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THE KENTUCKY 2001 MARE REPRODUCTIVE LOSS SYNDROME: OVERVIEW AND PRELIMINARY TOXICOLOGICAL APPROACHES

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During 2001, Central Kentucky experienced an epidemic of Early and Late Fetal Losses (EFL/LFL), together called Mare Reproductive Loss Syndrome (MRLS). LFL (n=550) began in late April, peaked May 5th, and declined rapidly. EFL (n=1,750) was identified April 26th and followed a similar time course. Also occurring were micro-epidemics of pericarditis (n=60) and unilateral pan-opthalmitis (n=50). These syndromes appeared simultaneously, and virological analyses were negative, suggesting a toxic environmental point source cause. The estimated economic loss was $336M.

EFL occurred in 30-100 day fetuses; they palpated normally, but ultrasound showed no heartbeat and cloudy, echogenic amniotic fluid, which was often followed by expulsion of the fetus. Two micro-anaerobes, Actinobacillus and α-Streptococcal species, were recovered from many LFL. LFL also showed characteristic pathology, including funisitis and pneumonic changes.

A rigorous epidemiological study established strong positive correlations between early breeding, exposure to Black Cherry trees, Eastern Tent Caterpillars (ETC), waterfowl and MRLS. Absence of caterpillars and supplementary hay were protective.

Fifty (50) sequential analyses for nitrate/nitrite were negative. Contemporary pasture samples (113), some from heavily fertilized pastures, were negative for nitrate/nitrite. Some pasture samples (508) yielded evidence of estrogenic activity. No cases showed clinical signs of estrogenic activity, and experimental administration of estrogens to small numbers of horses yielded no clinical signs of EFL.

Pathological reports and preliminary toxicological analyses suggested cyanide, presumably from Black Cherry trees or other sources, as a possible contributing or causative agent. Numerous cyanide analyses were performed, and preliminary infusion of cyanide into a small number of pregnant mares failed to produce clinical signs of EFL.

Approximately 400 tests were performed for estrogenic and other mycotoxins. These results indicated that MRLS is unlikely to be associated with known Fusarium mycotoxins on pasture plants. Preliminary work with mycotoxins in a small number of pregnant mares failed to produce clinical signs of EFL. Fifty (50) urine samples were analyzed for ergot alkaloids; ergot alkaloids were present in tall fescue but not in other species, but the presence of ergot alkaloids showed no correlation with MRLS. Hemlock alkaloids cause fetal malformations; 13 selected urine samples showed no significant concentrations of hemlock alkaloids. At this time each of these and other hypotheses must be considered as mechanisms to explain MRLS, with no compelling evidence to support any individual hypothesis.

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