Proceedings of the First Workshop on Mare Reproductive Loss Syndrome

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Summary

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MARE REPRODUCTIVE LOSS SYNDROME (MRLS) SEEMS TO BE strongly associated with the caterpillars. If you put the caterpillars near horses, or in horses, or, as was described here this morning, if the horses eat the caterpillars—and it seems that young horses may do more so than older horses, which may be associated with a learned response—you get problems.

On the other hand, muzzling seems to be very effective, which has been readily apparent in this 2002 season. This information has come back from the monitoring program, and we heard it again this morning from Dr. Riddle. So the bottom line is: if you can keep the caterpillars out of the mouth/intestinal tract of the horse, then we would seem to have gone a long way toward solving our problem.

When caterpillars do get into the oral cavity, what we pick up throughout the affected horses are bacteria that are apparently mouth commensals, the *Actinobacillus* and the non-hemolytic *Streptococcus* species. These bacteria, normally mouth residents, suddenly start to appear elsewhere in both pregnant and non-pregnant horses. They appear in the early and late fetal losses (EFL/LFL), and they also appear in the pericardial sac: we don't know what appears in the eye because we've not done bacteriology in the eye. Actinobacillus also appears in the brain: Drs. Sebastian and Harrison have reported three cases of Actinobacillus encephalitis occurring in or about the time of MRLS. When the caterpillars appear, something goes through *all* caterpillar-exposed horses, but it is in the pregnant mare that we see by far the most dramatic effects.

So, what happens when the caterpillars get into the horse's mouth? I like Dr. LeBlanc's analogy. Dentists working in my mouth put me on prophylactic antibiotics immediately because I have a heart murmur, and they don't want to risk a bacterial vegetative endocarditis. Likewise, something happens when horses are exposed to caterpillars in that we suddenly have oral commensal bacteria appearing shortly thereafter at multiple locations in the body. (In this regard, Dr. Sebastian has since drawn my attention to an un-referenced citation in an early edition of Blood and Henderson noting that mouth lesions in horses are associated with "hairy caterpillars" [1]).

We have been to some extent overwhelmed by the EFL and LFL—these are what has drawn attention to this whole problem—but there are also related things going on at a much lower rate in all Central Kentucky horses exposed to caterpillars.

Dr. Bernard isn't here, but I understand that he has shown that if you take a caterpillar and separate the exte-

rior from the interior, the fetal losses appear to be associated with the integument, the outside of the caterpillar. This and other considerations drove the first mouse setal experiments that Dr. Sebastian has described to you. The setal hypothesis started with the thought that perhaps there was a toxin associated with the setae. Then we backed up and said that perhaps it's simply the setae themselves facilitating the movement of bacteria into the blood. We wondered about the setae themselves becoming little septic emboli in the body and carrying little quanta of infected material to various locations in the body. The infected material would be contaminated with bacterial commensals from the point at which the setal fragments entered the body of the horse. In MRLS cases occurring in the field, these would be the mouth commensals, the Actinobacillus and the non-hemolytic Streptococcus species. In experimental MRLS, where we delivered the caterpillars into the stomach by nasogastric tube, the bacterial picture is different, apparently consistent with the different point of entry for the bacteria.

Tissue localization of such septic emboli would not cause significant problems in most areas of the body, where the immune system can handle it, but some areas of the body may be particularly susceptible, such as the fetal membranes, and perhaps the eye, where the results of such effects are easily visible, and also the pericardial sac.

So, let me just simply say: Do we need a toxin? Well. we don't have a candidate toxin. Dr. Whitwell very kindly asked the toxicologists here to nominate a toxin, and one wasn't forthcoming. My sense at this time is that we need to look carefully at the link between the bacterial commensals in the mouth and how the outside of the caterpillars (and those barbed setae) may facilitate distribution of mouth commensals to distant locations in the body. At this point, I am far from persuaded that there's a classic toxicity mechanism involved, and I am a toxicologist, more or less, by training.

Reference

 Blood, D. C.; Radostits, O. M., and Henderson, J. A. Veterinary medicine: a textbook of the diseases of cattle, sheep, pigs, goats and horses. 6th ed. London: Bailliere Tindall; 1983, p. 780.

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