Enzootic ataxia is a progressive neuropathy of young copper-deficient animals. This report describes enzootic ataxia in 3 goat kids from a small Louisiana goat herd.

The herd consisted of 10 crossbred does that were housed on a half-acre dirt paddock. The goats were fed shelled corn and hay and grazed in an essentially bare paddock during late autumn and winter. No mineral mix was available to the goats. Five apparently normal healthy kids were born to the herd in late winter. Four of the 5 kids developed hind limb ataxia between 3 and 4 weeks of age. Clinical signs in the affected kids progressively worsened over a 2-month period. One kid became recumbent and died. Another kid was euthanized and necropsied in the field. The tissues were sent to the Louisiana Veterinary Medical Diagnostic Laboratory where a histopathologic diagnosis of copper deficiency was made. Two weeks later, the 2 remaining kids were referred to the School of Veterinary Medicine teaching hospital at Louisiana State University.

Physical examination of the 2 affected kids (1 male, 1 female) revealed them to be bright and alert with normal vital signs. The animals were in good flesh and appetite was excellent. Neurologic examination demonstrated symmetrical ataxia in both hind limbs of both kids. The kids were ambulatory and could walk in a straight line with minimal stumbling but stumbled and fell when trying to negotiate turns. Knuckling was observed in both hind limbs and placing was abnormal. Clinically, cranial nerves were not affected. Peripheral stretch reflexes were normal in front and hind limbs, strength was maintained, and deep pain was present in all limbs of both goats.

A cerebrospinal fluid sample aspirated from 1 kid had a slight increase in white blood cell (WBC) count (13 WBC/mm³ corrected to 10 WBC/mm³; normal = 0.4 WBC/mm³) and a slight increase in protein concentration (43 mg/dl; normal = 0.39 mg/dl). Cytologically, the WBC’s were 90% mononuclear cells. These findings are compatible with a diagnosis of mild nonsuppurative inflammatory and/or degenerative processes.

Serum samples from both kids were submitted for measurement of antibodies to caprine arthritis and encephalomyelitis virus (CAEV) and from 1 kid for copper determination. Neither kid had a detectable titer to CAEV and the serum copper concentration was 0.27 ppm (normal = 0.80-1.20 ppm). 9

Histologically, cerebellar lesions consisted of patchy degeneration and loss of the Purkinje cells and reactive proliferation of the Bergmann glial cells. These changes were seen both in the vermis and in the hemispheres. Damaged Purkinje cells showed vacuolation, chromatolysis, and hyalinization of their cytoplasm. Similar lesions were observed in a few neurons of the vestibular nucleus and reticular formation of the medulla oblongata. A few blood vessels of the medulla had perivascular lymphocyte cuffs. The changes observed in the spinal cord presented a characteristic bilateral symmetrical pattern affecting both nerve cells and fibers. Lesions were consistently present in the white matter of the cervical, thoracic, and lumbar portions of the spinal cord and were more prominent along the ventromedial fissure, with less severe changes in the lateral funiculi (Fig. 1). These lesions consisted of deficiency of stainable myelin, degeneration and loss of myelinated axons, and lack of inflammatory cells (Fig. 2). The neuronal changes seen in the ventral horns and in the dorsal nucleus (Clarke’s column) of the spinal cord included cytoplasmic swelling and chromatolysis varying from eccentric location of the nuclei to, in some cases, disappearance of the nuclei. Those cells were rounded with homogeneous pink cytoplasm (Fig. 3). Some neurons were shrunken and eosinophilic, and swollen axons were observed in the proximity of damaged neurons. Spinal nerve roots and ganglia were not affected. A section of sciatic nerve showed Wallerian type of degeneration and fragmentation of myelinated axons with formation of digestion chambers. No relevant lesions were observed in other examined tissues.

Fresh liver copper concentrations from the 2 kids were 6.0 and 3.4 ppm (wet weight basis). Formalin-fixed liver from the kid that had been euthanized earlier had approximately 6.9 ppm copper (corrected for leaching of copper into formalin). 2 Copper concentrations below 25-150 ppm (wet weight basis) in goat liver are considered to be deficient. 9 Spring paddock grass was not a good estimate of what the pregnant does were grazing in the autumn and winter and was not collected for analysis. The corn contained 3.0 ppm copper.

