals that are loath to leave their favorite environment would travel such botanical roadways to reach other regions of appropriate habitat.

Schultz's conclusions "call into question this dogma about corridors," says ecologist Susan Harrison of the University of California, Davis. "There's a huge unanswered question about whether corridors work. . . . One big lack has been careful studies of how organisms move in real, patchy environments."

Ecologist Paul Beier of Northern Arizona University in Flagstaff says there probably isn't one general answer but adds that some animals clearly need corridors. The mountain lions he studies "can't walk through industrial parks."

Schultz's motivation for the study was a proposal to create a corridor between three areas of butterfly habitat near Eugene, Ore. Although there have been other studies of butterfly populations, few focused on whether the insects will use corridors, she says (SN: 4/4/98, p. 214). "My question was, 'Would a butterfly actually use a corridor if we took the time and money to create it?'"

To answer that question, Schultz chased butterflies. She and her assistants watched the insects' behavior inside areas with lupines and outside such prime habitat. The researchers dropped a small flag on the ground every time a butterfly alighted and every 20 seconds while the insect was in flight. For each flag, they recorded the time and what the butterfly was doing. On other days, the team measured how much time the butterflies spent flying.

Schultz calculated how far a Fender's blue butterfly, which is about 1.5 inches long, moves in its lifespan of about 9.5 days. When outside a lupine-rich spot, males might move as far as 2.4 kilometers and females might travel 1.7 km. A butterfly that stayed within a lupine patch would cover 1 meter or less.

Before European settlement of the prairie, Schultz says, lupine patches were probably only 0.5 km apart—plenty close for the butterflies. Now, pieces of prairie that harbor lupines are separated by 3 to 30 km. The next phase, she says, is to develop restoration techniques that will create the stepping-stones that the butterflies need. —*M.N. Jensen*

A female Fender's blue butterfly.

Ulcer bacterium's drug resistance unmasked

By any measure, the drug metronidazole is a potent disease fighter. Also known as Flagyl, MetroGel, and Protostat, it knocks out the bacterium *Helicobacter pylori,* as well as microbes that can cause amoebic dysentery, other intestinal ailments, and some vaginal infections.

In 1985, scientists fingered the spiral-shaped *H. pylori* as the agent responsible for stomach ulcers and have since relied chiefly on metronidazole in combination with other drugs to battle the illness. *H. pylori* has proved to be a worthy adversary, however, developing widespread resistance to metronidazole.

Canadian and U.S. researchers have now discovered how some *H. pylori* infections succumb to metronidazole and others withstand its onslaught.

In a search through *H. pylori* genes taken from individuals in Peru and Lithuania, where the bacterium is common, the scientists identified genetic sequences that make the microbe drug-resistant and differentiated them from normal sequences.

One of the enzymes made by normal *H. pylori* converts metronidazole into a harsh chemical called hydroxylamine—a protein-busting, DNA-damaging terror that causes mutations in *H. pylori*'s own genes. The besieged bacterium fights furiously to stem this DNA destruction. "If the rate of DNA damage is higher than the rate of repair, the cell dies," says microbiologist Avery C. Goodwin of Dalhousie University in Halifax, Nova Scotia.

Bacteria that escape the chemical reaper often have genetic mutations. If the gene encoding the enzyme that acts on metronidazole is damaged or destroyed, the bacterium becomes resistant to the drug. He and his colleagues report the findings in the April 14 MOLECULAR MICROBIOLOGY.

"This goes a long way toward helping us to understand metronidazole resistance in *H. pylori,*" says Martin J. Blaser, a microbiologist at Vanderbilt University in Nashville. Having identified the enzyme's critical role, researchers may eventually develop a compound that restores susceptibility, he says. In addition, the discovery of this novel resistance pathway, which is based on a disabled or missing enzyme, may provide insights into *H. pylori*'s unusual metabolic structure, which is neither entirely aerobic—oxygen-using—nor anaerobic.

Roughly one-third of people in the United States and Western Europe harbor *H. pylori*, and the proportion is higher in Asia and developing countries. Resistance to metronidazole among infected people is estimated at 40 percent in Europe and more than 70 percent in the developing world.

Although no biological connection has been demonstrated, epidemiological studies have linked *H. pylori* and stomach cancer. The bacterium may act by weakening the stomach lining's defenses against gastric acid, says study coauthor Douglas E. Berg, a microbial geneticist at Washington University in St. Louis. This can lead to chronic inflammation and a precancerous condition. Other research suggests that *H. pylori* may also disrupt programmed cell death, a natural check on runaway cell growth.

The researchers speculate that hydroxylamine, which can cause mutations in human as well as bacterial DNA, could contribute to the eventual development of gastric cancer. Delivering such a carcinogen to the cells of the stomach lining, Berg says, "strikes me as a very sobering and slightly scary type of thing."

Although this research is "fascinating," says Miklós Müller, a biochemical parasitologist at Rockefeller University in New York, the cancer connection to metronidazole is unlikely because hydroxylamine is short-lived in the stomach. Researchers looking for increased cancer risks linked to metronidazole haven't found convincing evidence, he says. —*N. Seppa*