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Unveiling a Six-Legged Equine Assassin: Mare Reproductive Loss Syndrome

by Joan Capuzzi Giresi, C’86 V’98

It was Derby week and all was supposed to be festive in Kentucky. But not so in 2001: Scores of future racers, still in utero, were dying mysteriously. And Lenn R. Harrison, V’67, director of the Livestock Disease Diagnostic Center at the University of Kentucky College of Agriculture, was left scratching his head.

“We weren’t coming up with any diagnosis,” Harrison remembers. “It was a real ‘What is it?’ situation.”

The problem became evident one sad April day when the center—which normally posts two or three aborted fetuses daily—received a staggering 35. Many more would follow. In fact, over the next three years, Mare Reproductive Loss Syndrome (MRLS), characterized by early fetal loss, late-term abortion, and foals born weak and dying, would cause thousands of mares to abort.

Except for spotty cases of pneumonia, the fetuses had no telltale lesions. The mares generally remained healthy—even foaling normally the following season. A number of cases of unilateral uveitis, pericarditis, and encephalitis were seen in nonpregnant stablemates.

Harrison considered several etiologies for MRLS, but quickly ruled out leading contenders like fescue toxicity—a major cause of late-spring equine fetal losses in Kentucky—caused by mycotoxins and cyanide.

Suspecting an infectious agent was at work, neighboring states considered closing their borders to horse shipments. Harrison met with the Kentucky governor and fielded calls from the FBI and the Justice Department about the possibility of contagion associated with agriterrorism.

But infectious etiology was also shelved early on: MRLS was hardly limited to the Thoroughbred industry of central Kentucky. At its far reaches, the epidemic—which afflicted 17 breeds—extended to remote farms in the eastern mountains of Kentucky that had no contact with Lexington’s Thoroughbred farms. And bacteria cultured from the fetuses, which included two rare strains of Streptococcus and Actinobacillus, were considered symbiotic organisms.

But as Harrison walked the affected farms of Lexington (where some 10,000 Thoroughbred mares foal each season) and elsewhere, there was something he could not dismiss:

“The caterpillars were piled so thick you could hardly step on the ground. Even in the food buckets, they were inches high,” says Harrison.

Kentucky was experiencing an eruption of the eastern tent caterpillar, Malacosoma americanum—an event that occurs every decade or two. The idea that caterpillars could somehow produce fetal losses, however, was considered “wild” by many, Harrison recalls.

Yet in exposure studies that soon followed, pregnant mares housed with the caterpillars aborted within days. Just how the caterpillars, which build their large, tented nests in the wild cherry and ornamental crab apple trees common on horse farms in the area, cause fetal harm is not known for sure.

Harrison and his colleagues theorize that the caterpillars produce a bio toxin deadly to the developing fetus. Others believe the fetus becomes infected with bacteria that enter the mare’s bloodstream via tissue penetration of septic barbed setae—or hairs—of ingested caterpillars.

The MRLS outbreak peaked the following year, 2001, to about 3,400 cases. By spring 2002, when the evidence was pointing toward the eastern tent caterpillar as the causal agent, many farms began cutting down their cherry and crab apple trees, and spraying insecticides.

Nevertheless, by the time the epidemic ran its course, some 4,000 mares had aborted, and economic losses had reached about $500 million. The emotional impact was also severe.

“I’ll never forget the expressions on the faces of horse farmers walking in with an aborted fetus and then going back to their farm only to return soon after with another fetus and then another,” says Harrison, who was called to perform necropsies around the clock during the disease outbreak.

After completing a pre-veterinary curriculum at Pennsylvania State University, Harrison began veterinary school. While at Penn, he worked in the genetics lab of Dr. Donald Patterson, professor of medical genetics, collecting samples.

Following graduation, Harrison returned to Penn State for a general residency. There, he sharpened his teeth in large-animal work and refined his surgical technique. Next, he moved to nearby Mifflin County to start a mixed-animal practice with a concentration in equine and bovine medicine.

But after just a few years, he developed a severe allergy to Staphylococcus that forced him to seek a career change.

Harrison soon learned of a job opening in the veterinary diagnostics lab at the University of Georgia. Although his pathology experience was limited, he landed the position. Working side-by-side with board-certified pathologists, Harrison took—and passed—his pathology boards in 1981.

Although he missed private practice, Harrison found that being on the other side of the diagnostics fence held a special appeal: It was an opportunity to assist veterinarians in the field to do their jobs.

“I get to find unexplained reasons for why animals are sick that can’t be learned any other way,” he says.

In 1991, Harrison, a lifelong horse enthusiast, took the helm at the University of Kentucky’s veterinary diagnostic center. One of two veterinary labs in Kentucky, it processes 60,000 accessions annually, about 60 percent of them equine.

As Harrison reflects back on MRLS, the most widespread epidemic he has dealt with in his career, he talks about ways to prevent—or at least foresee—Mother Nature’s next one–two punch. His latest initiative is introducing epidemiology into his lab.

“I have a strong feeling that epidemiology within a diagnostic lab can recognize disease trends like nothing else,” says Harrison, who recently hired an epidemiologist to work alongside him and his staff of veterinary pathologists, microbiologists, and a virologist.

Perhaps Kentucky’s horses will now be better protected against the next slew of caterpillars or moldy pasture or virulence.