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Tracking a Killer: On the Trail of a Fatal Liver Disease

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Tracking a Killer

ON THE TRAIL OF A FATAL LIVER DISEASE

On February 23, 1983, a comatose three-day-old Standardbred foal was admitted to the George D. Widener Hospital for Large Animals at New Bolton Center. The filly, healthy and normal for the first forty hours of its life, had suddenly become uncoordinated and then comatose. Tests indicated that its liver was not adequately functioning.

It was the first of eight foals with liver failure seen at New Bolton Center. Practitioners in other parts of the country also reported similar cases. During the spring of 1983 sixty foals in eleven states succumbed to liver failure caused by an unidentified disease.

Researchers at New Bolton Center and at other institutions launched a concentrated effort to determine the cause of this apparently new liver disease. A team of clinicians and research investigators at the New Bolton Center conducted extensive laboratory and pathologic studies as well as epidemiologic surveys. It was found that foals with the disease had been healthy and normal at birth and that the first signs of liver failure had appeared between the age of two and five days when the foals became disoriented, weak, and jaundiced. Some foals were hypoglycemic, others were hypothermic, and some had elevated heart rates when admitted to the hospital.

Pathologic studies showed the livers of these animals to be shrunkened and damaged. It was also found that the thymus glands were smaller than in healthy animals, and that microscopic lesions had occurred in the brains. The tissue and blood samples were examined for evidence of equine herpes virus 1 and viral hepatitis B, but no evidence of either disease was found. During further studies the researchers also ruled out foal septicaemia, neonatal isoerythrolysis and Tyzzer's disease as the possible causes of the liver failure.

The foals seen at New Bolton Center as well as the foals seen elsewhere had received a live culture product reported to enhance intestinal flora shortly after birth. The researchers examined the substances the foals had received shortly after birth as it seemed most likely, based upon epidemiologic evidence, that a hepatoxin administered to the foals shortly after birth was responsible for this new and unusual syndrome. The foals seen at New Bolton Center as well as the foals seen elsewhere had received a live culture product reported to enhance intestinal flora shortly after birth. The widely used product was given on the first and third day of life and this may explain why some cases of liver failure occurred at two and three days of age and others at the age of five days.

The product contains Aspergillus oryzae which may produce a potential hepatotoxic. The product also contains iron fumarate, another known hepatotoxic. It was found that the foals which died from the disease in 1981 also received an identical product. The product has been given to hundreds of foals without ill-effect. This could suggest that individual vials of the product are to blame. The sporadic incidence could also be related to the age of the foal at the time of product administration.

Universities are continuing the search for the exact cause of the liver failure in young foals. Epidemiologic surveys and toxicologic and immunologic tests are being conducted to determine the precise etiology and pathophysiology of this fatal disease.

The studies at New Bolton Center are being conducted by Dr. Thomas J. Divers, Dr. Helen Acland, Dr. Lawrence Glickman, Dr. Diane Gunson, Dr. Jonathan Palmer, Dr. Angeline Warner, Dr. Robert Whitlock, and Dr. Dennis Hill. The study is made possible by a grant to the Equine Research Fund at New Bolton Center, University of Pennsylvania.

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