Neurologic disease resulting from copper deficiency is important in lambs in Australia and other major sheep producing areas of the world but is rarely reported in North American lambs. There are only 2 previous reports of this disease in goats in the United States. 4,8

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Enzootic ataxia in Louisiana goat kids

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Two clinical neurologic disease syndromes associated with copper deficiency are referred to as congenital (swayback) and delayed-onset (enzootic ataxia) syndromes.\(^1\) Both forms have been reported in sheep and goats. In the congenital form, the condition develops in utero and symptoms are present at birth. Affected animals may be totally recumbent or ambulatory with severe ataxia.\(^3\) The major clinical signs include depression, head shaking, trembling, and ataxia, and most affected animals die soon after birth.\(^3\) The prominent lesion is absence or destruction of the white matter of the cerebral hemispheres.\(^5\) The changes in the white matter occur as small foci of gelatinous softening, or cavitation and collapse of the cerebral hemispheres.\(^5\) The large motor neurons of the red and vestibular nuclei are commonly rounded and swollen and usually show chromatolytic changes.\(^6\) In addition, demyelination in the motor tracts (ventral and dorsal portions of the ventrolateral column) of the white matter of the spinal cord has been reported.\(^5\)

The delayed form (enzootic ataxia) develops after birth. Animals appear clinically normal at birth and develop signs of the disease from 1 week to 6 months of age.\(^7\) The delayed form is characterized by incoordination, ataxia, and posterior paresis. Lesions are limited to the large neurons of the brain stem and spinal cord.\(^6\) However, many goats with enzootic
ataxia also have well defined lesions in the cerebellum, including patchy cerebellar hypoplasia, necrosis and loss of Purkinje cells, and depletion of the granule cell layer.\textsuperscript{11}

Swayback and enzootic ataxia may result from either primary or secondary copper deficiency. Primary copper deficiency is caused by a diet that is low in copper. Secondary copper deficiency results from dietary composition, which determines the proportion of the dietary copper that is absorbed. This proportion can vary widely so that the actual copper intake has little or no bearing on whether or not functional copper deficiency will develop.\textsuperscript{12} The three important considerations of dietary composition that determine dietary copper absorption include the type of food, the mineral composition of the foodstuff, and the interaction between food type and mineral composition.\textsuperscript{12} The type of foodstuffs from which copper is well absorbed are those low in fiber such as cereals.\textsuperscript{12} Copper is poorly absorbed from fresh herbage, and conversion of the grass to hay or silage improves copper availability, though rarely to the levels of nonfibrous crops.\textsuperscript{10} Copper deficiency is, therefore, a problem of grazing animals because of the poor availability of copper in grass.\textsuperscript{12} The mineral composition of the foodstuff can cause major effects on copper availability.\textsuperscript{12} Elevations of dietary molybdenum and total sulfur can interfere with proper copper utilization.\textsuperscript{10} The influence of dietary composition on copper absorption is further complicated by interactions between the food type and the mineral composition.\textsuperscript{12}

The critical factor in the pathogenesis of swayback and enzootic ataxia is reduction in the activity of the copper-dependent enzyme cytochrome oxidase, which is important in the production of myelin.\textsuperscript{10} The myelin formed during growth is abnormal because of the decreased activity of cytochrome oxidase,\textsuperscript{10} and there is also evidence that copper may be involved structurally in the stabilization of myelin.\textsuperscript{5} Both myelin dysplasia and demyelination appear to contribute to the pathogenesis of enzootic ataxia. The critical concentration of copper in liver and serum below which enzootic ataxia appears has not been reported. In the whole brain, a maximum concentration of 3 ppm copper (dry weight) may be the threshold value between clinical normality and disease.\textsuperscript{5}

