Genetic sleuths explain insects' resistance

Coffee drinkers, take note! The coffee bean's number one enemy, a bug that practices incest as a way of life and goes by the name of Hypothenemus hampei, or the coffee berry borer, can now outwit its archenemy, the pesticide endosulfan. Although investigators believe they have figured out the insect's secret genetic weapon, they have yet to defuse it.

In 1989, scientists discovered that many coffee berry borers on the South Pacific Island of New Caledonia tolerate endosulfan, the most common insecticide used against them. Unlike many other chemicals, this fumigant reaches inside the beans, where the insects lay their eggs. Whether coffee berry borers elsewhere have also become resistant isn't clear.

The secret of the bugs' success lies in their unusual genetic makeup and breeding habits, assert Luc O. Brun of the Institut Français de Recherche Scientifique pour le Développement en Coopération in Noumea, New Caledonia, and his colleagues in the Oct. 10 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

In a previous study, the researchers described a genetic mutation that enables the pests to withstand the insecticide. The mutation has a good chance of spreading through the population because the few males born in each brood mate only with their sisters.

However, there's more to the mutation's success, the team argues.

Most mammals and insects are diploids-they inherit a set of chromosomes from each parent. Haploids, including ants, wasps, and bees, inherit only their mother's chromosomes. Haplodiploids have diploid females and haploid males.

The coffee borers are functional haplodiploids. Although each sex inherits both sets of chromosomes, at some point in male development the father's set appears to shut down. Males function from then on using only their mother's



A female coffee berry borer makes her way into a red, unripe coffee bean to deposit her eggs.

genes, and their sperm contain only maternal genes, the team contends.

So if a male coffee berry borer inherits a resistance gene from his mother, all of his offspring will have a copy of it, explains coauthor Jeff Stuart of Purdue University in West Lafayette, Indiana.

If a diploid female inherits a resistance gene from her father, a susceptible gene from her mother, and then mates with a diploid male with two resistance genes, half of her progeny will have both resistant and susceptible genes and the other half will have only the resistance genes, savs Stuart.

Other studies have not examined the

genetics of resistance in a functional haplodiploid, says Richard W. Beeman of the U.S. Department of Agriculture's Agricultural Research Service in Manhattan, Kansas. "The resistance gene provided an easy way to follow the chromosomes," he says. Functional haplodiploidy may serve as a step in the evolution of true haplodiploidy, Beeman and the authors assert.

The flightless male borers are tough. A male with only one resistance gene and a female with two survive equally well.

'In view of the past global dispersal of H. hampei . . . the appearance of endosulfan-resistant lines in New Caledonia may represent a serious threat to the international coffee industry," the authors warn.

Outdoor carbon monoxide: Risk to millions

In the United States alone, an estimated 3 million individuals-most of them over the age of 65-suffer from congestive heart failure, the inability of the heart to pump out all of the blood that returns to it. A new study now indicates that even federally permissible levels of carbon monoxide, a common air pollutant, can aggravate this life-threatening condition enough to send its victims to the hospital.

"It was really striking," says Robert D. Morris of the Medical College of Wisconsin in Milwaukee, who led the study. "In every city we looked at, there was an elevation in heart-failure admissions on days that carbon monoxide went up. Moreover, this effect appeared regardless of the extent to which the patients had also been exposed to other major gaseous pollutants-nitrogen dioxide, sulfur dioxide, or even ozone.

Carbon monoxide reduces the blood's ability to carry oxygen. Previous studies have shown that high exposure to the pollutant for a short period lowers the exertion needed to trigger chest pain (angina) in susceptible individuals—the health impact upon which current federal limits are based. However, Morris' team found that the carbon monoxide concentrations linked to the increases in hospital admissions could be lower than those needed to bring on angina.

Morris and his colleagues obtained daily records of meteorological conditions and outdoor gaseous air pollutants in seven U.S. cities-Chicago, Detroit, Houston, Los Angeles, Milwaukee, New York, and Philadelphia. They compared the information with 4 years of Medicare data on hospital admissions for heart failure in these cities. Only carbon monoxide readings correlated consistently with hospitalizations, they report in the October AMERICAN JOURNAL OF PUBLIC HEALTH.

Approximately 11.6 million people live within the 73 U.S. areas that year after year exceed federal carbon monoxide

limits (8 parts per million for 9 hours or 35 ppm for 1 hour), notes Dave Ryan, an Environmental Protection Agency spokesman. But the problem isn't limited to those areas, because "there appears [to be] no threshold for this effect,' Morris notes. Even on days that fell well below EPA's limits, he says, his team charted a 20 to 40 percent increase in heart failure admissions for every 10 ppm increase in the gas.

Overall, between 2 and 11 percent of hospital admissions for congestive heart failure, depending on the city, could be traced to carbon monoxide. Those roughly 3,300 such incidents each year cost an estimated \$33 million.

Nationally, the tally could run many times that, says Joel Schwartz of the Harvard School of Public Health in Boston. While this constitutes a minor proportion of heart disease, it's not a minor environmental risk, he points out in an accompanying editorial. Hazardous waste sites and toxic air pollutants that pose "much lower" risks are regulated, he notes.

"As the population ages," Morris adds, "the subset that is vulnerable [to carbon monoxide] will only increase.

Though outdoor carbon monoxide results almost exclusively from motor vehicles, these sources spew other toxic pollutants as well, including dust-sized particles. Recently, Schwartz and Morris reported data from Detroit indicating that these easily breathed particles may be responsible for some share of the city's hospital admissions for congestive heart failure (SN: 7/1/95, p.5). Again, Morris says, "this carbon monoxide effect tended to be independent of the other pollutants [fine particles].

Morris concludes that any lessening of carbon monoxide pollution will show health benefits. To Schwartz, the new findings on congestive heart failure also suggest that "it is time to reevaluate the basis for regulating carbon monoxide.'

— J. Raloff

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