Diagnosis of swayback or enzootic ataxia is based on a history of regional copper status and clinical findings. Diagnosis is verified by the presence of deficient copper levels in blood and tissues of the affected animal and typical lesions of the brain and spinal cord. Differential diagnoses would include caprine arthritis encephalitis, white muscle disease, spinal trauma, vertebral body abscess, polyradiculoneuritis, meningoencephalitis, and toxic neuropathy (caused by organophosphates, lead, arsenic). Treatment of affected animals is supportive but usually unrewarding. The disease may be prevented through adequate copper supplementation of the pregnant doe during the last half of gestation.\textsuperscript{7}

The kids described in this report exhibited the delayed-onset syndrome (enzootic ataxia). We suspect the condition resulted from the types of foodstuffs available to the pregnant does. The does consumed grass and hay, from which copper is poorly absorbed, and corn, which is typically low in copper but from which copper is well absorbed. The contribution of other minerals in the diet, including molybdenum and sulfur, to the development of enzootic ataxia in this case is not known. No additional reports of enzootic ataxia have occurred in the herd after a copper sulfate mineral mix was added to the diet.

References
Relationship of mycotoxins to swine reproductive failure

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Reproductive failure in swine is often a difficult diagnostic problem. Recent estimates are that less than half of the incidents of swine abortion or infertility are correctly diagnosed.1 Many times, when diagnosis of infectious disease or management problems is not obtained, feed quality and safety may be questioned.2 Often these questions involve the relationship of molded feeds and/or mycotoxins to reproductive diseases. Field studies have generally been retrospective in nature and have not clearly implicated mycotoxins.3 Experimental studies to support the role of mycotoxins in swine reproductive diseases are limited.4,5,9,10 Most work has been done with the estrogenic mycotoxin zearalenone.4,5,10 Experimentally, zearalenone can produce anestrus in open mature sows. Estimates of the concentration of zearalenone required to produce anestrus range as low as 3 ppm.10 Zearalenone may also contribute to decreased litter size and early embryonic death. Experimental data indicate that dietary concentrations in excess of 30 ppm are required to produce this effect.5 Limited studies with T-2 toxin and diacetoxyscirpenol (DAS) are suggestive of some interference with gestation. Concentrations of both zearalenone and the trichothecene toxins (T-2 and DAS) required to produce experimental reproductive disease are usually far above those generally found in analysis of feed specimens by diagnostic laboratories.9

Increasingly, researchers and others interested in mycotoxin-related diseases are suggesting that low levels of mycotoxins such as vomitoxin or zearalenone, although not inherently detrimental to reproduction, may serve as markers for other agents not detectable by routine diagnostic procedures. This claim presents a dilemma to the diagnostician because laboratory confirmation often requires detection of an etiologic agent. Furthermore, there is sometimes a tendency to ignore the dose-response principles of toxicology and to assume that any amount of mycotoxin in feed is causally related to the clinical problem present concurrently.

The study described here represents an attempt to conduct a prospective evaluation to examine the hypothesis that increased incidence of mycotoxins in feeds is associated with higher incidence of reproductive failure in swine. A prospective cohort study was designed based on presence or absence of mycotoxins in finished feeds available to swine.6,7 Feed samples were gathered from each herd selected for the study. Each feed or grain collected was analyzed for aflatoxin, zearalenone, zearalenol, T-2 toxin, and vomitoxin utilizing a combination of thin layer chromatography and gas liquid chromatography according to procedures previously described.8 Producers were requested to submit a feed sample each time the source of grain or supplement changed. If the source did not change for at least a month, monthly samples of the same lot were requested.

Herds for study were obtained from 2 sources. Herds cooperating in the National Animal Health Monitoring System (NAHMS) study conducted at Iowa State University and supported by USDA consisted of approximately 30 herds selected at random and sampled for a period of 6 months. This survey was repeated in 2 consecutive years with different herds. The second source group included 24 sow herds selected for uniformity of management and served by a computer-assisted record system (SG)6 located in central Iowa. The SG herds cooperated for a period of 3 years. Each source group was evaluated separately because of the different nature of their selection criteria.

Herds included in the study had a spectrum of sow parities, and all-gilt herds were not used. Only confinement or drylot farrowing operations were utilized.